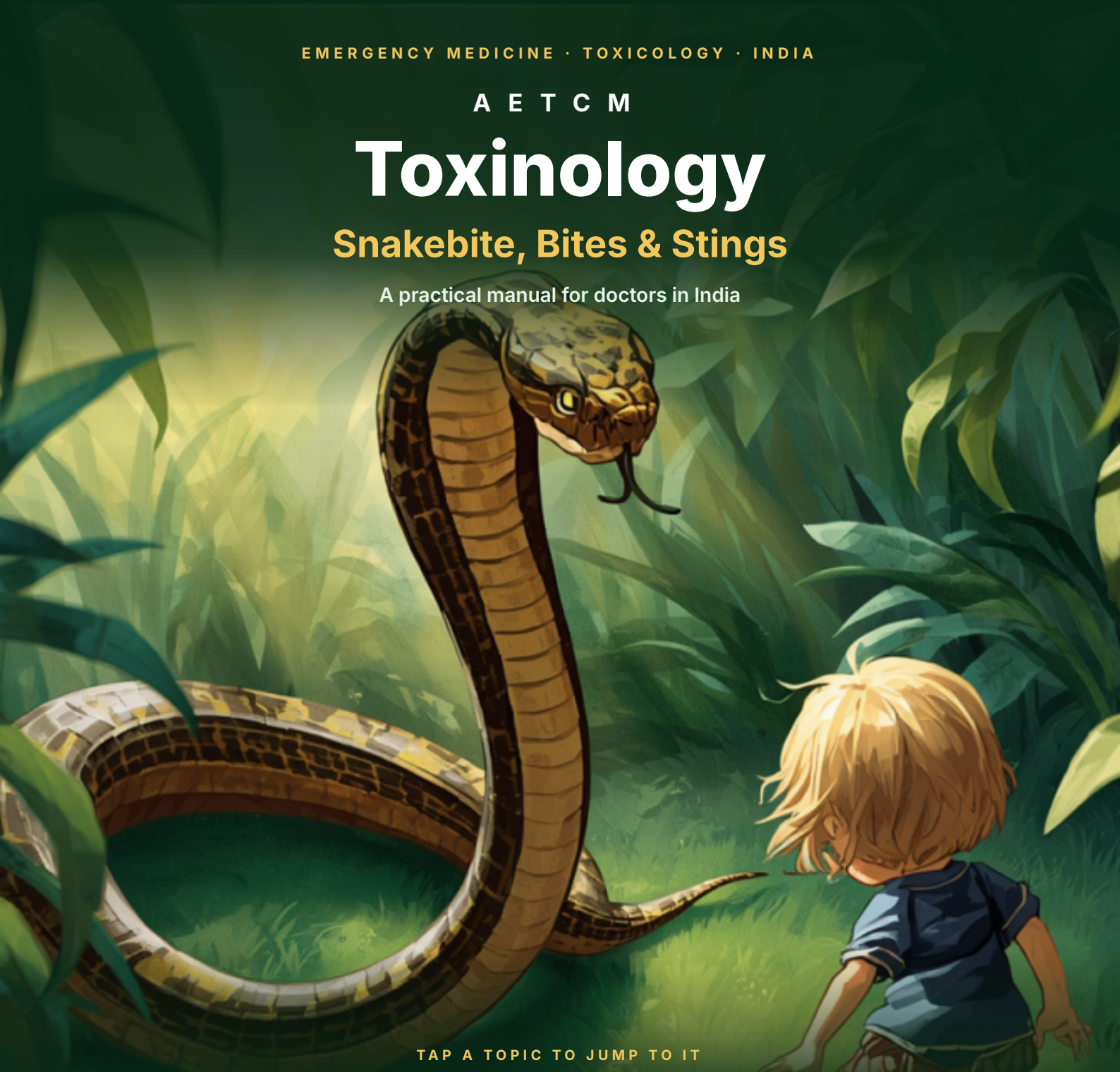
EMERGENCY MEDICINE · TOXICOLOGY · INDIA

AETCM

Toxinology

Snakebite, Bites & Stings

A practical manual for doctors in India



TAP A TOPIC TO JUMP TO IT

Snakebite in India › | Basic concepts › | Venomous snakes › | Snake ID table › | Regional guide › | Snake profiles ›
Venom actions › | Toxidromes › | Syndromic approach › | First aid › | ED assessment › | 20WBCT & investigations ›
ASV preparations › | ASV dilution › | ASV doses › | ASV reactions › | Neurotoxic protocol › | Haemotoxic protocol ›
Kidney & dialysis › | Complications › | Children › | Master algorithm › | Scorpion › | Bees & wasps › | Spiders & centipedes ›
Beetles & caterpillars › | Marine › | Animal bites › | Rabies PEP › | Anaphylaxis › | At a glance › | Visual atlas ›
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AETCM
Academy for Emergency, Trauma & Critical care Medicine

Conceptualised by

Dr. Gireesh Kumar K.P.

For emergency physicians, interns, postgraduates, medical officers and nurses

Toxinology — Snakebite, Bites & Stings

A practical manual for doctors in India

Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM) · First edition · November 2026

How to use this book

This book is built for the person who is woken at 3 a.m. by a farmer with two puncture marks, a child with a scorpion sting and cold sweaty limbs, or a fisherman stung by a jellyfish. Part A covers snakebite from the snake to the kidney: identification, venom actions, the six clinical syndromes, the 20-minute whole blood clotting test, anti-snake venom (ASV) and every common complication. Part B covers scorpions, bees and wasps, spiders, centipedes, beetles and caterpillars, marine creatures, animal and human bites with rabies prophylaxis, and a few unusual ones.

The book is written as a structured theory text with tables, flowcharts and summary boxes. **Green boxes** are "remember" summaries, **red boxes** are dangers and don'ts, **amber boxes** are clinical pearls and **blue boxes** point to pictures and guidelines online, and **violet "Landmark article" boxes** summarise important research published since 2020. The drug doses follow the Government of India Standard Treatment Guidelines for snakebite (MoHFW 2016), the WHO SEARO Guidelines (2016) and the National Action Plan for Snakebite Envenoming (NAPSE 2024); where practice differs between sources, both are shown.

Tap or scan to see it

Wherever you see a small button — ▶ Images ▶ Guideline — it opens a free picture page or an official guideline. A full **visual atlas** with QR codes is given near the end for the printed copy, together with a list of standard guidelines and an alphabetical **subject index**.

AETCM video lectures

Watch the AETCM teaching videos that accompany this book: ▶ AETCM video 1 ▶ AETCM video 2 ▶ AETCM video 3. QR codes for the videos are given in the visual atlas.

A word of caution

Doses and protocols in this book are for trained clinicians and are a guide, not a substitute for the current national guideline, the product insert of the ASV batch in your hand and senior advice. Antivenom potency and presentation vary between manufacturers and batches.

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PREFACE

Why a toxinology book for India?

From Dr. Gireesh Kumar K.P.



Dear colleagues,

Toxinology — the study of venoms and the toxins of living creatures — is one of the most practical sciences in Indian emergency medicine. India carries nearly half of the world's snakebite deaths: the Million Death Study estimated about 1.2 million snakebite deaths between 2000 and 2019, roughly 58,000 every year, mostly among farmers, labourers and children in rural areas during the monsoon. Scorpion stings, bee and wasp attacks, marine injuries and animal bites add a heavy burden of preventable illness and death.

Most of these deaths are avoidable. Snakebite envenoming is treatable with a simple, protocol-based approach: recognition of the clinical syndrome, the 20-minute whole blood clotting test, a full and timely dose of anti-snake venom given intravenously with adrenaline at hand, early airway support for neuroparalysis, and timely dialysis for acute kidney injury. Delay, under-dosing, harmful first aid and unfamiliarity with the national protocol remain the main causes of poor outcome.

This book is written as a standalone, structured text on toxinology for doctors in India. It covers the venomous snakes of India and their identification, venom actions and toxidromes, investigations, anti-snake venom — preparations, dilution, minimum and maximum doses and adverse reactions — the neurotoxic, haemotoxic and renal treatment protocols and the complications of snakebite. The second part deals with scorpion stings, Hymenoptera, spiders, centipedes, blistering insects, marine envenomations, animal bites and rabies prophylaxis. The content follows the Government of India Standard Treatment Guidelines, the WHO South-East Asia Regional guidelines and the National Action Plan for Prevention and Control of Snakebite Envenoming, with tables, flowcharts, dose cards, QR links to pictures and guidelines, and AETCM video lectures.

I hope this book will be useful to undergraduate and postgraduate students, interns, medical officers in primary and secondary care, emergency physicians and nurses — wherever a bitten or stung patient first seeks help. Keep it in the Emergency Medicine Department, the PHC and the ICU, and use the visual atlas to become familiar with the snakes and creatures of your region.

With best wishes for safe and confident practice,

Dr. Gireesh Kumar K.P.
Academy for Emergency, Trauma & Critical care Medicine (AETCM)
November 2026

Abbreviations used

ASV Anti-snake venom	**20WBCT** 20-minute whole blood clotting test	**VICC** Venom-induced consumption coagulopathy
AKI Acute kidney injury	**TMA** Thrombotic microangiopathy	**SBC** Single breath count
FFP Fresh frozen plasma	**AN** Atropine–neostigmine	**HNPV** Hump-nosed pit viper
PIB Pressure immobilisation bandage	**CK** Creatine kinase	**RIG** Rabies immunoglobulin
NAPSE National Action Plan for Snakebite Envenoming	**STG** Standard Treatment Guideline (MoHFW 2016)	**SEARO** WHO South-East Asia Regional Office

PART A

Snakebite Envenoming

From the snake in the paddy field to the dialysis unit — identification, toxidromes, investigations, anti-snake venom and complications.

1. Snakebite in India
2. Venomous snakes of India — identification
3. Snake profiles
4. Venom and how it harms
5. Clinical features and toxidromes
6. First aid and pre-hospital care
7. Assessment in the Emergency Medicine Department
8. Investigations
9. Anti-snake venom
10. ASV reactions
11. Neurotoxic protocol
12. Haemotoxic protocol
13. Kidney in snakebite
14. Other complications
15. Special situations, referral and records
16. Master algorithms

CHAPTER 1 · EPIDEMIOLOGY

Snakebite in India

A neglected tropical disease that kills more Indians than any other bite or sting

The size of the problem

- India accounts for roughly **half of global snakebite deaths**.
- The nationally representative **Million Death Study** (eLife 2020) estimated **1.2 million snakebite deaths** in India from 2000 to 2019 — an average of **about 58,000 deaths per year**. Nearly half were aged 30–69 years and more than a quarter were children under 15.
- About 70% of deaths occurred in eight high-burden states (Bihar, Jharkhand, Madhya Pradesh, Odisha, Uttar Pradesh, Andhra Pradesh including Telangana, Rajasthan and Gujarat), at low altitude, and half during the **monsoon (June–September)**.
- The lifetime risk of an Indian dying of snakebite before age 70 is about **1 in 250**, much higher in some districts.
- Official hospital-based reporting (CBHI) captures only a small fraction — about 3 lakh bites and 2,000 deaths a year — because most victims die at home or on the way.

Who gets bitten?	
Occupation	Farmers, agricultural labourers, plantation workers, herders, snake rescuers, fishermen (sea snakes)
Time	Vipers and cobras — dusk and early night while walking; krait — while **sleeping on the floor**
Season	Monsoon and harvest; flooding drives snakes into homes
Site	Feet and legs (~70%); hands while cutting grass or lifting firewood; head/trunk in floor-sleepers
Snakes	In reported series Russell's viper is the commonest killer, followed by kraits and cobras

See the evidence

▶ Paper Million Death Study ▶ Article WHO fact sheet

Landmark article (eLife 2020;9:e54076)

Trends in snakebite deaths in India from 2000 to 2019 in a nationally representative mortality study.

Suraweera W, Warrell D, Whitaker R, et al. *eLife 2020;9:e54076*.

Message: India had about 1.2 million snakebite deaths in 2000–2019 (≈ 58,000 per year) — far more than official figures. Nearly half occurred in the monsoon, most at home in rural areas, and about 70% in eight high-burden States; the lifetime risk of dying of snakebite before 70 is about 1 in 250.

▶ Read article

National response — NAPSE 2024

WHO listed snakebite envenoming as a priority **neglected tropical disease** in 2017 and in 2019 launched a strategy to **halve deaths and disability by 2030**. India launched the **National Action Plan for Prevention and Control of Snakebite Envenoming (NAPSE)** on 12 March 2024 under the Centre for One Health, NCDC, using a One Health approach.

NAPSE component	What it means for the doctor
Availability of ASV	ASV is on the essential drug list of States; procured under the National Health Mission and the Free Drugs Initiative — it should be available free at PHC/CHC level
Capacity building	Training of medical officers and nurses in the national protocol; "snakebite-ready" facilities with ASV, Ambu bag, oxygen, ventilator link, dialysis link
Surveillance	Reporting of snakebite cases and deaths through the IHIP/IDSP portal; Karnataka became the first State to make snakebite notifiable (2024)
Helpline	**Snakebite helpline 15400** piloted in Puducherry, Madhya Pradesh, Assam, Andhra Pradesh and Delhi

Community & wildlife	IEC material ("Let's put an end to snakebite deaths"), prevention, snake rescue with forest departments, veterinary component

Official documents

▶ Guideline NAPSE (NCDC 2024) ▶ Website NCDC programme page ▶ Guideline WHO strategy 2019

📰 Landmark article (Trans R Soc Trop Med Hyg 2024)

National Action Plan for the prevention and control of snakebite envenoming in India: strategies and challenges. Munshi H, Gajbhiye R. *Trans R Soc Trop Med Hyg 2024*.

Message: NAPSE (2024) adopts a One Health approach to halve snakebite deaths and disability by 2030 — sustained ASV availability, training of doctors, surveillance and community education are the pillars; implementation at district level is the challenge.

▶ Read article

Why do snakebite victims die?

Before hospital

Faith healers and delay · tight tourniquets · cutting and sucking · long transport without airway support · night-time krait bite not recognised

In hospital

No ASV or under-dosing · wasting time on test doses · missed coagulopathy · no Ambu bag/ventilator · late dialysis · ASV anaphylaxis not anticipated

After discharge

Late serum sickness · chronic kidney disease · hypopituitarism after Russell's viper · amputations and contractures · psychological trauma

Prevention — what we teach the community

- Wear boots and long trousers in fields; use a stick to beat grass ahead.
- Carry a **torch** (or mobile flashlight) at night.
- **Sleep on a cot** and under a well-tucked mosquito net — this alone prevents most krait bites.
- Keep the surroundings of the house free of rubbish, firewood stacks and rodents (rats attract snakes); store grain in closed containers.
- Do not put hands into holes, under stones or into dense bushes.
- Never handle or try to kill a snake — call trained rescuers or the forest department.
- Know the nearest centre with ASV and the ambulance number (**108/102**), and the helpline **15400** where available.
- Reach hospital immediately — the "golden hour" applies.

▶ Watch — AETCM video lectures

▶ AETCM video 1 ▶ AETCM video 2 ▶ AETCM video 3

Remember

About 70% of all snakebites in India are by non-venomous snakes, and even venomous snakes give "dry bites" (no venom) in 20–50%. Yet every bite is treated as venomous until 24 hours of observation prove otherwise.

CHAPTER 1 · BASIC CONCEPTS

Toxinology — Basic Concepts

Venoms, poisons, venomous animals of India and the principles of antivenom therapy

Definitions

- **Toxinology** is the study of toxins produced by living organisms — animal venoms, plant and fungal toxins, and bacterial toxins — and of their clinical effects and treatment.
- A **venom** is a toxin **actively delivered** by a bite or sting through a specialised apparatus (fangs, sting, spine, nematocyst). A **poison** is passively absorbed when the organism is eaten or touched (toads, puffer fish, poisonous plants).
- **Envenoming** means clinical illness from injected venom; a bite without venom injection is a **dry bite**.
- **Toxidrome** — the pattern of features produced by a group of toxins (neurotoxic, haemotoxic, myotoxic, autonomic storm etc.).

Venomous and poisonous animals of medical importance in India

Group	Examples
Reptiles	Elapids (cobras, kraits, coral snakes, sea snakes), vipers and pit vipers, rear-fanged colubrids
Arachnids	Scorpions (red, black), spiders (widows, tarantulas), ticks (paralysis), mites (scrub typhus vector)
Insects	Bees, wasps, hornets, ants, blister and Paederus beetles, caterpillars
Myriapods	Centipedes, millipedes
Marine animals	Jellyfish, sea anemones, stingrays, catfish, stonefish, sea urchins, cone snails
Amphibians	Toads (bufotoxins — poisonous, not venomous)
Mammals	Not venomous, but bites transmit rabies and bacterial infection

Principles of antivenom therapy

1. **Specificity** — an antivenom neutralises the venoms used to raise it (specific) and sometimes related venoms (paraspecific). Indian ASV covers the Big Four; it does not cover HNPV, pit vipers or sea snakes.
2. **Neutralisation, not reversal** — antivenom binds circulating and tissue-accessible venom; it cannot undo necrosis, destroyed nerve terminals or tubular necrosis. Hence **early** treatment.
3. **Dose by venom load, not body weight** — children get the same dose as adults.
4. **Route** — intravenous only for rapid distribution.
5. **Re-dosing by clinical end-points** — clotting restored (6-hour rule), neurological improvement (1-hour rule).
6. **Safety** — heterologous (equine) protein; early, pyrogenic and late reactions are expected and must be managed, not feared.

Types of antivenom worldwide

Type	Notes
Whole IgG	Long half-life; more reactions (older products)
F(ab')$_2$ fragments	Pepsin digestion — Indian ASV; intermediate half-life
Fab fragments	Papain digestion (e.g. CroFab, digoxin-Fab); short half-life, recurrence common
Recombinant / monoclonal / small-molecule	Under development (e.g. varespladib — PLA$_2$ inhibitor; broadly neutralising antibodies)

Pearl — the future

Region-specific Indian antivenoms, small-molecule inhibitors such as **varespladib** (PLA$_2$) and **marimastat** (metalloproteinase), and recombinant antibody cocktails are in clinical development and may one day be given in the field before transport.

Landmark article (Sci Transl Med 2024;16:eadk1867)

Synthetic development of a broadly neutralizing antibody against snake venom long-chain α-neurotoxins. Khalek IS, Senji Laxme RR, … Sunagar K, Jardine JG. *Sci Transl Med 2024;16:eadk1867*.

Message: An Indian (IISc)–US team produced a fully human synthetic antibody that neutralises long-chain α-neurotoxins of many elapids and protected mice from lethal venom — a step towards monoclonal, reaction-free antivenoms.

▶ Read article

Landmark article (Cell 2025;188:3117–3134)

Snake venom protection by a cocktail of varespladib and broadly neutralizing human antibodies. Glanville J, Bellin M, Pletnev S, et al. *Cell 2025;188:3117–3134*.

Message: Two broadly neutralising antibodies from a hyper-immune human donor, combined with the PLA_2 inhibitor varespladib, protected mice against venoms of 19 WHO category 1–2 elapids (including cobras, king cobra, common and banded kraits) — proof of principle for a 'universal' elapid antivenom.

▶ Read article

Landmark article (BMJ Glob Health 2024;9:e015985)

Oral varespladib for the treatment of snakebite envenoming in India and the USA (BRAVO): a phase II randomised clinical trial. Gerardo CJ, Carter RW, Kumar S, et al. *BMJ Glob Health 2024;9:e015985*.

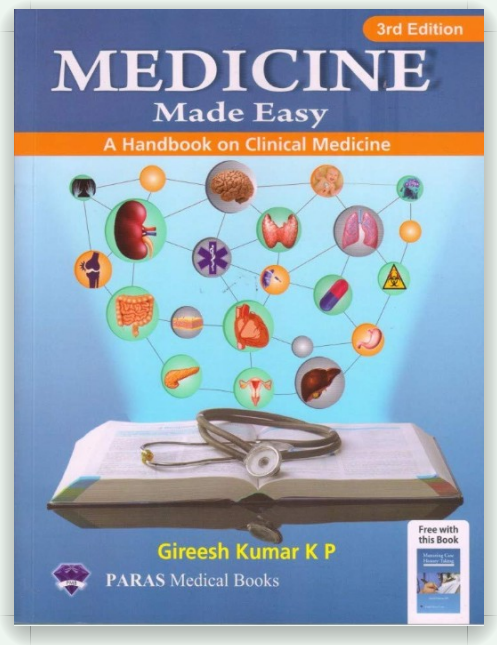
Message: The first trial of a direct toxin inhibitor in snakebite (95 patients, India and USA): oral varespladib added to antivenom was safe; the primary end-point was not met, but there was a signal of benefit when treatment began within 5 hours of the bite.

▶ Read article

ALSO BY THE AUTHOR

Dr. Gireesh Kumar K.P.

Books for the practising doctor



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3rd Edition

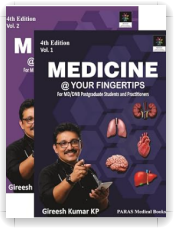
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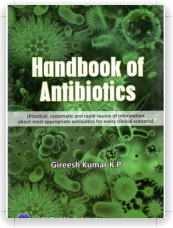
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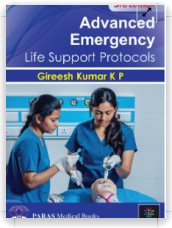
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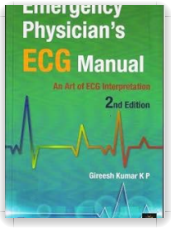
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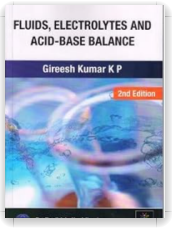
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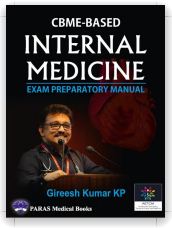
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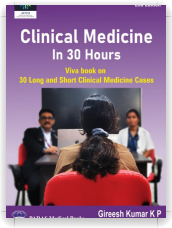
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CHAPTER 2 · IDENTIFICATION

Venomous Snakes of India

Families, the Big Four, the neglected species, and how to tell venomous from harmless

India has about 300+ species of snakes, of which around 60 are venomous and a much smaller number are of medical importance. The polyvalent ASV made in India is raised against only **four species — the "Big Four"**. Knowing them, and their common look-alikes, is the first clinical skill.

Classification of venomous snakes

Family / subfamily	Fangs	Important Indian snakes	Main venom action
Elapidae (land elapids)	Short, fixed, front (proteroglyph)	Spectacled cobra, monocled cobra, King cobra, Common krait, Banded krait, Sind krait, coral snakes	Neuro ± Local (cobra)
Elapidae – Hydrophiinae (sea snakes)	Short, fixed, front	Beaked sea snake and other Hydrophis species on both coasts	Myo Neuro
Viperidae – Viperinae (true vipers)	Long, hinged, front (solenoglyph)	Russell's viper, Saw-scaled viper	Haemo Local Renal
Viperidae – Crotalinae (pit vipers)	Long, hinged; heat-sensing **loreal pit**	Hump-nosed pit viper, Malabar pit viper, Bamboo (green) pit viper, other Trimeresurus	Local Haemo ± Renal
Colubridae (rear-fanged, mildly venomous)	Grooved rear fangs (opisthoglyph)	Cat snakes (Boiga), vine snakes, red-necked keelback (Rhabdophis)	Usually local only; Rhabdophis can cause coagulopathy

The older textbook grouping "Elapidae · Viperidae · Hydrophidae · Crotalidae" (cobras and kraits; Russell's viper; sea snakes; pit vipers) is the same as the table above in modern terms.

Fang arrangement. Fang marks alone cannot tell venomous from non-venomous: harmless snakes leave rows of small teeth marks; venomous snakes may leave one, two or several punctures, or only a scratch (krait). ▶ Article

The Big Four and the important "neglected" snakes

Snake	Distribution	ASV covers?	Dominant syndrome
Spectacled cobra *Naja naja*	Whole mainland India	Yes (Big Four)	Neurotoxic + painful local swelling/ necrosis
Common krait *Bungarus caeruleus*	Whole mainland India	Yes (Big Four)	Neurotoxic, minimal local signs, night bite, abdominal pain
Russell's viper *Daboia russelii*	Whole India, plains and agricultural land	Yes (Big Four)	Coagulopathy, AKI, shock, capillary leak; neurotoxicity in parts of South India
Saw-scaled viper *Echis carinatus*	Dry areas — Maharashtra, Rajasthan, Gujarat, Deccan, Tamil Nadu	Yes (Big Four)	Coagulopathy and local swelling; AKI uncommon
Monocled cobra *N. kaouthia*	East & North-East (WB, Odisha, Assam, Bihar)	Partial	Neurotoxic + severe local necrosis
King cobra *Ophiophagus hannah*	Western Ghats, NE, Eastern Ghats, Andamans	No specific Indian ASV	Rapid neuroparalysis (rare bites)
Banded krait *B. fasciatus*; Sind krait *B. sindanus*	NE & East; Rajasthan, Punjab, Maharashtra	Poor	Neurotoxic
Hump-nosed pit viper *Hypnale hypnale*	Western Ghats — Kerala, Karnataka, Goa	No	Local swelling, coagulopathy, AKI, TMA

Malabar & bamboo pit vipers	Western Ghats	No	Local pain and swelling, mild coagulopathy
Sea snakes (*Hydrophis*)	Both coasts, fishermen	No	Myotoxic + neurotoxic

Pearl — regional variation

Venom of the same species differs between regions. Russell's viper in Tamil Nadu and Sri Lanka causes ptosis and paralysis; saw-scaled vipers of Rajasthan (*E. c. sochureki*) are poorly neutralised by southern-venom ASV. Indian ASV made from southern (Tamil Nadu) venom may need larger doses elsewhere. ▶ Paper

CHAPTER 2 · IDENTIFICATION TABLE

Identifying Poisonous Snakes of India — at a Glance

Use with a photograph of the snake; never ask relatives to catch or kill it

Feature	Spectacled cobra	Common krait	Russell's viper	Saw-scaled viper	Hump-nosed pit viper
Length	1–2 m	1–1.5 m	1–1.5 m, stout	30–60 cm, small	30–55 cm, small
Colour	Brown, black, yellowish; very variable	Glossy steel-blue/ black	Brown/tan	Grey-brown, sandy	Grey-brown with dark blotches
Key pattern	**Hood** with **spectacle (binocellate) mark** on back of hood; 1–2 dark bands under the neck	**Paired thin white cross-bands**, clearer towards the tail; belly white	**Three rows of dark brown oval spots** ringed black-white, forming chains	White **zig-zag line** on flanks; **"bird-foot"/ arrow mark** on head	Upturned, **"hump" snout**; triangular head
Head	Not distinct from neck (until hooded); large head shields	Small, not distinct; large shields	**Large triangular**, small scales on top, big nostrils	Pear-shaped, small scales	Triangular; **loreal pit** between eye and nostril
Scales	Smooth	**Enlarged hexagonal vertebral scales** down the mid-back; single subcaudals	Strongly keeled	Keeled, serrated flank scales	Keeled
Behaviour	Raises hood, hisses; active dusk/day	Nocturnal; enters houses, bites sleepers; docile by day	Loud prolonged **hiss**; strikes fast	Coils in "figure of 8", rubs scales — **sizzling/saw sound**	Leaf litter, plantations; bites when stepped on
Bite site	Pain, swelling, blisters, necrosis	Often **no pain, no mark** or a pinhead mark	Severe pain, swelling, bleeding from fang marks	Painful swelling, local bleeding	Painful swelling, blistering
Look-alike	Rat snake (no hood)	**Common wolf snake**	**Sand boa**, Python	Cat snake	—

Krait or wolf snake?

Krait bands are **paired and thin**, best seen on the posterior body; the wolf snake has single broad bands fading towards the tail, a flattened head and no enlarged mid-dorsal scales. ▶ Images ▶ Images

Russell's viper or sand boa?

The rough-scaled sand boa has blotches that resemble Russell's viper, but it is short and thick with a **blunt stubby tail**, small eyes and a narrow head, and it does not hiss loudly. The Indian rock python also has blotches but is much larger with **heat-sensing labial pits** on the lips. ▶ Images ▶ Images ▶ Images

Simple bedside rules

- A snake with a **hood** is a cobra (or king cobra).
- Large **head shields + hexagonal vertebral scales + paired bands** = krait.
- Small scales on a **broad triangular head** = viper.
- A **pit** between eye and nostril = pit viper (no Indian ASV).
- Paddle-shaped flattened tail = sea snake.
- Shape of the pupil and "triangular head" myths are unreliable in India — cobras and kraits have round pupils and narrow heads.

Do not

Do not send relatives back to catch or kill the snake — a "dead" snake can reflexly bite for an hour. A mobile-phone photograph is enough. Never delay ASV for identification: treat the syndrome you see.

Learn the faces

▶ Website Indian Snakes portal (state-wise photographs) ▶ Images cobra ▶ Images saw-scaled viper ▶ Images hump-nosed pit viper

CHAPTER 2 · REGIONAL GUIDE

Medically Important Snakes by Region of India

Know the snakes of your district — venom and ASV response vary across the country

Region	Medically important venomous snakes	Clinical notes
North India (Punjab, Haryana, UP, Delhi, Uttarakhand plains, J&K)	Spectacled cobra, common krait, Russell's viper, saw-scaled viper (drier west), Sind krait (Punjab), Central Asian cobra (*Naja oxiana*, far north-west), Himalayan pit vipers (hills)	Krait neuroparalysis during monsoon floods; saw-scaled vipers of the north-west (*E. c. sochureki*) need more ASV
West India (Rajasthan, Gujarat, Maharashtra, Goa)	Saw-scaled viper (Ratnagiri, Konkan, Rajasthan), Russell's viper, spectacled cobra, common krait, Sind krait (Rajasthan, Maharashtra); Malabar and bamboo pit vipers and HNPV in the Goa–Konkan Ghats; sea snakes on the coast	Ratnagiri district is a classic saw-scaled viper area; red scorpion stings in Konkan villages
Central India (MP, Chhattisgarh, Vidarbha)	Spectacled cobra, common krait, Russell's viper, saw-scaled viper	High rural mortality; long transport — give ASV at PHC level
East India (Bihar, Jharkhand, Odisha, West Bengal)	**Monocled cobra**, spectacled cobra, common krait, **banded krait**, **greater black krait** (north Bengal), Russell's viper; sea snakes on the Odisha/Bengal coast	Bihar, Jharkhand and Odisha among the highest burden States; monocled cobra necrosis; floor-sleeping krait bites
North-East India	Monocled cobra, **king cobra**, banded krait, greater black krait, several green pit vipers (*Trimeresurus* spp.), red-necked keelback	Many species not covered by Indian ASV — airway and ventilation are crucial; pit viper bites managed supportively
South India — Tamil Nadu, Andhra, Telangana, Karnataka	Russell's viper (neurotoxic as well as haemotoxic in parts of Tamil Nadu), saw-scaled viper, spectacled cobra, common krait; HNPV and pit vipers in the Ghats	Indian ASV is made from Tamil Nadu venom — usually good response here
Kerala and Western Ghats	Russell's viper, spectacled cobra, common krait, saw-scaled viper (north Kerala), **hump-nosed pit viper**, Malabar and bamboo pit vipers, **king cobra**, coral snakes; sea snakes along the coast	HNPV is a major cause of coagulopathy and AKI with no antivenom; Kerala's "SARPA" programme trains snake rescuers
Islands (Andaman & Nicobar, Lakshadweep)	Andaman cobra, Andaman krait, Andaman pit vipers, king cobra; sea snakes and kraits (sea kraits) around islands	Mainland ASV has limited efficacy; evacuation planning essential
High Himalaya	Himalayan pit viper (*Gloydius himalayanus*) and other pit vipers	Mostly local envenoming

Pearl — the district snake list

Every Emergency Medicine Department and PHC should display photographs of the venomous snakes and common harmless look-alikes found in its own district, with the local names used by villagers. Patients often name the snake in the local language — learn these names.

Regional photographs

▶ Website state-wise species lists and photographs ▶ Paper venom variation and antivenom gaps

Remember

The **syndrome** guides treatment everywhere in India. Regional knowledge tells you **when to expect a poor ASV response** (north-west saw-scaled vipers, Sind krait, monocled cobra, HNPV, NE pit vipers) and therefore when to plan early for ventilation, dialysis or blood products rather than escalating ASV endlessly.

CHAPTER 2 · CLASSIC EXAM TABLES

Venomous vs Non-venomous, and Elapid vs Viper

Differences frequently asked in examinations and in court

Venomous and non-venomous snakes — traditional differences

Feature	Venomous	Non-venomous
Fangs	Present — grooved or canalised, front or rear	Absent; rows of small solid teeth
Bite marks	One or two distinct fang punctures, ± small teeth marks	Two curved rows of many small teeth marks
Head scales	Small scales (vipers) or large shields with special features (third supralabial touching eye and nasal shield — cobra; enlarged vertebrals — krait)	Usually large shields
Belly scales (ventrals)	Broad, cover the full width of the belly	Broad in most land snakes too (pythons, boas have narrow ventrals)
Tail	Compressed, paddle-like in sea snakes; tapering in land snakes	Tapering; blunt in sand boas
Pupil	Round (cobra, krait) or vertical (vipers)	Round or vertical (cat snakes)
Local reaction	Pain, swelling, bleeding, necrosis (except krait/sea snake)	Minor local pain, bleeding from teeth marks
Systemic features	Present in envenoming	Absent (anxiety symptoms only)

Caution

No single external feature reliably separates venomous from harmless snakes in India — kraits have round pupils and narrow heads, and harmless cat snakes have vertical pupils. Learn the species by sight; do not rely on rules alone.

Elapid bite and viper bite — clinical differences

Feature	Elapid (cobra, krait)	Viper (Russell's, saw-scaled)
Venom	Neurotoxic (+ cytotoxic in cobra)	Haemotoxic/vasculotoxic, nephrotoxic
Fangs	Short, fixed	Long, hinged
Local pain and swelling	Cobra — marked; krait — absent	Marked, spreading within hours
Local bleeding	Absent	Present from fang marks
Necrosis	Cobra — common ("wet")	Common, with blisters
Onset of systemic features	Minutes to hours (krait up to 24 h)	Minutes to hours
Neurological	Ptosis, ophthalmoplegia, bulbar and respiratory paralysis	Usually absent (except some Russell's viper populations)
Haematological	Normal clotting; 20WBCT clotted	Unclotted 20WBCT, systemic bleeding
Kidney	Rarely involved (krait rhabdomyolysis)	AKI common with Russell's viper
Cause of death	Respiratory failure	Shock, ICH, AKI
Response to ASV	Cobra good; krait slow	Clotting restored in ~6 h
Neostigmine	Useful in cobra	Not indicated
Post-mortem	Few local findings (krait); asphyxial signs	Local haemorrhage, bleeding into organs, swollen kidneys, petechiae

Spectacled Cobra

Naja naja · Elapidae

Also known as: Nag (Hindi) · Moorkhan (Malayalam) · Nalla pambu (Tamil) · Indian cobra

Identify	1–2 m; colour very variable (black, brown, yellowish). Raises a **hood** showing a **spectacle (two connected rings)** mark at the back; one or two dark bands on the throat. Smooth scales, round pupil.
Habitat & time	All over mainland India; fields, termite mounds, rat holes, near houses. Active at dusk and early night; bites while stepping on it or putting a hand in a hole.
Venom	Post-synaptic α-neurotoxins (three-finger toxins) bind nicotinic ACh receptors (curare-like, **reversible**); cytotoxins (cardiotoxins) and PLA_2 cause local necrosis.
Local	Immediate pain, rapidly spreading swelling, **blisters**, darkening and **"wet" necrosis** with a putrid smell (days 2–5); tender regional lymph nodes.
Systemic	Onset 15 min–6 h: drowsiness, vomiting, hypersalivation, **ptosis**, ophthalmoplegia, diplopia, dysphagia, dysarthria, neck-flexor weakness, then **respiratory paralysis**. Descending pattern.
Tests	20WBCT usually clots; CBC (neutrophilia), SBC, SpO_2, ABG; CK; wound swab when infected.
Treatment	ASV 10 vials loading; **atropine–neostigmine trial is very useful** (post-synaptic block); early bag-mask/intubation; wound care and debridement after demarcation.
Pitfalls	Neuroparalysis may be quick (within an hour); a normal 20WBCT does not exclude cobra envenoming. Local necrosis continues even after ASV.

Spectacled cobra

▶ Images

Naja naja — hood & spectacle mark

Envenoming profile

Local	**mild**	**moderate**	severe
Neuro	**mild**	**moderate**	**severe**
Haemo	mild	moderate	severe
Renal	mild	moderate	severe

Remember

Painful local swelling + early ptosis = think cobra. Cobra paralysis responds well to ASV and neostigmine.

CHAPTER 3 · SNAKE PROFILE 2 OF 11

Monocled Cobra & King Cobra

Naja kaouthia · Ophiophagus hannah · Elapidae

Also known as: Monocled cobra: Gokhra (Bengali) · King cobra: Raja nag (Hindi), Rajavembala (Malayalam)

Monocled cobra	East and North-East India (West Bengal, Odisha, Bihar, Assam). Single **"O" (monocle)** mark on the hood. Venom rich in cytotoxins — **severe local necrosis** — plus α-neurotoxins. Indian polyvalent ASV neutralises it only partly; larger doses may be needed.
King cobra — identify	World's longest venomous snake (3–5 m); olive-brown with pale cross-bands, narrow hood without spectacle mark, a pair of large **occipital scales** behind the head shields. Western Ghats, North-East, Eastern Ghats, Andamans; eats other snakes.
King cobra — venom	Large venom yield; mainly neurotoxic (α-neurotoxins) with local swelling. Untreated mortality has been quoted as high as 75%, but most bites are rare and many involve non-fatal amounts; venom is less potent per mg than Indian cobra venom.
King cobra — treatment	No King cobra antivenom is manufactured in India (Thai Red Cross makes one). Give Indian polyvalent ASV if neurotoxic signs (some cross-neutralisation), neostigmine trial, and **early airway support and ventilation** — the mainstay.
Spitting	Some cobras spit venom into the eyes (cobra-spit ophthalmia): intense pain, blepharospasm, chemosis, corneal erosion. Irrigate copiously with water or saline, topical antibiotic, cycloplegic, analgesia. ASV is not needed for eye exposure alone.

Monocled cobra

▶ Images



Naja kaouthia — single 'O' mark

Envenoming profile

Local	**mild**	**moderate**	**severe**
Neuro	**mild**	**moderate**	**severe**
Haemo	mild	moderate	severe
Renal	mild	moderate	severe

Pearl

In the North-East a cobra bite with massive necrosis and modest paralysis is typical of the monocled cobra; prepare for debridement and skin grafting.

See them

▶ Images King cobra

Common Krait

Bungarus caeruleus · Elapidae

Also known as: Karait (Hindi) · Vellikettan (Malayalam) · Kattu viriyan (Tamil) · Manyar (Marathi)

Identify	1–1.5 m; glossy **steel-blue/black** with **paired thin white cross-bands** (often absent on the front third); white belly; **enlarged hexagonal vertebral scales**; undivided subcaudals; small head with large shields.
Habit	Strictly **nocturnal**; enters homes searching for rats and other snakes; bites people **sleeping on the floor** (10 p.m.–6 a.m.), often on the hand, face or trunk. Usually docile in daytime.
Venom	**Pre-synaptic β-bungarotoxin** (PLA_2) destroys nerve terminals — stops ACh release; also α-bungarotoxin. Block is **not reversed** by neostigmine and recovers only as terminals regenerate (days–weeks).
Local	Painless; often **no visible fang mark** or a tiny pinhead mark; no swelling. Patients wake with symptoms and deny a bite.
Systemic	Early-morning **severe epigastric/abdominal pain**, vomiting, chest or joint pain, then ptosis, ophthalmoplegia, bulbar palsy, neck weakness, **respiratory failure**; fixed dilated pupils and "coma" may be **locked-in paralysis**, not brain death. Hyponatraemia (natriuresis), and occasionally rhabdomyolysis/AKI.
Tests	20WBCT clots; serum sodium; CK; ABG; SBC/neck-lift test; ECG.
Treatment	ASV 10 vials early (it cannot reverse established pre-synaptic damage but stops further binding); a single AN trial is reasonable when species uncertain, but **do not persist** if no response; prolonged ventilation (days 5–10); correct sodium.
Pitfalls	Misdiagnosed as gastritis, food poisoning, stroke, "hysteria" or brain death. Do not declare brain death in a paralysed krait victim.

Common krait

▶ Images



Bungarus caeruleus — paired white bands

Envenoming profile

	mild	moderate	severe
Local			
Neuro	**mild**	**moderate**	**severe**
Haemo			
Renal	**mild**		

Never miss

Floor-sleeper + early-morning abdominal pain + ptosis, with no bite mark = common krait until proved otherwise.

CHAPTER 3 · SNAKE PROFILE 4 OF 11

Banded, Sind & Other Kraits

Bungarus fasciatus · B. sindanus · B. niger · B. walli

Also known as: Banded krait: Shankhini (Bengali)

Banded krait	Bold alternating **yellow and black bands of equal width** that encircle the body; triangular cross-section with a raised vertebral ridge and a **blunt tail**. North-East and East India. Relatively docile; neurotoxic envenoming.
Sind krait	*B. sindanus* — Rajasthan, Gujarat, Punjab, Maharashtra. Similar to common krait but narrower white bands; venom reported to be several times more potent than common krait in mouse studies and poorly neutralised by Indian ASV.
Greater black krait	*B. niger* — North-East; uniform black, causes neuroparalysis and rhabdomyolysis with AKI.
Clinical	Same pattern as common krait: painless bite, delayed (up to 12–24 h) descending paralysis; abdominal pain; prolonged ventilation.
ASV	Use Indian polyvalent ASV (10 vials, may repeat once) — partial cross-neutralisation. Ventilation is the mainstay. Region-specific antivenoms are under development.

Banded krait

▶ Images

Yellow & black bands, NE India

Envenoming profile

	mild	moderate	severe
Local			
Neuro	**mild**	**moderate**	**severe**
Haemo			
Renal	**mild**		

Pearl

If neuroparalysis progresses despite 20 vials of ASV, think of a non-Big-Four elapid and concentrate on ventilation rather than more ASV.

See them

▶ Images Sind krait

Russell's Viper

Daboia russelii · Viperidae (Viperinae)

Also known as: Daboia/Chandrabora (Bengali) · Ghonas (Marathi) · Anali (Malayalam) · Kannadi viriyan (Tamil)

Identify	1–1.5 m, stout; brown with **three longitudinal rows of large dark oval spots**, each edged black and white (chain pattern); large flat triangular head with a pale "V"; strongly keeled scales; large nostrils; **loud, long hiss**.
Habitat	Plains, rice fields, grassland and scrub all over India; the commonest cause of fatal snakebite in most series.
Venom	Factor X and V activators (procoagulants) → **venom-induced consumption coagulopathy**; haemorrhagins (SVMP) damage vessels; PLA_2; direct nephrotoxicity; capillary-leak factors; in some regions neurotoxic PLA_2.
Local	Severe pain, swelling spreading up the limb within hours, bleeding from fang marks, blisters, bruising; compartment syndrome is uncommon but possible.
Systemic	Bleeding — gums, epistaxis, haematemesis, haematuria, subconjunctival, ecchymoses, **intracranial haemorrhage**; shock; **AKI** (oliguria, dark urine) in 20–30%; capillary leak — **bilateral parotid swelling, conjunctival oedema (chemosis)**, pleural effusion; myocardial injury; ptosis/paralysis in Tamil Nadu and Sri Lanka; later **hypopituitarism** (Sheehan-like).
Tests	20WBCT unclotted; PT/INR, aPTT, fibrinogen, D-dimer; CBC with smear (schistocytes, low platelets — TMA); creatinine, K^+, urine for blood/myoglobin; CK; ECG.
Treatment	ASV loading dose 10 vials; 20WBCT every 6 h and repeat ASV until clotting restored; fluids, urine output ≥1 mL/kg/h; early dialysis; blood products only after ASV.
Follow-up	Creatinine at discharge and later; screen for hypopituitarism (fatigue, hypotension, hypoglycaemia, amenorrhoea, loss of libido) weeks to years later.

Russell's viper

▶ Images



Daboia russelii — 3 chains of spots

Envenoming profile

Local	mild	moderate	severe
Neuro	mild	moderate	severe
Haemo	mild	moderate	severe
Renal	mild	moderate	severe

Remember

Bleeding + swelling + AKI + parotid swelling/ chemosis = Russell's viper (WHO syndrome 2).

Saw-scaled Viper

Echis carinatus · Viperidae (Viperinae)

Also known as: Phoorsa (Marathi) · Churuta (Malayalam) · Surutai viriyan (Tamil) · Little Indian viper

Identify	Small (30–60 cm), grey-brown/sandy; white **zig-zag line** along each flank; a white **"bird-foot" or arrow-head mark** on top of the head; serrated keeled flank scales. When threatened it forms C-shaped coils and rubs them to produce a **sizzling/saw-like sound**.
Habitat	Dry, rocky, sandy areas — Maharashtra (Ratnagiri), Rajasthan, Gujarat, Deccan plateau, Tamil Nadu; often hides under stones and in loose soil.
Venom	Prothrombin activator (ecarin) and haemorrhagins → coagulopathy and bleeding; local cytotoxic effects. Small venom yield, but highly potent.
Local	Painful swelling, local bleeding, blisters; necrosis less than with cobras.
Systemic	Incoagulable blood, spontaneous bleeding (gums, haematuria, haemoptysis, intracranial), shock in severe cases. **AKI is uncommon** compared with Russell's viper. Coagulopathy may recur after initial correction.
Tests	20WBCT (often unclotted), PT/INR, platelets, creatinine.
ASV	Low-dose regimen in the STG: **6 vials** loading infusion over 30 min, then 2 vials 6-hourly as needed; or the standard 10-vial loading dose. Repeat 6-hourly until clotting restored.
Pitfalls	Northern and western *E. c. sochureki* venom is poorly neutralised by Indian ASV — expect larger doses and slower response.

Saw-scaled viper

▶ Images



Echis carinatus — 'bird-foot' mark

Envenoming profile

Local	**mild**	**moderate**	severe
Neuro	mild	moderate	severe
Haemo	**mild**	**moderate**	**severe**
Renal	**mild**	moderate	severe

Remember

Small snake, big bleed: saw-scaled viper causes coagulopathy out of proportion to its size; the kidney is usually spared.

Hump-nosed Pit Viper

Hypnale hypnale (also H. nepa) · Viperidae (Crotalinae)

Also known as: Kerala, Karnataka, Goa, Western Ghats foothills and plains; Sri Lanka

Identify	Small (30–55 cm), grey-brown with dark triangular blotches; triangular head with a distinctly **upturned "hump" snout**; **loreal pit** between eye and nostril. Lies in leaf litter and plantations; often mistaken for a saw-scaled viper.
Importance	Emerging as a major cause of snakebite in South-West India (especially Kerala). Earlier thought harmless; now known to cause severe envenoming.
Local	Pain, swelling, blistering, sometimes necrosis; local effects may be the only feature.
Systemic	Coagulopathy (VICC, low fibrinogen), **AKI** — often with **thrombotic microangiopathy** (anaemia, schistocytes, thrombocytopenia), occasionally myocardial injury and death.
ASV	**No specific antivenom in India; Indian polyvalent ASV does not neutralise HNPV venom.** Coagulopathy may persist for up to 3 weeks. Treat with supportive care: FFP/ cryoprecipitate if bleeding, close renal monitoring, early dialysis; plasma exchange considered in TMA.
Dilemma	If the snake was not seen and the patient is in Kerala with coagulopathy, the syndrome may be Russell's viper — give ASV. If the snake is **confirmed** as HNPV, do not keep giving ASV.

Hump-nosed pit viper

▶ Images

Hypnale hypnale — upturned snout

Envenoming profile

Local	**mild**	**moderate**	**severe**
Neuro	mild	moderate	severe
Haemo	**mild**	**moderate**	severe
Renal	**mild**	**moderate**	**severe**

Do not

Do not give repeated doses of polyvalent ASV for a confirmed hump-nosed pit viper bite — it does not work, and exposes the patient to reactions.

Pearl

Persistent prolonged INR for days after a 'small viper' bite in Kerala with normal response to ASV absent — suspect HNPV.

Green & Malabar Pit Vipers

Craspedocephalus malabaricus · C. gramineus · Trimeresurus spp.

Also known as: Western Ghats, North-East and Andaman pit vipers; 'green pit viper', 'bamboo pit viper'

Identify	Arboreal, often bright **green** (bamboo pit viper) or variable green/brown/orange (Malabar pit viper); triangular head, prehensile tail, loreal pit. Found on bushes and low branches in hill forests, tea and coffee estates.
Clinical	Painful local swelling, blistering and lymphangitis; mild, usually self-limiting coagulopathy; systemic bleeding and AKI are rare.
ASV	**No ASV for confirmed green pit viper bites** (national STG): Indian polyvalent ASV has no antibodies against them. Supportive care — limb elevation, analgesia (paracetamol/ tramadol), tetanus, wound care, observe 24 h with 20WBCT.
When to worry	Rapidly spreading swelling, spontaneous bleeding or falling platelets — admit, monitor coagulation; FFP if bleeding.

Malabar pit viper

▶ Images

Craspedocephalus malabaricus

Envenoming profile

Local	**mild**	**moderate**	severe
Neuro	mild	moderate	severe
Haemo	**mild**	moderate	severe
Renal	mild	moderate	severe

See them

▶ Images Bamboo pit viper

Remember

Pit = no Indian ASV. Confirmed pit viper bites are managed supportively.

Sea Snakes

Hydrophis schistosus and other Hydrophiinae · Elapidae

Also known as: Both coasts of India; bites fishermen sorting nets, or bathers

Identify	Laterally flattened, **paddle-shaped tail**; small head; valvular nostrils; usually banded grey-blue. Beaked sea snake has a pointed snout.
Venom	Potent neurotoxins acting at **pre- and post-synaptic** sites; the pre-synaptic toxin (PLA_2) first releases then inhibits ACh release; the post-synaptic toxin binds nearly irreversibly to ACh receptors. PLA_2 myotoxins cause **generalised rhabdomyolysis**. Enzymes include acetylcholinesterase, hyaluronidase, leucine aminopeptidase, 5′-nucleotidase and phosphodiesterases.
Clinical	Bite painless; within 30 min–3 h: generalised **muscle pain, stiffness and tenderness** (trismus), then **dark brown/black urine** (myoglobinuria) by 3–8 h, flaccid paralysis including respiratory muscles; **hyperkalaemia** → cardiac arrest; AKI.
Tests	CK (very high), K^+, creatinine, urine for myoglobin (dipstick positive, no RBCs), ECG.
Treatment	**No Indian ASV** (sea snake antivenom from Australia/CSL if available). Ventilation, treat hyperkalaemia, alkalinised fluids for myoglobinuria, early haemodialysis — theoretically helpful in refractory cases because the neurotoxin is small (6–8 kDa).

Beaked sea snake

▶ Images

Hydrophis schistosus — paddle tail

Envenoming profile

	mild	moderate	severe
Local			
Neuro	**mild**	**moderate**	
Haemo			
Renal	**mild**	**moderate**	

Watch the potassium

In sea snake bites the killer is often hyperkalaemia from rhabdomyolysis — monitor ECG and K^+ hourly in the first hours.

Mildly Venomous & Rarely Dangerous Snakes

Colubridae (rear-fanged) and coral snakes

Also known as: Cat snakes (Boiga), vine snakes (Ahaetulla), red-necked keelback (Rhabdophis), coral snakes (Calliophis)

Cat snakes	*Boiga* spp. — vertical pupils, large head; may mimic saw-scaled viper. Bites cause mild local pain and swelling only.
Vine snakes	*Ahaetulla nasuta* — thin bright green with pointed snout and horizontal pupil; mild local effects.
Red-necked keelback	*Rhabdophis* — rear fangs with a potent prothrombin activator; prolonged chewing bites can cause serious **coagulopathy and AKI**. Indian ASV does not neutralise it.
Coral snakes	*Calliophis* — small, secretive elapids with bright red/blue undersides; bites are rare, can cause neurotoxicity; manage supportively and with ventilation if needed.
Approach	Observe 24 h with serial 20WBCT and neurological checks; ASV only if a clear Big-Four syndrome develops (snake may have been misidentified).

Common cat snake

▶ Images



Boiga trigonata — mildly venomous

Envenoming profile

	mild	moderate	severe
Local	■		
Neuro	■		
Haemo	■		
Renal			

See them

▶ Images Red-necked keelback ▶ Images Coral snake

Non-venomous Look-alikes

The snakes that are brought to the Emergency Medicine Department most often

Also known as: Rat snake (Dhaman), wolf snake, sand boa, python, keelbacks, trinket, bronzeback

Indian rat snake	*Ptyas mucosa* — long (2 m+), fast, yellow-brown with dark bars near the tail, large eyes. No hood. Often mistaken for cobra. Harmless; may bite repeatedly.
Common wolf snake	*Lycodon aulicus* — brown with single pale bands broader near the head, flat head, **krait mimic**; enters houses at night.
Sand boas	*Eryx* — thick, short with blunt tail; rough-scaled sand boa has **Russell's viper-like blotches**. Harmless (target of illegal "two-headed snake" trade).
Pythons	Indian rock python — large, blotched; labial heat pits; may cause deep lacerations and constriction injuries, not envenoming.
Keelbacks	Checkered keelback (water snake) — commonest bite near ponds and paddy; painful bites with teeth marks; no envenoming.
Approach	Even with a positive ID of a harmless snake, observe for at least **24 h** (identification may be wrong), give tetanus prophylaxis, clean the wound, reassure. **No ASV.**

Indian rat snake

▶ Images

Ptyas mucosa — non-venomous

Envenoming profile

Local	mild	moderate	severe
Neuro	mild	moderate	severe
Haemo	mild	moderate	severe
Renal	mild	moderate	severe

Reassure

Most patients arriving with a non-venomous bite are terrified; reassurance reduces vasovagal syncope and the 'panic' tachycardia that is often misread as envenoming.

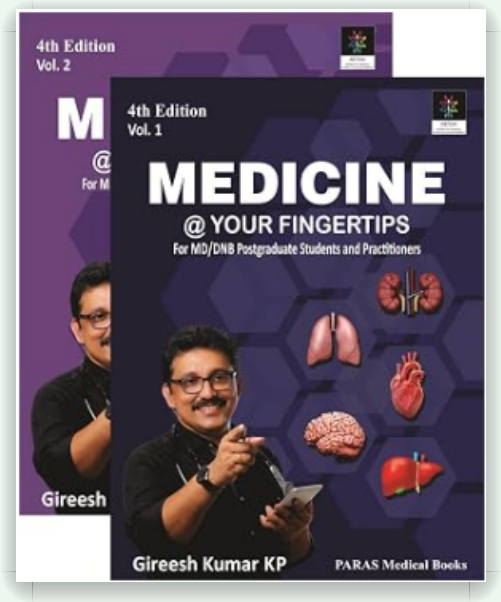
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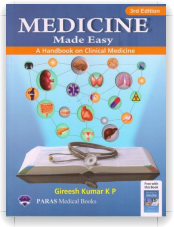
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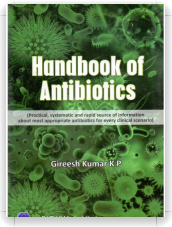
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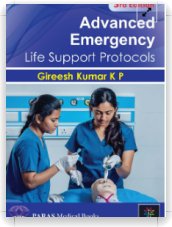
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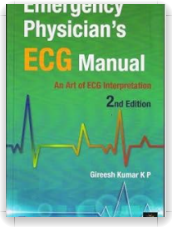
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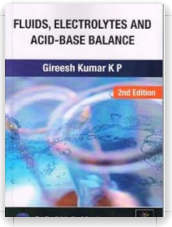
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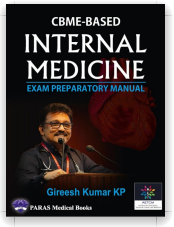
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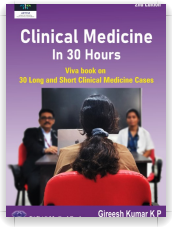
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CHAPTER 4 · VENOM

Venom and How It Harms

Composition, mechanisms, and the link between toxin and toxidrome

Snake venom is a modified saliva produced by specialised glands (homologous to parotid glands) and delivered through fangs. It is a cocktail of **20–100 proteins and peptides**, most of them enzymes; 90% of the dry weight is protein. The amount injected varies widely with the size of the snake, its last meal, the defensive or predatory nature of the bite and clothing — which is why the same species may give a dry bite or a fatal one.

Major toxin families

Toxin family	Snakes	Action	Clinical effect
Three-finger toxins — α-neurotoxins	Cobras, kraits, sea snakes, king cobra	Bind post-synaptic nicotinic ACh receptors (curare-like), competitive and reversible for short-chain toxins	Ptosis, ophthalmoplegia, bulbar and respiratory paralysis; responds to neostigmine
Three-finger toxins — cytotoxins (cardiotoxins)	Cobras (especially monocled)	Insert into cell membranes, lyse cells	Local swelling, blistering, necrosis; myocardial toxicity
Phospholipase A_2 (PLA_2)	All; β-bungarotoxin in kraits	Hydrolyse membrane phospholipids; pre-synaptic neurotoxicity, myotoxicity, haemolysis, anticoagulant effects	Irreversible pre-synaptic paralysis (krait, sea snake), rhabdomyolysis, AKI
Snake venom metalloproteinases (SVMP)	Vipers	Digest basement membrane of capillaries ("haemorrhagins"); activate prothrombin/factor X	Local and systemic bleeding, blistering, necrosis, capillary leak
Serine proteases & procoagulants	Russell's viper (factor X and V activators), saw-scaled viper (prothrombin activator — ecarin), HNPV	Activate the clotting cascade inside the vessel; thrombin-like enzymes cleave fibrinogen	Venom-induced consumption coagulopathy (VICC) — unclotted blood, low fibrinogen
C-type lectins, disintegrins	Vipers	Inhibit or activate platelets	Thrombocytopenia, platelet dysfunction
L-amino acid oxidase, hyaluronidase	All	Tissue damage; "spreading factor" speeds venom absorption	Rapid spread of swelling and venom
Natriuretic peptides, kallikrein-like enzymes	Kraits, vipers	Vasodilatation, natriuresis	Hypotension, hyponatraemia (krait)

Neuromuscular junction — where elapids strike

Two kinds of neurotoxicity. Pre-synaptic block explains why krait paralysis is prolonged and why early ASV (before the toxin enters the terminal) matters.

Coagulopathy — venom-induced consumption coagulopathy (VICC)

Procoagulant venom (factor X/V activators, prothrombin activator)

▼

Massive intravascular thrombin generation

▼

Fibrinogen consumed → microclots lysed (high D-dimer/ FDP)

▼

Blood will not clot — 20WBCT unclotted, INR unrecordable

▼

Bleeding where vessels are damaged by haemorrhagins (gums, GI, brain, fang marks)

- VICC is **not classical DIC**: there is little organ thrombosis and platelets may be normal; the defect is absence of fibrinogen.
- Clotting factors return only when venom is neutralised; the liver then needs about **6 hours** to rebuild fibrinogen — the basis of the "**6-hour rule**" for repeating ASV.
- FFP or cryoprecipitate given before ASV is simply consumed ("fuel to the fire").
- A subset (Russell's viper, HNPV) develop **thrombotic microangiopathy (TMA)** — haemolysis with schistocytes, thrombocytopenia and AKI.
- Anticoagulant PLA_2 and antiplatelet toxins add to bleeding.

Kidney, muscle, heart and vessels

Organ	Mechanisms
Kidney	Hypotension and hypovolaemia (bleeding, capillary leak) → ischaemic ATN; direct nephrotoxicity of venom; haemoglobinuria and myoglobinuria (pigment nephropathy); microthrombi/TMA; acute cortical necrosis; immune-complex nephritis (serum sickness)
Muscle	Myotoxic PLA_2 (sea snakes, some kraits, Russell's viper in Sri Lanka) → rhabdomyolysis, hyperkalaemia
Heart	Cardiotoxins, hyperkalaemia, coronary vasospasm/thrombosis, myocarditis → arrhythmias, low output, ECG changes
Vessels	Increased permeability (capillary leak) → parotid swelling, chemosis, pleural/peritoneal effusions, hypotension; vasodilators (bradykinin potentiation)
Endocrine	Haemorrhagic infarction of the **anterior pituitary** (Russell's viper) → acute adrenal crisis or chronic hypopituitarism

Remember — the toxin–toxidrome link

Elapid = paralysis (+ local necrosis with cobras). **Viper** = bleeding + swelling (+ kidney with Russell's viper). **Sea snake** = muscle + paralysis. ASV neutralises **circulating** venom; it cannot reverse damage already done to tissues, nerve terminals or kidneys.

CHAPTER 4 · VENOM IN THE LABORATORY AND THE PHARMACY

From Venom to Medicine

Snake toxins that became drugs and diagnostic tests

The same toxins that kill have taught us physiology and given us drugs. Understanding them helps students remember venom actions.

Venom source	Toxin / principle	Drug or test that resulted
Brazilian pit viper (*Bothrops jararaca*)	Bradykinin-potentiating peptides (inhibit ACE)	**Captopril** — the first ACE inhibitor; the whole class followed
Pygmy rattlesnake (*Sistrurus miliarius barbouri*)	Disintegrin barbourin (KGD motif, blocks GP IIb/IIIa)	**Eptifibatide** — antiplatelet in acute coronary syndromes
Saw-scaled viper (*Echis carinatus*)	Disintegrin echistatin (RGD motif)	Template for **tirofiban** (GP IIb/IIIa antagonist); **ecarin** clotting time for direct thrombin inhibitors
Russell's viper (*Daboia russelii*)	Factor X activator (RVV-X)	**Dilute Russell's viper venom time (dRVVT)** — test for lupus anticoagulant
Malayan pit viper (*Calloselasma*)	Thrombin-like enzyme (ancrod)	Ancrod — studied as a defibrinogenating agent in stroke
Lancehead pit vipers (*Bothrops atrox, B. moojeni*)	Thrombin-like enzymes (batroxobin, reptilase)	**Reptilase time** — distinguishes heparin effect from fibrinogen defects
Many-banded krait (*Bungarus multicinctus*)	α-bungarotoxin	Research tool that identified and counted **nicotinic ACh receptors** — key to understanding myasthenia gravis
Cone snails	ω-conotoxin MVIIA	**Ziconotide** — intrathecal analgesic for intractable pain
Gila monster (lizard)	Exendin-4	**Exenatide** — the first GLP-1 receptor agonist for diabetes
Medicinal leech	Hirudin	**Bivalirudin, lepirudin** — direct thrombin inhibitors; leech therapy in plastic surgery
Bee venom	Melittin, PLA_2	Venom immunotherapy extracts for Hymenoptera allergy

Remember — linking toxin to toxidrome

- ACE-inhibiting peptides → venom hypotension → captopril
- Disintegrins → platelet dysfunction → eptifibatide/tirofiban
- Factor X activators → VICC → dRVVT
- Thrombin-like enzymes → low fibrinogen → reptilase test
- Bungarotoxin → paralysis → ACh receptor research

Pearl — Russell's viper and the coagulation lab

The procoagulant that makes Russell's viper venom lethal is used every day in haematology laboratories to detect lupus anticoagulant — dilute Russell's viper venom time. A good viva answer to "Name a medical use of snake venom".

Venom research in India

Venom for Indian ASV comes largely from snakes caught and milked by the **Irula Snake Catchers' Industrial Cooperative Society** in Tamil Nadu. Indian research groups (venomics, recombinant antivenoms, region-specific antivenoms and point-of-care venom detection) are working to close the "beyond the Big Four" gap identified in recent studies.

CHAPTER 5 · CLINICAL FEATURES

Clinical Features and Toxidromes

Local and systemic envenoming according to the class of snake

Timeline after the bite

Time after bite	What to expect
Minutes	Fear, palpitations, sweating, hyperventilation, vasovagal faint (also in non-venomous bites); local pain and swelling with vipers and cobras; early anaphylaxis to venom (rare, in snake handlers)
15 min – 2 h	Cobra: ptosis and bulbar palsy may begin. Viper: swelling spreads, bleeding from fang marks, 20WBCT becomes unclotted
2 – 6 h	Systemic bleeding, hypotension, vomiting; krait: abdominal pain, ptosis; sea snake: myalgia and dark urine
6 – 24 h	Krait paralysis can still appear (up to 12–24 h); oliguria and AKI; compartment syndrome; blisters
Days	Local necrosis demarcates (cobra, viper), AKI peaks, secondary infection, serum sickness (5–12 days), pituitary failure (weeks–years)

Dry bites and non-envenomation

About 20–50% of venomous bites inject no venom. Fang marks with no local or systemic signs after **24 hours** of observation (serial 20WBCT and neurological checks) mean no envenoming. Do not give ASV "just in case".

Local envenoming

- Fang marks: one, two or more punctures, scratches, or none (krait); bleeding from marks suggests viper coagulopathy.
- Pain: burning, out of proportion (viper, cobra); absent in krait and sea snake bites.
- Swelling: starts within 15 min–2 h; progression is a sign of active venom.
- Blisters (bullae), bruising, lymphangitis, **tender enlarged draining lymph nodes** — an early sign of absorption.
- Necrosis: cobra (wet, foul-smelling, "skip" lesions), viper; may lead to gangrene and amputation.
- Compartment syndrome — rare but important (see Chapter 14).

Grading local swelling (useful for charting)	
Grade 0	No swelling
Grade 1	Swelling at bite site (hand/foot)
Grade 2	Up to the wrist/ankle
Grade 3	Up to the elbow/knee
Grade 4	Beyond elbow/knee, > half the limb
Grade 5	Whole limb and beyond onto trunk

Mark the edge of swelling with a pen and the time, and measure limb circumference at fixed points every 1–2 h.

Systemic envenoming by class — the toxidromes

Toxidrome	Symptoms and signs	Typical snakes
Neurotoxic	Heavy eyelids, **ptosis** (earliest objective sign), blurred/double vision, external ophthalmoplegia, facial weakness, dysphagia, pooling of saliva, nasal voice, regurgitation, dysarthria, **"broken-neck" sign** (neck-flexor weakness), descending limb weakness, paradoxical breathing, respiratory arrest; pupils may become dilated and unreactive; consciousness preserved until hypoxia	Cobra, krait, king cobra, sea snake, Russell's viper (South India/Sri Lanka)

Haemotoxic	Bleeding from fang marks and venepuncture sites, gums, epistaxis, subconjunctival haemorrhage, haemoptysis, haematemesis, malaena, haematuria, ecchymoses, menorrhagia/antepartum haemorrhage; **intracranial haemorrhage** (headache, vomiting, focal deficit, coma); unclotted 20WBCT	Russell's viper, saw-scaled viper, HNPV, pit vipers
Nephrotoxic	Loin pain, oliguria/anuria, dark or red urine, rising urea/creatinine, hyperkalaemia, acidosis, uraemic symptoms; may begin after a few hours or present days later	Russell's viper, HNPV, sea snake (myoglobin)
Myotoxic	Generalised muscle pain, tenderness and stiffness, trismus, dark brown urine, very high CK, hyperkalaemia	Sea snake; some kraits
Cardiovascular	Hypotension, shock, bradycardia or tachycardia, arrhythmias, ECG changes (ST-T, AV block), pulmonary oedema	Russell's viper, cobra, saw-scaled viper
Capillary leak	**Bilateral parotid swelling**, conjunctival oedema (**chemosis**), facial oedema, pleural effusion, hypoalbuminaemia, haemoconcentration, shock	Russell's viper
Endocrine	Acute: hypotension, hypoglycaemia, hyponatraemia (adrenal crisis); late: amenorrhoea, loss of libido and body hair, lethargy — hypopituitarism	Russell's viper
General	Nausea, vomiting, malaise, abdominal pain (krait), weakness, drowsiness, headache	All

CHAPTER 5 · SYNDROMIC APPROACH

Which Snake Was It? — The Syndromic Approach

When the snake is not seen, the clinical syndrome points to the likely species (adapted from WHO SEARO 2016)

#	Clinical syndrome	Most likely snake(s) in India	Indian ASV?
1	Local envenoming (swelling, blisters, bruising) **+ bleeding / unclotted blood**	Russell's viper, saw-scaled viper; in the Western Ghats also hump-nosed pit viper; pit vipers in hills/NE	Yes for Big-Four vipers; No for HNPV/ pit vipers
2	Syndrome 1 **+ shock or AKI**; ± **parotid swelling, chemosis**, pituitary failure	Russell's viper (HNPV in Kerala — AKI, TMA)	Yes (Russell's viper)
3	Local envenoming **+ descending paralysis**	Cobra (spectacled, monocled), king cobra	Yes (cobra); partial (king cobra)
4	**Paralysis with minimal or no local signs**; bite at night indoors, abdominal pain	Common krait and other kraits; bite in sea/ estuary — sea snake	Yes (common krait); No (sea snake)
5	Paralysis **+ generalised myalgia, dark brown urine**, AKI, hyperkalaemia	Sea snake; greater black krait; Russell's viper (South India/Sri Lanka) if also coagulopathic	No for sea snake
6	Paralysis **+ bleeding/unclotted blood**	Russell's viper (Tamil Nadu, Sri Lanka) — neurotoxic PLA_2; occasionally mixed bites	Yes
7	Local pain and swelling only, normal clotting, no systemic features	Pit viper, mildly venomous colubrid, dry/ partial viper or cobra bite	Not indicated unless progression

Bedside syndromic triage for the Big Four.

Grading severity of envenoming

Grade	Local	Systemic	Action
No envenoming (dry bite)	Fang marks only	None; 20WBCT clotted at 0, 6, 12, 24 h	Observe 24 h, tetanus, discharge with advice
Mild	Swelling limited to bite area, slow progression	None	Observe closely; ASV only if progression to criteria
Moderate	Swelling beyond the joint, progressing	Mild coagulopathy without bleeding, or early ptosis	ASV full loading dose
Severe	Swelling > half limb, rapid, necrosis	Spontaneous bleeding, shock, AKI, bulbar or respiratory paralysis	ASV full dose + resuscitation, ICU/ventilator/dialysis

Clues to severe envenoming (STG)

Rapid extension of swelling · early tender lymph nodes · early ptosis or weakness · breathlessness · early spontaneous bleeding · collapse or unconsciousness · passage of dark brown urine · snake identified as cobra or Russell's viper.

Mimics and confounders

Panic attack/hyperventilation (tingling, carpopedal spasm) · tourniquet ischaemia and pain · effects of herbal remedies, alcohol, cut-and-suck wounds · scorpion, centipede or insect sting · krait mimics: gastritis, organophosphate poisoning, Guillain–Barré, botulism, stroke, myasthenia.

Remember

Examine the whole patient: eyelids, eye movements, swallowing, neck flexion, breathing, gums, conjunctivae, urine colour, parotids, blood pressure — not only the bite site.

Typical time course by snake

Snake	Local signs begin	Systemic signs begin	Usual peak	Recovery with treatment
Spectacled cobra	Minutes	15 min – 6 h (ptosis)	2–8 h	Paralysis 24–48 h; necrosis weeks
Common krait	None	1–12 h (up to 24 h), often early morning	6–24 h	Ventilation 3–10 days
Russell's viper	15 min – 2 h	Coagulopathy 30 min – 6 h; AKI 6–72 h	24–72 h (AKI)	Clotting 6–24 h; kidney 2–4 weeks
Saw-scaled viper	15 min – 2 h	Coagulopathy 1–6 h	6–24 h	Clotting 6–24 h; recurrence possible
Hump-nosed pit viper	Minutes – hours	Coagulopathy 6–24 h; AKI 1–3 days	Days	Coagulopathy up to 3 weeks
Sea snake	None	Myalgia 30 min – 3 h; dark urine 3–8 h	6–24 h	Days to weeks

Pearl — the 24-hour rule

Because krait paralysis and viper coagulopathy can appear late, an apparently well patient must stay under observation with repeated 20WBCT and neurological checks for 24 hours. Most "sudden deaths" after early discharge are late-onset envenoming.

CHAPTER 6 · PRE-HOSPITAL

First Aid and Pre-hospital Care

"Do it R.I.G.H.T." — and do no harm on the way to ASV

The R.I.G.H.T. approach (national protocol)

Letter	Action
R — Reassure	Most bites are non-venomous or dry; panic increases heart rate and venom spread. Keep the victim calm and still.
I — Immobilise	Immobilise the **whole limb** with a splint or sling, as for a fracture, without blocking blood flow; avoid any movement of the limb; carry the victim rather than letting them walk.
G.H. — Go to Hospital	Immediately, to a facility with ASV (call **108/102**; helpline **15400** where available). If no ambulance, a motorbike with the victim between two people. The victim must not run, ride or drive.
T — Tell the doctor	Tell the doctor of any symptoms (ptosis, difficulty in swallowing, bleeding, passing less urine) that develop on the way; show the photograph of the snake if available.

DO

- Remove rings, watches, bangles, anklets, tight clothing and shoes from the bitten limb (swelling will follow).
- Lay the victim in the **recovery position** (left lateral) to protect the airway if vomiting or drowsy; nothing by mouth.
- Leave blisters intact; keep the bite site clean.
- Note the time of bite and the progression of symptoms.
- Take a photograph of the snake from a safe distance, if possible.
- **Pressure immobilisation bandage (PIB)** may be used by trained persons for suspected **neurotoxic (elapid) bites** when transport will take 30 min–3 h: a crepe bandage wound firmly (one finger should slip under it) over the bite and the whole limb, plus a splint.

DON'T

- **No tourniquets** or tight ligatures — they cause ischaemia, gangrene and a sudden venom surge on release.
- No cutting, incision, suction (mouth or pump), scarification, cautery, burning or electric shock.
- No "black stone", herbs, chemicals, ice or cryotherapy; do not wash the wound vigorously.
- No alcohol, aspirin or NSAIDs; no intramuscular injections.
- Do not waste time with faith healers or traditional practitioners.
- Do not try to catch or kill the snake.
- Do not inject ASV locally at the bite site.

Pressure immobilisation — the controversy

PIB was developed for Australian elapids. It is **not advised for viper bites** — it can worsen local necrosis and compartment pressure. Since the species is often unknown in India, the STG reserves PIB for trained personnel; splinting of the whole limb is the universal advice. If a PIB has been applied, remove it in hospital only after ASV has started and resuscitation equipment is ready.

Arrival with a tourniquet or ligature

Patient arrives with tourniquet/ligature(s)

▼

Check distal pulse and limb viability; secure IV line; draw blood for 20WBCT and labs

▼

Start ASV loading infusion; keep atropine–neostigmine, adrenaline and Ambu bag ready

▼

Release ligatures one at a time (if several, release all except the most proximal first) and watch for sudden collapse, paralysis or bleeding

▼

Remove the last ligature; assess for ischaemia and compartment syndrome

Transport and referral from the periphery

- A PHC/CHC with ASV should **give the loading dose before transfer** — ASV is the first and most important treatment; the 6-hour gap after the loading dose in viper bites gives time to transport.
- For neurotoxic bites give one atropine–neostigmine dose before transfer and send with an **Ambu bag and airway**, oxygen, and a trained person who can ventilate.
- Telephone the receiving centre to confirm ASV, ventilator and dialysis availability. Send a referral note with the time of bite, ASV batch and vials given, 20WBCT results and other treatment.

Guidelines

▶ Guideline STG Quick Reference ▶ Guideline NHSRC clinician module

CHAPTER 7 · EMERGENCY MEDICINE DEPARTMENT

Assessment in the Emergency Medicine Department

Rapid primary survey, focused history and the snakebite examination

Primary survey — the first 5 minutes

Step	Look for	Act
A — Airway	Pooled secretions, inability to swallow, stridor (angioedema), vomiting	Suction, recovery position, airway adjuncts, early intubation
B — Breathing	SpO_2, respiratory rate, paradoxical breathing, use of accessory muscles, single breath count, cyanosis	Oxygen, bag-mask ventilation, prepare ventilator
C — Circulation	BP, pulse, capillary refill, bleeding, urine output	Two large-bore IV lines (avoid femoral/jugular puncture in coagulopathy), fluids for shock
D — Disability	Ptosis, eye movements, pupils, neck flexion, limb power, GCS (unreliable when paralysed), glucose	Do not misinterpret paralysis as coma
E — Exposure	Bite site, swelling extent, ligatures, other wounds, bleeding sites	Mark swelling, remove jewellery, splint

Focused history — the five questions

1. **Where** on the body were you bitten? (show me)
2. **When** were you bitten — what time? Where were you (field, house, bed, water)?
3. **What** did the snake look like? Do you have a photo? Did it have a hood, bands, spots, hiss?
4. **What was done** — tourniquet, cuts, herbs, alcohol, injections elsewhere (ASV given? how many vials?)
5. **How are you feeling now** — pain, vomiting, abdominal pain, heavy eyelids, double vision, difficulty swallowing or breathing, bleeding, urine colour/quantity?

Also ask: previous snakebite and ASV, previous horse-serum products (ATS, ERIG), atopy/asthma, pregnancy, last menstrual period, chronic illness, current drugs (anticoagulants, antiplatelets, β-blockers), tetanus immunisation status, weight (children).

> **Pearl**
> "Was the person sleeping on the floor?" is the single most useful question when there is no bite mark and the patient has abdominal pain or ptosis in the early morning.

Examination checklist

System	Check
Bite site	Fang marks, bleeding, swelling grade and edge (mark with pen + time), blisters, necrosis, lymph nodes, limb pulses, compartment tension
Bleeding	Gums, nose, conjunctivae, skin (ecchymoses), venepuncture sites, vomit, stool, urine, vaginal bleeding; signs of intracranial bleed
Neurological	Ptosis (ask patient to look up for 60 s), eye movements and diplopia, pupils, facial weakness, mouth opening, tongue protrusion, swallowing (pooling of saliva), speech, **neck lift**, limb power and reflexes, **single breath count**, breath-holding time
Cardiovascular	Pulse, BP (lying and sitting), JVP, heart sounds, ECG monitor
Renal	Urine colour, catheter output hourly, loin tenderness
Capillary leak	Parotid swelling, chemosis, facial oedema, effusions
Muscles	Tenderness, pain on passive stretch, trismus

Bedside respiratory tests

Single breath count (SBC)

Patient takes a deep breath and counts aloud ("one, two, three…") at about 2 counts per second without taking another breath. Normal ≥ 30–50. **< 20 suggests respiratory weakness**; falling SBC needs ventilatory support.

Neck-lift test

Lying supine, the patient lifts the head off the bed and holds it. Inability to lift the neck against gravity ("broken-neck sign") predicts impending respiratory failure — **refer/ventilate**.

Breath-holding time

Normal > 45 s. Less than 45 s, inability to complete a sentence in one breath, SpO_2 < 90% or saliva pooling are indications for assisted ventilation.

Remember

Admit **every** snakebite for at least **24 hours**, even if the snake was "harmless" or the patient looks well. Envenoming from kraits and some vipers can be delayed. If ASV is given, observe for at least 48 hours.

CHAPTER 8 · INVESTIGATIONS

Investigations in Snake Envenomation

The 20-minute whole blood clotting test and the laboratory panel

The 20-minute whole blood clotting test (20WBCT)

1. Take a **new, clean, dry, plain glass** test tube (not plastic, not washed with detergent).
2. Draw a few mL of venous blood and place **2 mL** in the tube.
3. Leave it **undisturbed** at room temperature for **20 minutes** (do not shake or tilt in between).
4. After exactly 20 min, **tilt the tube once to 45°**.
5. If the blood is still liquid → **"Not clotted"** = coagulopathy (venom-induced consumption coagulopathy) → ASV indicated.
6. If a firm clot forms → **"Clotted"**.

Report as "clotted" or "not clotted" — never "positive/negative", which confuses staff.

When to do the 20WBCT	
On admission	Before ASV (also send first sample for grouping & cross-match)
First 3 h	Hourly (detects late-onset coagulopathy)
Then	Every 6 h for 24 h
After ASV	6 h after each dose — guides repeat ASV

Pitfalls of the 20WBCT

- Plastic tubes or tubes washed with detergent and water may fail to activate clotting → false "not clotted". Glass that has been reused may give false "clotted" results.
- Shaking or repeated tilting breaks the clot. Low ambient temperature slows clotting.
- Severe anaemia may give a small, friable clot. Very early sampling may miss evolving coagulopathy.
- When in doubt, run the test in duplicate with a **control sample from a healthy person** in an identical tube.
- A "clotted" 20WBCT with obvious systemic bleeding still needs ASV — bleeding is an indication in itself.

Laboratory panel

Test	Expected abnormality	Significance
CBC	Neutrophil leucocytosis; falling Hb (bleeding, haemolysis); thrombocytopenia; high haematocrit (capillary leak)	WBC > 20,000/mm³ suggests severe envenoming; platelets < 50,000 + schistocytes → TMA
Peripheral smear	Schistocytes (fragmented RBCs), spherocytes	Microangiopathic haemolysis (Russell's viper, HNPV)
PT/INR, aPTT	Prolonged or unrecordable	Recheck 6-hourly after ASV until stable; target INR < 2 after products
Fibrinogen, D-dimer/FDP	Low fibrinogen, very high D-dimer	Confirms consumption coagulopathy; guides cryoprecipitate
Urea, creatinine, electrolytes	Rising creatinine; hyperkalaemia; hyponatraemia (krait)	AKI, dialysis decisions; check daily or 12-hourly
Urine	Dipstick blood +; RBCs (haematuria) vs no RBCs (Hb/myoglobin); proteinuria; red or brown colour	Pink supernatant after centrifuging = haemoglobinuria; clear supernatant + RBCs in deposit = haematuria; brown with dipstick + and no RBCs = myoglobinuria
CK	Raised (often > 10,000 U/L in sea snake bite)	Rhabdomyolysis, compartment syndrome

LFT, LDH, bilirubin	Raised LDH and indirect bilirubin, low haptoglobin	Haemolysis; transaminases ↑ in severe envenoming
ABG & lactate	Respiratory acidosis (paralysis), metabolic acidosis (shock, AKI), hypoxaemia	Ventilation and dialysis decisions
Blood glucose	Hypoglycaemia (adrenal/pituitary failure), hyperglycaemia (stress)	Check in shocked or drowsy patients
Blood group & cross-match	—	Use the **pre-ASV sample**; venom and ASV can interfere later
ECG	Sinus bradycardia/tachycardia, ST-T changes, AV block, tall T waves (K^+)	Myocardial toxicity, hyperkalaemia; monitor continuously
Chest X-ray	Pulmonary oedema, effusions, aspiration, haemorrhage	Capillary leak, fluid overload, aspiration in bulbar palsy

Imaging and special tests

- **Ultrasound/Doppler** of the bitten limb — subcutaneous oedema vs true compartment involvement, arterial flow; helps avoid unnecessary fasciotomy.
- **Compartment pressure** measurement (needle manometry) before fasciotomy (> 40 mmHg).
- **CT brain** for headache, altered sensorium or focal signs (intracranial haemorrhage, infarct) — after correcting coagulopathy if possible.
- **Ultrasound abdomen** for renal size and echogenicity (cortical necrosis), effusions, retroperitoneal bleed.
- **Nerve conduction/repetitive stimulation** — decrement in neuromuscular block; rarely needed.
- **Venom detection kits (ELISA)** — research tool in India; not used for treatment decisions.
- **Hormones** (cortisol, TSH/FT4, LH/FSH, prolactin) when hypopituitarism suspected after Russell's viper.

Monitoring chart (minimum)

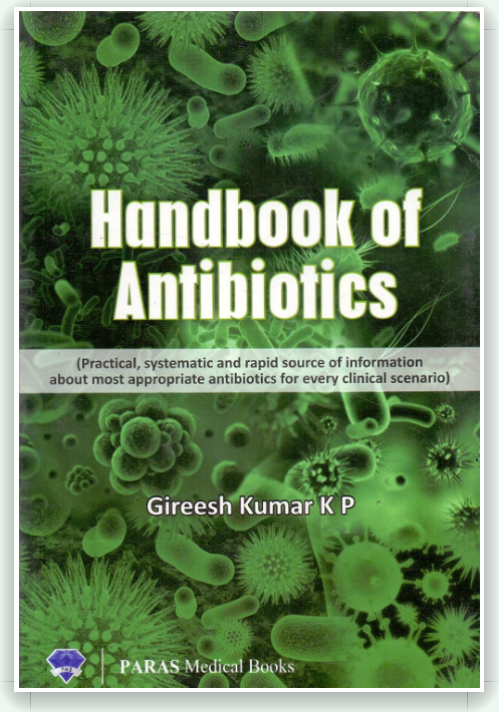
Parameter	Frequency
Pulse, BP, RR, SpO_2	Every 15 min for 2 h after ASV starts, then hourly
Ptosis, SBC, neck lift	Hourly (neurotoxic) for 24 h
Swelling edge & circumference	Every 1–2 h
Urine output	Hourly (catheter if oliguric/ shock)
20WBCT	Hourly × 3, then 6-hourly × 24 h
Creatinine, K^+	Daily (12-hourly if AKI)
CBC, PT/INR	6–12-hourly till stable

> **Pearl**
> After antivenom therapy, laboratory values should be rechecked every 6 hours until the patient is clinically stable.

ALSO BY THE AUTHOR

Dr. Gireesh Kumar K.P.

Books for the practising doctor



Handbook of Antibiotics

Practical, systematic and rapid source of information about the most appropriate antibiotics for every clinical scenario

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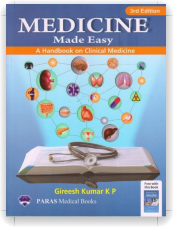
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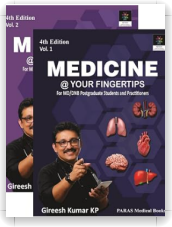
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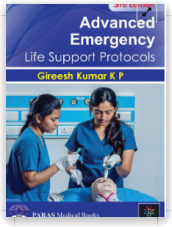
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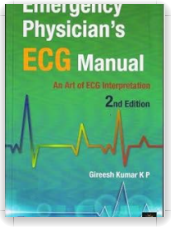
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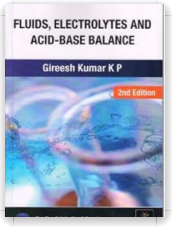
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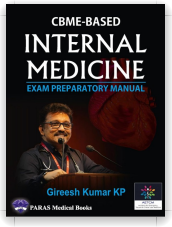
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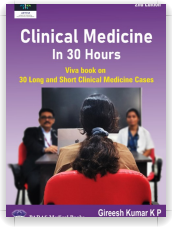
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CHAPTER 9 · ANTIVENOM

Anti-Snake Venom (ASV) — The Product

What is in the vial, Indian preparations, storage and indications

What is Indian ASV?

- **Polyvalent (polyspecific)** antivenom raised in **horses** (equines) hyper-immunised with venoms of the **Big Four**: spectacled cobra, common krait, Russell's viper and saw-scaled viper — mostly from venom collected by the Irula snake catchers' cooperative in Tamil Nadu.
- Purified, **enzyme-refined (pepsin-digested) $F(ab')_2$** immunoglobulin fragments; preservative phenol or cresol (not more than 0.25%).
- Available as **lyophilised powder** (10 mL vial, reconstituted with the 10 mL sterile water supplied) or as **liquid** (10 mL vial). Monovalent ASV is not made in India.
- Potency is defined by the Indian Pharmacopoeia — the **minimum** neutralising capacity per mL (below). Each 10 mL vial therefore neutralises about **6 mg of Russell's viper venom**; a Russell's viper injects on average 63 mg (range 5–147 mg), which is the basis of a starting dose of about **10 vials**.

Venom	Neutralised by 1 mL (IP minimum)	By one 10 mL vial
Indian cobra (*Naja naja*)	0.60 mg	6.0 mg
Common krait (*B. caeruleus*)	0.45 mg	4.5 mg
Russell's viper (*D. russelii*)	0.60 mg	6.0 mg
Saw-scaled viper (*E. carinatus*)	0.45 mg	4.5 mg

Pearl

Children receive the **same dose as adults** — the snake injects the same amount of venom whatever the size of the victim; small bodies simply receive a higher concentration.

ASV preparations available in India

Manufacturer (examples)	Form	Storage	Notes
Haffkine Bio-Pharmaceutical Corporation, Mumbai (Govt. of Maharashtra)	Lyophilised polyvalent, 10 mL + sterile water	Cool, dark place; lyophilised form tolerates higher room temperature	"Snake Antivenin I.P." — enzyme-refined equine globulins; also makes scorpion antivenom
Bharat Serums and Vaccines, Mumbai	Lyophilised and liquid	Liquid 2–8 °C, do not freeze	"Snake Venom Antiserum I.P."
VINS Bioproducts, Hyderabad	Lyophilised and liquid	As per label	Large supplier to States and export
Premium Serums & Vaccines, Pune	Lyophilised (combipack with water)	As per label	Some labels state potency above IP minimum
Other licensed makers	Lyophilised/liquid	As per label	Serum Institute of India, Central Research Institute Kasauli, King Institute Chennai and others have produced ASV; check current State supply

Brand names, potency claims and availability change; always read the insert of the batch in hand. Lyophilised ASV has a longer shelf life (often ~5 years) and is preferred where cold chain is unreliable; liquid ASV needs refrigeration (2–8 °C) and has a shorter shelf life (~2 years). ▶ Product

Indications for ASV (national STG / WHO SEARO)

Give ASV if ANY of these

- **Haemostatic abnormality**: spontaneous systemic bleeding (gums, epistaxis, haematuria, haemoptysis, GI, intracranial) or **non-clotting 20WBCT**, or prolonged PT/INR, or thrombocytopenia < 100,000/mm³.
- **Neurotoxic signs**: ptosis, external ophthalmoplegia, bulbar or respiratory paralysis.
- **Cardiovascular**: hypotension, shock, arrhythmia, abnormal ECG.
- **AKI**: oliguria/anuria, rising creatinine — *with* evidence of ongoing venom activity.
- **Haemoglobinuria/myoglobinuria**, other evidence of intravascular haemolysis or rhabdomyolysis.
- **Local envenoming**: swelling involving **more than half the bitten limb** (without a tourniquet) within 48 h, swelling after bites on digits, **rapid extension** of swelling, or **tender enlarged draining lymph nodes**.

Do NOT give ASV for

- Fang marks or a "history of bite" only, or pain alone.
- Purely local, slowly progressing or old swelling with normal 20WBCT.
- Dry bites after 24 h observation.
- **Sea snake bites**, and **confirmed** green pit viper or hump-nosed pit viper bites — Indian ASV has no antibodies against them.
- AKI presenting days later with a **clotted** 20WBCT — treat the kidney, not with ASV.

Contraindications

There is **no absolute contraindication** to ASV in a patient with life-threatening envenoming. Previous reaction to equine serum or severe atopy means higher risk — give ASV with adrenaline ready, not withhold it.

How late can ASV help?

ASV is most effective when given early, but it is indicated **as long as signs of systemic envenoming persist** — coagulopathy has been corrected by ASV given days after the bite. It cannot reverse established necrosis, renal tubular necrosis or pre-synaptic nerve damage.

CHAPTER 9 · ADMINISTRATION

Giving ASV — Test Dose, Premedication, Reconstitution and Dilution

Step-by-step preparation at the bedside

No test dose

Skin or conjunctival sensitivity tests are **not recommended**. They do not predict early or late reactions, delay treatment by up to 30 minutes and may themselves sensitise the patient. Some product inserts still mention them — national guidelines over-rule this.

Premedication — what the guidelines say

STG (India 2016): adrenaline **0.25 mg of 0.1% (1:1000) solution SC** just before ASV (children **0.005 mg/kg**), except in known hypertension, cardiovascular disease or BP > 140/90; draw up adrenaline 0.5 mg in two syringes ready for IM use. **WHO SEARO:** low-dose SC adrenaline may be considered where reaction rates are high; **antihistamines and hydrocortisone have not been shown to prevent reactions**.

Table 9.1 — Steps for reconstitution and dilution of ASV

Step	What to do	Why / caution
1	Confirm the indication; record time of bite, weight, BP; secure a dedicated IV line (avoid the bitten limb); keep **adrenaline, oxygen, Ambu bag, suction and intubation kit** at the bedside	Reactions occur within 10–180 min; be ready before you start
2	Check label, batch number and expiry; liquid ASV must be clear to slightly opalescent and colourless/pale yellow	Discard if turbid, discoloured or with particles
3	Reconstitute each lyophilised vial with the **10 mL sterile water for injection** supplied (or normal saline)	Each vial = 10 mL ASV
4	**Swirl gently** or roll between the palms until dissolved (a few minutes) — **do not shake**	Shaking causes foaming and protein denaturation
5	Draw up the loading dose (e.g. 10 vials = 100 mL)	Never give an incomplete dose
6	**Dilute** in isotonic fluid — **normal saline or 5% dextrose**, **5–10 mL/kg** (adults usually 200–500 mL; children keep the running bottle ≤ 200 mL; oliguric or fluid-overloaded patients — smaller volume via syringe/infusion pump)	Larger dilution is easier to titrate; volume must suit the patient's fluid status
7	Start at a slow rate for the first 10–15 min with the doctor at the bedside, and observe; if no reaction, increase to give the full loading dose over **30–60 minutes**	Most early reactions appear in the first 10–30 min
8	**Alternative:** slow IV push of reconstituted (undiluted) ASV at **not more than 2 mL/min** (≈ 1 vial over 5 min); or 1 vial diluted in 10 mL saline over 10 min	Useful when infusion sets/pumps are not available
9	Monitor pulse, BP, RR, SpO_2, skin every **5 min for 30 min**, then every 15 min for 2 h	Early anaphylaxis
10	Never give ASV **IM, SC or into the bite site**; never mix with other drugs in the same line	IM ASV is poorly absorbed and causes haematomas in coagulopathy

Preparing a 10-vial loading dose.

Remember — ASV is a full-dose drug

Neither a test dose nor a "trial of two vials". Calculate, prepare and give the **whole loading dose**; then reassess at fixed time points (1 h for neurotoxic, 6 h for haemotoxic).

CHAPTER 9 · DOSING

ASV Dose — Minimum, Repeat and Maximum in Each Type

National STG regimens with WHO SEARO notes

Situation	Initial (loading) dose	Repeat dose	Usual maximum
Neurotoxic (cobra, krait)	**10 vials** IV over 30 min (–1 h)	If no improvement or worsening at **1 hour** → second dose of **10 vials**. A repeat may be needed after 2–3 h if neurotoxicity relapses (delayed absorption)	**20 vials**; beyond this concentrate on ventilation
Haemotoxic — STG low-dose infusion	**10 vials** (Russell's viper) or **6 vials** (saw-scaled viper) over 30 min	**2 vials every 6 h** as infusion in 100 mL NS until clotting time normalises, or for 3 days, whichever is earlier	Usually 10–30 vials in total
Haemotoxic — STG high-dose intermittent bolus	**10 vials** over 30 min	**6 vials every 6 h** as bolus until clotting time normalises and/or local swelling subsides	Reassess after **30 vials**
Haemotoxic — WHO / 2009 national protocol	**8–10 vials** over 1 h	20WBCT 6 h after the dose; if not clotted, repeat **5–10 vials** (half to one full dose) every 6 h until clotted	Rarely > 25–30 vials
Brisk ongoing bleeding	As above	Repeat dose within **1–2 hours** without waiting 6 h	As above
Life-saving surgery needed (e.g. intracranial haematoma, obstetric emergency)	Up to **30 vials** may be given initially	Guided by coagulation	—
Children	**Same dose as adults** — dilute in 5–10 mL/kg; running bottle ≤ 200 mL		Same
Pregnancy	**Same dose**; ASV is indicated for the mother's sake; involve obstetrician; fetal monitoring		Same
Local envenoming only (meets criteria)	Single loading dose (8–10 vials); no routine repeat for swelling alone		—
Late presentation (days) with AKI	Give ASV only if 20WBCT is **not clotted** or bleeding continues; otherwise treat AKI		—

Minimum effective dose — the logic

- There is no "minimum test" quantity: the smallest recommended **full** dose is the STG low-dose regimen — **6 vials** for confirmed saw-scaled viper and **10 vials** for Russell's viper or neurotoxic bites.
- Low-dose infusion has been shown to be as effective as high-dose bolus in Indian trials and saves scarce ASV; the high-dose regimen is used for severe envenoming or poor response.
- A dose that fails to restore clotting at 6 h is **repeated**, not increased step by step.

When to stop and rethink

If **30 vials** have been given and coagulopathy persists, ask: Is this a non-Big-Four snake (HNPV, Sind krait, sochureki saw-scaled viper)? Is there another cause (sepsis, liver failure, TMA)? Is the ASV batch poor? Is systemic bleeding actually present? Then use **FFP 10–15 mL/kg, cryoprecipitate** (fibrinogen, factor VIII) or fresh whole blood.

Landmark article (J Forensic Leg Med 2020;73:101996)

Comparison of two anti-snake venom protocols in hemotoxic snake bite: a randomized trial. Sagar BKP, Bammigatti C, Kadhiravan T, et al. *J Forensic Leg Med 2020;73:101996*.

Message: In 140 haemotoxic snakebite patients (JIPMER), the national protocol (100 mL then 100 mL 6-hourly) did not reduce mortality or morbidity compared with a lower-dose protocol (70 mL then 30 mL 6-hourly); about 12% had persistent coagulopathy until death — more ASV is not always better.

▶ Read article

Signs that ASV is working

Feature	Expected response
Blood pressure	Improves within 30–60 min
Spontaneous bleeding	Stops within 15–30 min
Coagulation	20WBCT clots by 6 h (liver re-synthesis); sometimes 12–24 h
Post-synaptic paralysis (cobra)	Begins to improve within 30 min–hours; full recovery 24–48 h
Pre-synaptic paralysis (krait, sea snake)	Little immediate change; slow recovery over days–weeks
Haemolysis/rhabdomyolysis	Urine clears over hours–days (red urine may persist without needing more ASV)
Local swelling	Stops spreading; resolves over days (ASV does not reverse existing necrosis)

Recurrence

Coagulopathy may **recur** 24–48 h after initial correction (venom depot at the bite site, shorter half-life of $F(ab')_2$), especially after saw-scaled viper bites. Continue 6-hourly 20WBCT for 24 h after the first clotted result; re-treat if it recurs.

▶ Watch — AETCM video lectures

▶ AETCM video 1 ▶ AETCM video 2 ▶ AETCM video 3

Read the protocols

▶ Guideline STG 2016 (full) ▶ Guideline WHO SEARO 2016 ▶ Paper Indian protocol highlights

CHAPTER 9 · QUESTIONS DOCTORS ASK

ASV — Frequently Asked Questions

Practical answers for the night shift

Question	Answer
Should I wait for the 20WBCT result before giving ASV?	No, if there is obvious spontaneous systemic bleeding or neurotoxicity — start ASV. For local swelling alone, the 20WBCT (20 minutes) helps decide.
The snake was not seen. Can I still give ASV?	Yes. Treat the syndrome (Chapter 5). Indian polyvalent ASV covers the Big Four, which cause most envenoming.
The patient was bitten 3 days ago. Is ASV useless now?	Not if there is ongoing coagulopathy or bleeding — ASV can still correct it. If clotting is normal and only AKI remains, ASV is not needed.
Can I give ASV intramuscularly when IV access fails?	No. Persist with IV access (external jugular, intra-osseous in children/ emergencies). IM ASV is poorly absorbed and dangerous in coagulopathy.
How fast can I give the loading dose?	Over 30–60 minutes after a slow start; in neurotoxic bites the STG gives 10 vials over 30 minutes.
Is the dose different for a 10 kg child?	No — same number of vials; reduce only the volume of diluent (5–10 mL/kg, running bottle ≤ 200 mL).
Should I give hydrocortisone and an antihistamine routinely before ASV?	They have not been shown to prevent reactions. The STG recommends low-dose SC adrenaline (0.25 mg) unless contraindicated. Always keep adrenaline 0.5 mg IM drawn up.
The patient reacted to the first vials — should I stop ASV?	Treat the reaction (adrenaline IM), then restart slowly and complete the dose. Severe envenoming outweighs the risk.
Local swelling is increasing after 10 vials — should I repeat?	Swelling alone after an adequate dose is not an indication for routine repeat. Repeat if clotting is still abnormal or systemic signs persist; reconsider if swelling is very rapid and threatening the limb.
The urine is still red after the blood has clotted. More ASV?	No. Haemoglobin/myoglobin and RBCs may take days to clear; manage fluids and kidney.
What if I have only 5 vials?	Give what you have **and** arrange transfer immediately to a centre with ASV; do not let a partial dose create false security. Inform the receiving hospital.
Expired ASV — can I use it?	Only in an emergency when no in-date ASV is available, if the solution is clear; lyophilised ASV retains much potency beyond expiry. Record the batch.
Does a second snakebite years later need a different approach?	Same indications and dose; higher risk of reactions — be prepared (Chapter 10.1).
Does ASV help a confirmed hump-nosed pit viper bite?	No — Indian ASV has no activity against HNPV venom. Treat supportively.
Can ASV be given with blood or FFP in the same line?	Use a separate line; never mix with other drugs or products.
Is ASV safe in pregnancy and breastfeeding?	Yes — envenoming is far more dangerous to the fetus than ASV. Give the full dose.

Remember

Adequate ASV, given early, intravenously, in full dose, with adrenaline at hand, and repeated by the clock (1 h neurotoxic, 6 h haemotoxic) — that is the whole art.

CHAPTER 10 · ADVERSE REACTIONS

ASV Reactions — Anaphylaxis and Other Complications of ASV

Early, pyrogenic and late (serum sickness) reactions, with doses

Equine ASV causes adverse reactions in **20–80%** of recipients depending on the product and dose; most are mild, but severe anaphylaxis can kill. They are **not a reason to withhold ASV** from a patient with envenoming — they are a reason to be prepared.

Types of reaction

Type	Timing	Features	Mechanism
Early (anaphylactic / anaphylactoid)	10–180 min after starting (most in first 30 min)	Itching, **urticaria**, dry cough, flushing, nausea, vomiting, abdominal colic, diarrhoea, tachycardia, fever. Severe: **hypotension, bronchospasm, angioedema** (lips, tongue, larynx), collapse	Mostly complement activation by IgG aggregates (non-IgE); IgE-mediated in previously sensitised
Pyrogenic (endotoxin)	1–2 h	Chills, rigors, high fever, vasodilatation, hypotension; febrile convulsions in children	Pyrogen contamination of product
Late (serum sickness)	1–12 days (mean ~7)	Fever, itching, recurrent urticaria, arthralgia/arthritis, myalgia, lymphadenopathy, periarticular swelling, mononeuritis multiplex, proteinuria with immune-complex nephritis; rarely encephalopathy	Type III immune-complex reaction

Management of early reactions (STG doses)

1. Stop the ASV infusion temporarily

▼

2. Airway, high-flow oxygen; lie flat with legs raised (sitting if breathless)

▼

3. Adrenaline 0.5 mg IM (0.5 mL of 1:1000) into the anterolateral thigh/deltoid
Children **0.01 mg/kg** IM (max 0.5 mg)
Repeat every 5–15 min if no improvement

▼

4. Fresh IV line with normal saline; fluid bolus 20 mL/kg for hypotension

▼

5. Chlorpheniramine 10 mg IV (children 0.2 mg/kg) — for itching/urticaria

▼

6. Hydrocortisone 100–200 mg IV (children 2–4 mg/kg) — slow onset, prevents late features

▼

7. When the reaction has settled, **restart ASV slowly** for 10–15 min under close observation, then return to the normal rate and complete the dose

Refractory anaphylaxis

Persistent hypotension after 2–3 IM doses and fluids: **adrenaline infusion** 0.05–0.5 µg/kg/min (e.g. 1 mg in 100 mL NS titrated) via a large vein with monitoring; nebulised salbutamol for bronchospasm; glucagon 1–2 mg IV if on β-blockers; consider intubation early for angioedema.

Pearl — IV adrenaline

Intravenous bolus adrenaline is only for cardiac arrest or peri-arrest by experienced clinicians (50–100 µg aliquots in adults). IM adrenaline is safe even in coagulopathy — use a thin needle and apply pressure.

Pyrogenic reactions

Cool the patient (tepid sponging, fans), **paracetamol** 15 mg/kg (adults 500–1000 mg) orally or IV, IV fluids for hypotension; treat any concurrent anaphylaxis as above. Continue ASV once stable.

Late serum sickness

- **Chlorpheniramine 2 mg six-hourly** (adults) for 5 days; children 0.25 mg/kg/day in divided doses.
- If no response in 24–48 h: **prednisolone 5 mg six-hourly** (adults) for 5 days; children 0.7 mg/kg/day in divided doses.
- Warn every patient at discharge to return with fever, rash or joint pains in the next 1–2 weeks.

CHAPTER 10 · HIGH-RISK PATIENTS

High-risk Patients, Desensitisation and Other ASV Problems

Atopy, previous equine serum, repeat bites and ASV shortage

Who is at high risk of ASV reactions?

- Previous reaction to ASV or other **equine sera** (anti-tetanus serum, equine rabies immunoglobulin, anti-diphtheria serum).
- Strong history of **atopy** — asthma, eczema, allergic rhinitis, food allergy; occupational exposure to horses.
- Previous ASV for an earlier bite (repeat bites are common in farmers and snake rescuers).

How to give ASV to a high-risk patient

- Give ASV **only if there are clear signs of systemic envenoming**.
- Pre-treat: **hydrocortisone 200 mg IV** and **chlorpheniramine 10 mg IV** (children 0.2 mg/kg), plus SC adrenaline 0.25 mg if no contraindication.
- Start in a resuscitation bay with adrenaline drawn up, airway equipment and a senior doctor present.
- Start very slowly; increase the rate gradually.

Desensitisation (rarely needed)

For a patient who develops severe anaphylaxis with shock after very small volumes of ASV: change the ASV **batch** if possible; premedicate with hydrocortisone 100 mg IV and adrenaline 0.5 mL (1:1000) SC/IM ± promethazine; then give ASV in serial dilutions — 1 mL ASV in 10 mL saline (solution A), and further 1:10 dilutions (1:100, 1:1000, 1:10,000) — starting with the most dilute and stepping up every 15 min while monitoring, then the full infusion.

Other complications and pitfalls of ASV

Problem	Prevention / management
Fluid overload and pulmonary oedema	Use smaller dilution volumes in oliguric, elderly, cardiac patients and small children; infusion pump; monitor chest and SpO_2
Volume of repeat doses in AKI	Concentrate (e.g. 10 vials in 100 mL) and give over 30 min by pump
Wrong route	Never IM, SC, intra-lesional — haematomas in coagulopathy and poor effect
Under-dosing	"2 vials to see the response" fails; always a full loading dose
Over-dosing	Giving ASV every few hours for persistent swelling or red urine alone wastes ASV and adds reactions — follow the 6-hour/20WBCT rule
Interference with cross-match	Send blood grouping and cross-match on the **pre-ASV** sample
Expired or frozen ASV	Expired lyophilised ASV may retain potency and can be used in an emergency if nothing else is available and it dissolves clear; never use frozen liquid ASV
Shortage	Use the STG low-dose regimen; give ASV only for valid indications; refer early to a centre with stock; States maintain buffer stocks (NAPSE)

Don't

Do not "pre-medicate and hope": premedication does not replace bedside vigilance. Do not abandon ASV after a reaction — the envenoming that needed ASV is still there.

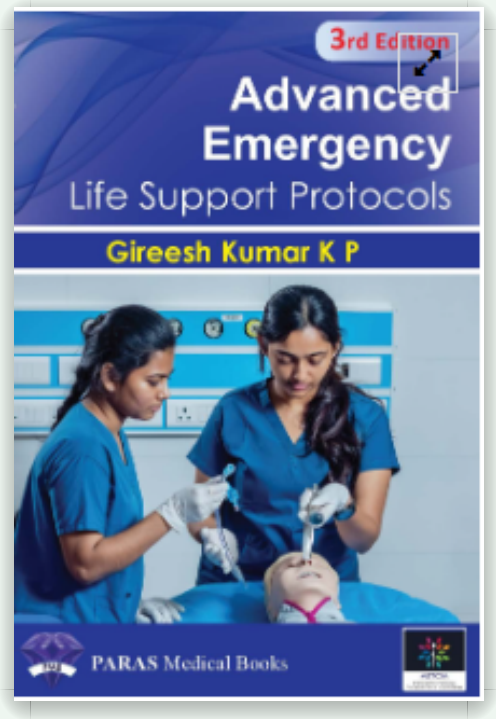
Recognising anaphylaxis

▶ Article Anaphylaxis — recognition and treatment

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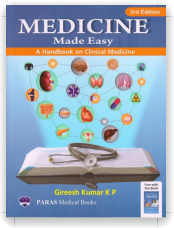
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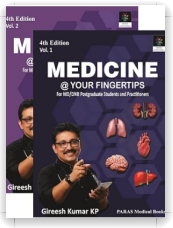
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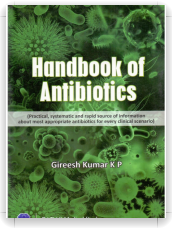
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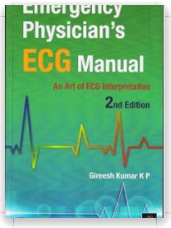
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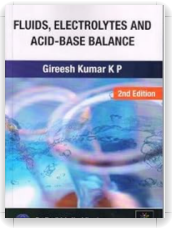
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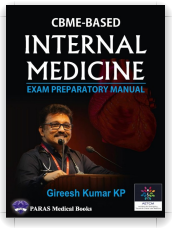
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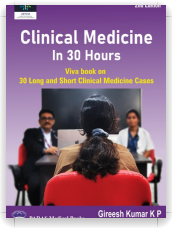
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CHAPTER 11 · PROTOCOL

Treatment Protocol — Neurotoxic Envenomation

ASV, the atropine–neostigmine trial, airway and ventilation

Step-by-step protocol

Neurotoxic signs — ptosis, ophthalmoplegia, bulbar palsy, neck weakness, breathlessness

▼

Airway & oxygen; suction; recovery position; SpO_2, SBC, neck lift

IV line; blood for 20WBCT, CBC, Na^+, K^+, creatinine, CK, ABG

▼

ASV 10 vials IV over 30 min (–1 h) + **atropine–neostigmine trial** (especially if cobra suspected)

▼

Reassess at **1 hour**: ptosis, SBC, neck lift, swallowing, SpO_2

▼

Improving → continue observation; neostigmine maintenance if AN-responsive

Same or worse → **second 10 vials**; ventilate if criteria met

▼

Relapse after 2–3 h → may repeat ASV once; **maximum 20 vials** — then supportive ventilation until recovery

The atropine–neostigmine (AN) trial

Drug	Adult	Child
Atropine (first)	**0.6 mg IV** stat	0.02 mg/kg IV (min 0.1 mg) — WHO; STG lists 0.05 mg/kg
Neostigmine (then)	**1.5–2.0 mg IV/IM** (STG: 1.5 mg IV)	0.025–0.08 mg/kg IM — WHO; STG 0.04 mg/kg IV
or Edrophonium	10 mg IV (if available)	0.25 mg/kg
Maintenance if positive	Neostigmine **0.5 mg every 30 min** with atropine (STG: for 5 doses, then taper at 1, 2, 6 and 12 h)	0.01 mg/kg every 30 min with atropine
Atropine infusion (WHO)	0.6 mg over 8 h	0.02 mg/kg over 8 h

Fixed-dose neostigmine–glycopyrrolate (2.5 mg + 0.5 mg) can be used instead of separate drugs.

Interpreting the trial

- Measure ptosis (gap between lids), SBC and neck lift before and 30–60 min after.
- **Positive**: ≥ 50% improvement in ptosis/ophthalmoplegia or SBC within 1 h → continue neostigmine maintenance (post-synaptic toxin, usually cobra).
- **Negative** after 3 doses → stop; suspect krait (pre-synaptic).
- **Stop** AN if full recovery, fasciculations, bradycardia, excessive secretions or bronchospasm.
- Give one AN dose **before transfer** of any neurotoxic patient.

Caution

Neostigmine increases salivary and bronchial secretions — always give atropine first and keep suction ready. AN does **not** replace ASV or ventilation.

Landmark article (PLoS Negl Trop Dis 2026;20(9):e0014736)

Adjunctive varespladib after antivenom administration for snakebite: a phase II randomized clinical trial (BRAVIO). Gerardo CJ, Bhalla A, Mohanty CR, et al. *PLoS Negl Trop Dis 2026;20(9):e0014736*.

Message: IV-then-oral varespladib given late (mean 7 h after the bite, after ASV) in 140 patients in India and the USA did not meet its primary end-points; signals in krait bites are only hypothesis-generating. ASV and ventilation remain the standard of care.

▶ Read article

Krait-specific measures (STG)

- AN is not effective in confirmed krait bites; do not persist beyond a trial.
- The STG suggests **calcium gluconate 10%, 10 mL IV slowly over 5–10 min, 6-hourly** (children 1–2 mL/kg of a 1:1 dilution) until neuroparalysis recovers — evidence is limited; monitor ECG.
- Check and correct **hyponatraemia** (natriuresis); look for rhabdomyolysis and AKI with greater black krait/banded krait.
- Expect **prolonged ventilation** — often 3–10 days; recovery of ptosis lags behind respiration.

Differential diagnosis of acute descending paralysis

Condition	Distinguishing features
Snakebite (elapid)	Bite/floor-sleeping history, abdominal pain (krait), local swelling (cobra), rapid onset, clear sensorium
Organophosphate poisoning	Miosis, secretions, fasciculations, bradycardia, smell; low cholinesterase
Botulism	Food history, dilated pupils, dry mouth, constipation, slower evolution, several family members
Guillain–Barré / Miller Fisher variant	Ascending (GBS) or ophthalmoplegia–ataxia–areflexia (Miller Fisher), preceding infection, CSF protein
Myasthenic crisis	Known myasthenia, fluctuating fatigable weakness
Tick paralysis	Ascending, child, attached tick — removal cures
Diphtheria	Pharyngeal membrane, palatal palsy weeks after sore throat
Brainstem stroke	Focal signs, altered sensorium, asymmetry; CT/MRI
Hypokalaemic paralysis	Flaccid limb weakness without ptosis; low K^+

Pitfalls in neurotoxic envenoming

Sending the patient home with "gastritis" · waiting for ptosis to "become severe" before ASV · sedating an anxious patient with early bulbar palsy · inserting an NG tube in a patient with poor gag and no airway protection · transferring without bag-mask ventilation · stopping ventilation too early when the neck is still weak.

CHAPTER 11 · RESPIRATORY SUPPORT

Airway, Ventilation and Recovery in Neuroparalysis

An Ambu bag in the hands of a trained person is life-saving at every level of care

Indications for assisted ventilation

- Inability to lift the neck against gravity (**broken-neck sign**)
- Pooling of saliva, poor gag, aspiration risk
- Paradoxical breathing, use of accessory muscles
- SBC falling or < 20; breath-holding time < 45 s; cannot finish a sentence
- SpO_2 < 90% on oxygen; rising $PaCO_2$ (> 45–50 mmHg) or respiratory acidosis
- Exhaustion, cyanosis, respiratory arrest

Pearl

Because the patient is conscious but paralysed, **explain everything**, sedate adequately once intubated and never discuss prognosis at the bedside as if the patient cannot hear. Avoid sedatives, opioids and neuromuscular blockers **before** the airway is secured.

Airway and ventilation — practical points

Step	Details
Positioning	Head-up or recovery position; suction of secretions; nil by mouth; NG tube only if gag is poor or mentation is depressed
Bag-valve-mask	Oxygen 10–15 L/min with reservoir; may be needed for hours during transfer — rotate operators; airway adjunct
Intubation	Cuffed endotracheal tube (or supraglottic airway as a bridge). Avoid suxamethonium where there is rhabdomyolysis/hyperkalaemia; with complete paralysis little or no relaxant is needed; give sedation (midazolam/fentanyl or ketamine)
Ventilator settings	Lung-protective volume control: tidal volume 6–8 mL/kg predicted body weight, PEEP 5 cmH_2O, FiO_2 to SpO_2 94–98%, rate 12–16/min; lungs are usually normal
No ventilator?	Manual bag ventilation by relatives/staff in shifts has saved many lives in rural India while awaiting transfer
ICU care	Eye care (lagophthalmos, corneal exposure), pressure-area care, DVT prophylaxis once coagulation normal, chest physiotherapy, nutrition, VAP prevention
Weaning	Begin when the patient can **lift the head** and has good cough and SBC; pressure-support trial; ptosis may persist after successful extubation

Do not declare brain death

Fixed dilated pupils, absent doll's-eye and corneal reflexes and no motor response can all occur with complete neuromuscular paralysis. **Never** declare brain death in a snakebite victim without confirmatory tests (EEG, nerve conduction studies, cerebral blood flow) and a long enough wait.

Expected recovery

Snake	Recovery
Cobra (post-synaptic)	Begins within hours of ASV ± neostigmine; full in 24–48 h; ventilation usually 1–3 days
Common krait (pre-synaptic)	Slow; ventilation often 3–10 days, occasionally weeks; recovery as nerve terminals regenerate
Sea snake	Slow; depends on rhabdomyolysis and renal recovery

CHAPTER 12 · PROTOCOL

Treatment Protocol — Haemotoxic (Vasculotoxic) Envenomation

ASV by the 6-hour rule, blood products, bleeding and shock

Bleeding and/or unclotted 20WBCT ± local swelling (viper)

Strict bed rest; avoid IM injections and arterial/central punctures (use compressible sites)

Group & cross-match on first sample; CBC, PT/INR, fibrinogen, creatinine, urine

ASV loading dose: 10 vials (Russell's viper / unknown viper) or 6 vials (confirmed saw-scaled viper) over 30–60 min

Repeat **20WBCT 6 hours** after the start of the loading dose

Clotted → no more ASV; 20WBCT 6-hourly for 24 h to detect recurrence

Not clotted → repeat ASV (2 vials low-dose or 6 vials high-dose regimen; WHO 5–10 vials) and recheck 6 h later

Brisk bleeding at any time → repeat ASV within 1–2 h; after 30 vials with persisting coagulopathy → FFP/ cryoprecipitate

Blood products — when and how

Product	Indication and dose
Fresh frozen plasma (FFP)	After adequate ASV, for persisting coagulopathy with bleeding, or before essential surgery: **10–15 mL/kg** over 30–60 min (ideally within 4 h of ASV); aim INR < 2 at 6 h
Cryoprecipitate	Low fibrinogen (< 1 g/L) with bleeding: 1 unit/10 kg (fibrinogen, factor VIII)
Fresh whole blood	When FFP and cryoprecipitate are not available; also replaces red cells
Packed red cells	Significant bleeding or Hb falling; keep **PCV ≥ 30%** in active bleeding (measure 4–6-hourly)
Platelets	Platelets < 20,000/mm³ with bleeding, or < 50,000 before surgery; avoid in TMA unless life-threatening bleeding

Never

Heparin (worsens bleeding, does not stop VICC), **antifibrinolytics** (tranexamic acid, EACA), aspirin/ NSAIDs, IM injections, routine NG tubes in coagulopathy, central lines in non-compressible sites, or corticosteroids for coagulopathy.

Intracranial haemorrhage

Headache, vomiting, seizures, focal signs, falling GCS: correct coagulation urgently (ASV up to 30 vials, then FFP/cryo), CT brain, neurosurgical opinion; control BP; seizures — levetiracetam/ phenytoin. Poor prognosis; most deaths in viper bite come from ICH, shock or AKI.

Shock and capillary leak

- **Fluid challenge** (STG): 200 mL normal saline over 5 min (children 20 mL/kg crystalloid over 5–10 min, or over 30 min in small children); repeat while BP improves, aiming JVP 8–10 cm above the sternal angle with the patient at 45°; **stop** if crepitations, hepatomegaly or pulmonary oedema develop.

- Persistent hypotension: **noradrenaline** 0.05–0.5 µg/kg/min (first choice, especially if sepsis), **dopamine/dobutamine** if myocardial dysfunction (hepatomegaly, rales); **atropine** 0.6 mg IV for hypotension with bradycardia.
- Consider **acute adrenal/pituitary insufficiency** in refractory shock with hypoglycaemia and hyponatraemia after Russell's viper bite — **hydrocortisone 100 mg IV 6–8-hourly**.
- Capillary leak with hypoalbuminaemia: albumin may be needed; watch for pulmonary oedema once leak reverses.

Other haemotoxic problems

Problem	Management
Thrombotic microangiopathy (TMA)	Schistocytes, thrombocytopenia, high LDH, AKI despite corrected clotting (Russell's viper, HNPV): ASV does not reverse TMA; supportive care and **dialysis**; plasma exchange has been used but evidence is weak
GI bleeding	ASV first; PPI (pantoprazole 40 mg IV 12-hourly); transfusion; endoscopy after coagulation corrected
Haematuria	Red urine may persist for days after clotting is corrected — not an indication for more ASV
Bleeding in pregnancy	Antepartum haemorrhage/abruption, fetal loss: ASV, obstetric team, fetal monitoring, blood products before delivery
Recurrent coagulopathy	Re-treat with ASV if 20WBCT becomes unclotted again

CHAPTER 13 · RENAL TOXICITY

The Kidney in Snakebite — AKI, FAD and Dialysis

The commonest cause of death after the first 24 hours in Russell's viper bites

Causes and mechanisms

- Hypotension/hypovolaemia (bleeding, capillary leak, vomiting) → ischaemic **acute tubular necrosis (ATN)**
- Direct venom nephrotoxicity (tubular, glomerular, mesangial)
- Pigment nephropathy — haemoglobin (haemolysis), myoglobin (rhabdomyolysis)
- Microvascular thrombosis/**TMA**; **acute cortical necrosis** (poor prognosis)
- Immune-complex glomerulonephritis (serum sickness), interstitial nephritis
- Drugs (NSAIDs, aminoglycosides) and sepsis

Risk factors for AKI

Russell's viper or HNPV bite, delay to ASV > 6 h, hypotension, haemoglobinuria/myoglobinuria, prolonged coagulopathy, TMA, older age.

Monitoring for AKI

Urine output	Hourly; catheterise if shock or oliguria; target **≥ 1 mL/kg/h** (children 1–2 mL/kg/h)
Urine	Colour, dipstick for blood, microscopy for RBCs and casts; centrifuge to separate haemoglobinuria from haematuria
Blood	Creatinine, urea, K^+, bicarbonate daily (12-hourly if rising)
Fluid balance	Weight, input–output chart, JVP, chest

Prevent AKI

Early full-dose ASV · correct hypovolaemia promptly · avoid nephrotoxins (NSAIDs, aminoglycosides, contrast) · treat pigmenturia with adequate fluids · watch urine output hourly.

Forced alkaline diuresis (FAD) — as described in the national STG

If urine output does not improve after rehydration, or the urine dipstick is positive for blood, the STG advises a trial of FAD **within the first 24 hours** of the bite to prevent pigment nephropathy; delayed FAD has no role.

Step	Adult regimen (one cycle ≈ 2 h 30 min)
1	Inj. **furosemide 40 mg IV** stat
2	Normal saline 500 mL + **20 mL $NaHCO_3$** (7.5–8.4%) over 20 min
3	Ringer's lactate 500 mL + 20 mL $NaHCO_3$ over 20 min
4	5% dextrose 500 mL + **10 mL KCl** over 90 min
5	**Mannitol** (20%) 150 mL over 20 min

Expected urine output about **3 mL/min**. If the patient responds to the first cycle, continue for up to 3 cycles. If there is **no response to furosemide**, stop FAD and refer for dialysis.

Caution — read before using FAD

FAD is **not part of the WHO SEARO recommendations**, and diuretics and mannitol have no proven benefit in established AKI. It must **never** be given to a patient who is fluid overloaded, anuric, hyperkalaemic or with pulmonary oedema; potassium must not be added when K^+ is high or urine is not flowing; bicarbonate can precipitate hypocalcaemia and seizures, especially with rhabdomyolysis. Many units today use **isotonic fluid resuscitation to a urine output of 1–2 mL/kg/h** with bicarbonate only for acidosis, and proceed early to dialysis.

Metabolic emergencies

Emergency	Treatment (STG)
Hyperkalaemia (K^+ > 6.5–7 mmol/L or ECG changes)	**Calcium gluconate 10%, 10 mL IV over 2–5 min** (repeat up to 3 times, ECG monitoring) · **50 mL of 50% dextrose + 10 units soluble insulin** IV · **sodium bicarbonate 8.4% 40 mL** slowly if acidotic · **salbutamol** nebulisation 5–10 mg · arrange dialysis
Severe metabolic acidosis (pH < 7.1 with hypotension)	Sodium bicarbonate 8.4% 40 mL (2–3 ampoules) in 5% dextrose, or half the calculated deficit over 3–4 h; dialysis if no improvement

Fluid overload / pulmonary oedema	Sit up, oxygen, stop fluids, furosemide trial if urine flowing; dialysis/ultrafiltration

Drug dosing in snakebite AKI (examples)

Drug	Adjustment
Amoxicillin–clavulanate	eGFR 10–30: 625 mg 12-hourly; < 10: 625 mg daily (supplement after HD)
Ceftriaxone	No adjustment usually (max 2 g/day)
Metronidazole	No adjustment; caution in severe hepatic failure
Tramadol	eGFR < 30: 12-hourly, max 200 mg/day
Ranitidine/famotidine	Halve dose; prefer pantoprazole (no adjustment)
Aminoglycosides, NSAIDs, contrast	Avoid

CHAPTER 13 · RENAL REPLACEMENT

Dialysis and Renal Follow-up

Indications, modality and long-term outcome

Indications for dialysis (STG)

- Clinical uraemia — encephalopathy, pericarditis, uraemic bleeding
- Blood urea > 130 mg/dL (≈ 27 mmol/L) or creatinine > 4 mg/dL (≈ 500 µmol/L)
- **Hypercatabolic AKI**: daily rise of urea > 30 mg/dL, creatinine > 1 mg/dL, potassium > 1 mEq/L, or fall of bicarbonate > 2 mmol/L
- Fluid overload with pulmonary oedema
- **Hyperkalaemia** > 7 mmol/L or ECG changes not controlled medically
- Severe metabolic acidosis
- Oliguria/anuria not responding to conservative management

Modality	When
Intermittent haemodialysis	Preferred for hyperkalaemia, hypercatabolic AKI; minimise heparin (coagulopathy) — saline flushes or regional anticoagulation
Peritoneal dialysis	Useful at secondary hospitals and in children; avoid with abdominal bleeding/surgery
Continuous renal replacement therapy / SLED	Haemodynamically unstable ICU patients; effluent 20–25 mL/kg/h

Outcome and follow-up

- Most survivors with ATN recover renal function in 2–4 weeks (diuretic phase — watch for polyuria, hypokalaemia, hyponatraemia).
- **Acute cortical necrosis** (no recovery after 4 weeks; small echogenic kidneys, cortical calcification later) → dialysis dependence; renal biopsy if no recovery by 3–4 weeks.
- A significant minority develop **chronic kidney disease** — check creatinine, eGFR, urine protein and BP at 3 months and annually.
- Avoid NSAIDs in survivors; adjust doses of renally excreted drugs.

Remember

A patient arriving days later with AKI and a **clotted** 20WBCT needs fluids, electrolyte correction and dialysis — not ASV. ASV is given only if coagulopathy or other evidence of active venom persists.

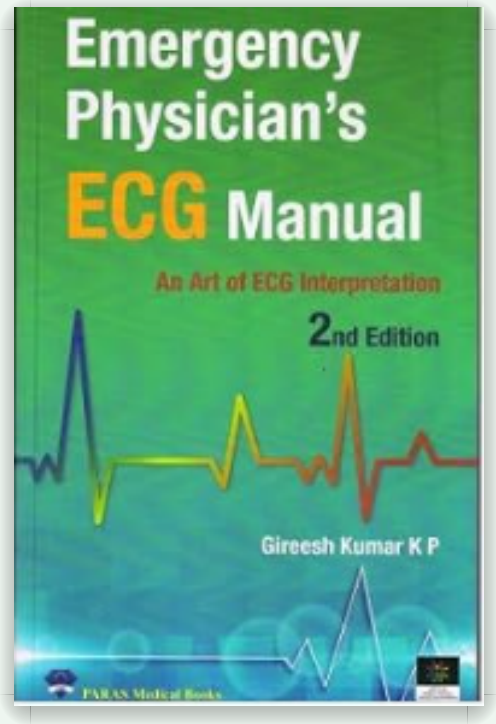
Guideline

▶ Guideline STG 2016 — volume replacement, FAD, dialysis ▶ Guideline WHO SEARO 2016

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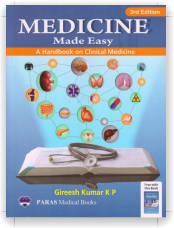
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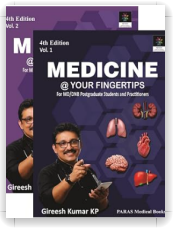
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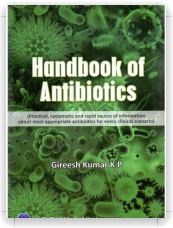
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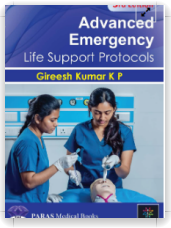
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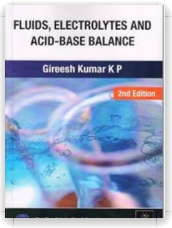
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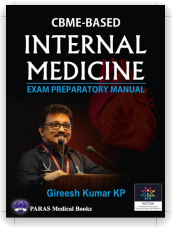
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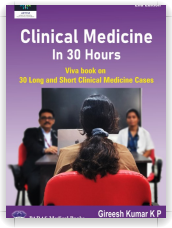
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CHAPTER 14 · COMPLICATIONS

Complications of Snakebite

Local tissue damage, compartment syndrome, infection, endocrine, cardiac and long-term sequelae

Local necrosis and wound care

- Clean the wound with povidone-iodine or chlorhexidine; **no tight dressings**; leave blisters intact (aspirate large tense bullae with a fine sterile needle only if necessary).
- Elevate the limb (on a pillow/sandbag) once ASV has been given; splint in a functional position.
- Necrosis: wait for a clear **line of demarcation (5–7 days)**, then surgical debridement, saline dressings, split-skin grafting; early physiotherapy.
- Cobra necrosis may be "wet" and spread under intact skin — regular inspection.
- Amputation is a last resort for gangrene (often tourniquet-induced).

Antibiotics (STG) — for local necrosis/infection	
Amoxicillin–clavulanate	1.2 g IV 8-hourly × 7 days, then 625 mg orally 8-hourly × 3–7 days (children 100 mg/kg/day IV in 3 doses, then 50 mg/kg/day)
or Ceftriaxone	1 g IV 12-hourly × 7 days (children 100 mg/kg/day in 2 doses)
+ Metronidazole	400–500 mg IV 8-hourly × 7 days (children 30 mg/kg/day in 3–4 doses) for anaerobes

The STG also states that routine prophylactic antibiotics are not needed for a clean bite. Give them when there is necrosis, interference with the wound (incisions, herbs), abscess or cellulitis; reduce doses in AKI. Snake mouth flora includes Gram-negatives (Morganella, Proteus, Aeromonas) and anaerobes.

Compartment syndrome and fasciotomy

True compartment syndrome is **uncommon** after snakebite: most swelling is subcutaneous. Unnecessary fasciotomy in a coagulopathic patient causes massive bleeding and infection.

Clinical features (5 Ps): **Pain** out of proportion and on **passive stretch**, Pallor, Paraesthesia/hypoaesthesia, Paralysis/weakness of the muscles of the compartment, Pulselessness (very late). Tense, woody compartment.

Measure: needle manometry (16G needle connected through a three-way tap and saline column to a manometer/transducer), Doppler for arterial flow, rising CK.

Fasciotomy only when ALL three are present (STG)

1. Clinical evidence of compartment syndrome, **and**
2. Intracompartmental pressure **> 40 mmHg** (adults), **and**
3. **Haemostatic abnormalities corrected** (adequate ASV ± clotting factors).

Before cutting

Give adequate ASV first (it lowers compartment pressure), elevate the limb, consider mannitol; repeat measurement. Corticosteroids are not recommended for local effects. ▶ Article

Other complications

Complication	Features and management
Tetanus	Give tetanus toxoid (Td 0.5 mL IM) if not immunised in the last 5 years or unknown status — **after coagulopathy is corrected** to avoid haematoma
Pain	**Paracetamol** 500–1000 mg 4–6-hourly (max 4 g/day; children 10–15 mg/kg/dose); **tramadol** 50 mg orally/IV for severe pain; **avoid NSAIDs and aspirin**; avoid opioids/sedatives in neurotoxic envenoming unless ventilated

Acute pituitary/adrenal insufficiency	Russell's viper — haemorrhagic infarction of the anterior pituitary: refractory hypotension, hypoglycaemia, hyponatraemia → hydrocortisone 100 mg IV 6–8-hourly; check cortisol
Chronic hypopituitarism (Sheehan-like)	Months–years later: fatigue, hypotension, loss of axillary/pubic hair, amenorrhoea, infertility, impotence, hypothyroidism → hormone replacement (steroid first, then thyroxine); ask at follow-up
Cardiac	Myocarditis, arrhythmias, ischaemia (coronary thrombosis/vasospasm), hyperkalaemic arrest — ECG, troponin, echocardiography; supportive
Cerebral	Intracranial haemorrhage; ischaemic stroke (thrombosis, hypotension, vasculitis), sometimes days after bite; hypoxic brain injury after arrest
Pulmonary	Aspiration pneumonia (bulbar palsy), pulmonary oedema (capillary leak, fluids), alveolar haemorrhage, ARDS, pleural effusion
Ocular	Cobra-spit ophthalmia; exposure keratitis in prolonged ptosis/paralysis; cortical blindness (rare)
Multi-organ dysfunction	Shock + AKI + DIC + ARDS + liver injury — ICU care, early dialysis; high mortality
Sepsis	Wound, line, ventilator and catheter infections in prolonged stays
Long-term disability	Contractures, chronic ulcers, amputation, CKD, hypopituitarism, chronic pain, post-traumatic stress, loss of livelihood — physiotherapy, rehabilitation, counselling and social support (State compensation schemes exist in several States)

CHAPTER 15 · SPECIAL SITUATIONS

Special Situations, Referral, Discharge and Records

Children, pregnancy, the elderly, levels of care and medico-legal aspects

Children

- Same ASV dose as adults; dilute in 5–10 mL/kg
- Envenoming more severe and faster (higher venom/kg)
- Weight-based doses for adrenaline (0.01 mg/kg), atropine, neostigmine, fluids
- Watch fluid volume; hypoglycaemia; febrile convulsions with pyrogenic reactions

Pregnancy

- Treat the mother — same ASV dose; benefits outweigh risks
- Risks: APH, abruption, preterm labour, fetal death, PPH
- Left lateral position, fetal monitoring, obstetric team
- Correct coagulopathy before delivery/LSCS; postpartum: watch for Sheehan-like pituitary failure

Elderly & comorbid

- Prone to fluid overload, AKI, ICH
- Adrenaline caution in IHD/ hypertension
- Patients on anticoagulants/ antiplatelets — bleeding harder to interpret; INR baseline
- β-blockers blunt anaphylaxis response (glucagon)

Levels of care — who does what

Level	Minimum capability
Community / ASHA / first responder	RIGHT first aid, no harmful practices, call 108, transport lying down, photo of snake
PHC / sub-district hospital	Triage, 20WBCT, **ASV loading dose**, adrenaline for reactions, AN trial, bag-mask ventilation, oxygen, IV fluids, referral with escort
District hospital / CHC with ICU	Repeat ASV, blood products, ventilator, peritoneal dialysis, basic surgery (debridement)
Tertiary centre / medical college	Prolonged ventilation, haemodialysis/CRRT, neurosurgery, plastic surgery, endocrine evaluation, rehabilitation

Referral criteria (STG)

Vasculotoxic

No ASV available (transfer to a centre confirmed by phone) · still unclotted after the loading dose · continued bleeding after full ASV · rising creatinine/ oliguria (dialysis) · progressive sepsis or need for surgery.

Neurotoxic

Progressive paralysis · cannot lift the neck · SpO_2 < 90% · falling SBC or pooling of saliva · need for ventilator. Transfer with a trained person and Ambu bag, after giving ASV and one AN dose.

Discharge criteria and advice

- No envenoming after 24 h of observation with normal serial 20WBCT and neurology → discharge.
- After ASV: clinically stable, clotting normal for > 24 h, creatinine stable/falling, no ptosis/bulbar weakness, wound clean (at least 48 h observation).
- Tetanus status updated; limb physiotherapy plan.

Tell the patient to return if

Bleeding from gums/nose or in urine · increasing pain, swelling or discharge at the bite · fever, rash, joint pains (serum sickness, 5–12 days) · reduced urine · drowsiness or weakness · later — tiredness, dizziness, loss of periods or libido (pituitary).

Documentation and medico-legal aspects

- Snakebite deaths are **unnatural deaths** — inform police; post-mortem as per local rules (needed for State compensation in many States).
- Record: time and place of bite, description/photo of snake, first aid, time of arrival, 20WBCT times and results, ASV batch, number of vials and times, reactions and treatment, referral details. Use the STG snakebite proforma.
- Report cases and deaths through the IDSP/IHIP system as directed under NAPSE; snakebite is notifiable in some States (e.g. Karnataka).
- Consent: explain risk of reactions, but do not delay life-saving ASV for written consent in an emergency.

Sample referral note

```
To: Medical Officer, Emergency Medicine Department, _____________ (confirmed by phone with Dr ______ at
__:__)
Patient: _________, __ y, M/F, weight __ kg. Bitten on (site) ______ at __:__ on __/__/____ in field/house/
bed.
Snake: seen / not seen / photo attached — likely ______. First aid: splint / tourniquet removed at __:__.
Findings on arrival (__:__): BP ___/___, HR ___, SpO2 __%, ptosis Y/N, neck lift Y/N, SBC __, bleeding
______, urine ______.
20WBCT: __:__ clotted/not clotted; __:__ clotted/not clotted.
ASV: __ vials (batch ______) from __:__ to __:__. Premedication: ______. Reaction: none / ______ treated
with ______.
Other: AN trial (time/response) ______; fluids ______; tetanus ______.
Reason for referral: ventilator / dialysis / persistent bleeding / no ASV stock / surgery.
Escort: Dr/Nurse ______ with Ambu bag, oxygen, adrenaline. Ambulance 108 no. ______.
Signature, name, registration no., phone: _____________________
```

CHAPTER 15 · SYSTEMS OF CARE

The Snakebite-ready Facility

ASV stock, cold chain, emergency kit, staff training and audit

ASV stock and storage

- Keep a minimum buffer stock — a PHC should hold enough for at least **two full treatments (20–30 vials)**; district hospitals more during the monsoon (estimate from last year's case load).
- **Lyophilised ASV**: store in a cool, dark, dry place (below 25 °C) — preferred where power supply is unreliable.
- **Liquid ASV**: 2–8 °C in the refrigerator; **never freeze**; record temperatures twice daily.
- First-expiry, first-out; check stock weekly; indent early through the State drug portal (e-Aushadhi/State systems) before the monsoon.
- Keep sterile water/saline and IV sets with the ASV in a single labelled "Snakebite box".

Snakebite emergency kit

Item	Purpose
New, dry glass test tubes, timer	20WBCT
Adrenaline 1 mg/mL ampoules, 1 mL syringes	Premedication and anaphylaxis
Chlorpheniramine, hydrocortisone injections	Reactions
Atropine, neostigmine (or neostigmine–glycopyrrolate)	AN trial
Ambu bag (adult and paediatric), masks, oropharyngeal airways, suction, oxygen	Respiratory support
Laryngoscope, ET tubes, supraglottic airway	Intubation
IV cannulae, NS, RL, 5% dextrose, infusion sets	ASV and fluids
Calcium gluconate, insulin, 50% dextrose, bicarbonate	Hyperkalaemia
Splints, crepe bandages, marking pen, measuring tape	Limb care and monitoring
Tetanus toxoid, paracetamol, tramadol	Supportive
Case proforma, observation chart, referral forms, phone list	Records and referral (Appendix A and B)

People and processes

- Every medical officer and staff nurse trained in the national protocol, 20WBCT, ASV preparation and anaphylaxis management — refresher before every monsoon (NAPSE capacity building).
- Bag-mask ventilation skills for all staff including drivers and attenders.
- Written referral pathway with phone numbers of the nearest ventilator, dialysis and blood bank facilities; 108 ambulance coordination.
- Display posters: first aid do's and don'ts, local venomous snakes, the master algorithm (Chapter 16).
- Community link: ASHAs and panchayats informed of the facility's ASV availability and the helpline.

Audit indicators

Indicator	Target
Door-to-ASV time (when indicated)	< 30 min
20WBCT done before ASV	100%
Test doses given	0%
Patients receiving a full loading dose	100%
Reactions treated with IM adrenaline	100%
Days with ASV stock-out	0
Cases reported on IHIP/IDSP	100%
Deaths reviewed (death audit)	100%

Programme resources

▶ Guideline NAPSE — capacity building and ASV supply ▶ Guideline NHSRC module for clinicians

CHAPTER 15 · CRITICAL CARE

Snakebite in the ICU — The Daily Care Bundle

A checklist for ward rounds in envenomed patients

Domain	Check every day (or every shift)
Venom activity	20WBCT/PT-INR 6-hourly until stable for 24 h; bleeding sites; swelling progression; ptosis and neck lift; recurrence of coagulopathy (24–48 h)
ASV record	Total vials, times, reactions; whether a further dose is truly indicated (6-hour and 1-hour rules); total ≥ 20 (neuro) or ≥ 30 (haemo) → stop and rethink
Airway & ventilation	Sedation score, cuff pressure, head-up 30°, VAP bundle, daily neck-lift/cough assessment for weaning readiness; eye care (taping/lubricant) for lagophthalmos
Circulation	MAP ≥ 65 mmHg, lactate, fluid balance, capillary leak (effusions), vasopressor needs; consider adrenal crisis
Kidney	Urine output hourly, creatinine and K^+ 12–24-hourly, bicarbonate; dialysis needs; drug dose adjustment
Blood	Hb/PCV, platelets, smear for schistocytes (TMA), LDH; transfusion thresholds; no heparin or antifibrinolytics
Limb	Swelling grade, pulses, compartment pressure if tense, blisters, necrosis demarcation, wound swab, splint in functional position, physiotherapy
Infection	Wound, lines, catheter, chest; antibiotics only when indicated; remove unnecessary lines
Metabolic	Glucose, sodium (krait natriuresis), potassium, calcium (bicarbonate/rhabdomyolysis), magnesium
Nutrition & GI	Early enteral feeding once airway protected and no GI bleed; PPI for stress-ulcer prophylaxis in coagulopathy
Prophylaxis	DVT prophylaxis (mechanical first; pharmacological only once coagulation normal); pressure-area care; tetanus toxoid after coagulation corrected
Communication	Explain to the conscious paralysed patient; family updates; avoid prognostic statements of "brain death"; document daily
Before discharge	Serum sickness warning, renal follow-up, pituitary symptoms advice, rehabilitation plan, death/ medico-legal documentation where applicable

Pearl — late deterioration

A patient who was improving and then deteriorates on day 2–4 may have: recurrent coagulopathy, intracranial bleed, sepsis, aspiration pneumonia, pulmonary oedema from leak reversal, hyperkalaemia, adrenal crisis or a late ASV reaction. Work through this list before giving more ASV.

Remember

Most snakebite deaths after admission are due to **respiratory failure not ventilated in time**, **AKI not dialysed in time**, or **intracranial haemorrhage**. The ICU bundle aims at all three.

CHAPTER 15 · PAEDIATRICS

Snakebite in Children — Weight-based Quick Chart

Same ASV, different everything else

Drug / fluid	Dose per kg	10 kg	20 kg	30 kg
ASV loading dose	Same as adult	10 vials	10 vials	10 vials
Diluent for ASV	5–10 mL/kg (max 200 mL running)	50–100 mL	100–200 mL	150–200 mL
Adrenaline SC premedication	0.005 mg/kg (1:1000)	0.05 mg (0.05 mL)	0.1 mg	0.15 mg
Adrenaline IM anaphylaxis	0.01 mg/kg (max 0.5 mg)	0.1 mg (0.1 mL)	0.2 mg	0.3 mg
Chlorpheniramine IV	0.2 mg/kg	2 mg	4 mg	6 mg
Hydrocortisone IV	2–4 mg/kg	20–40 mg	40–80 mg	60–120 mg
Atropine IV (WHO)	0.02 mg/kg (min 0.1 mg)	0.2 mg	0.4 mg	0.6 mg
Neostigmine IV/IM	0.04 mg/kg (range 0.025–0.08)	0.4 mg	0.8 mg	1.2 mg
Neostigmine maintenance	0.01 mg/kg every 30 min	0.1 mg	0.2 mg	0.3 mg
Fluid bolus (shock)	10–20 mL/kg over 10–30 min	100–200 mL	200–400 mL	300–600 mL
FFP	10–15 mL/kg	100–150 mL	200–300 mL	300–450 mL
Paracetamol	15 mg/kg/dose	150 mg	300 mg	450 mg
Prazosin (scorpion)	30 µg/kg/dose	0.3 mg	0.6 mg	0.9 mg
Calcium gluconate 10% (hyperkalaemia)	0.5 mL/kg (max 10 mL) slow IV	5 mL	10 mL	10 mL
Urine output target	1–2 mL/kg/h	10–20 mL/h	20–40 mL/h	30–60 mL/h

Paediatric pearls

Children deteriorate faster; keep them in a monitored bed with a parent. Count the SBC only in children old enough to count (≈ > 6 years); otherwise watch for drooling, nasal voice, inability to sit or hold the head up. Use intra-osseous access if IV fails — ASV can be given IO.

Watch the volume

Ten vials of ASV are 100 mL — in a 10 kg child that is already 10 mL/kg. Add the diluent carefully and use a burette or infusion pump; review for fluid overload before each repeat dose.

CHAPTER 15 · FORENSIC TOXINOLOGY

Forensic and Medico-legal Aspects of Bites and Stings

Post-mortem findings, homicidal snakebite and compensation

Post-mortem findings in snakebite

- **Bite site**: fang punctures (may be invisible in krait bites — search fingers, toes, face and scalp under a lens), local haemorrhage, swelling, blistering, necrosis.
- **Viper bites**: extensive subcutaneous and intermuscular haemorrhage, petechiae and haemorrhages on serous surfaces, haemorrhage into the brain, GI tract, adrenals and pituitary; swollen pale kidneys with tubular necrosis or cortical necrosis.
- **Elapid bites**: few local signs (krait), signs of asphyxia, pulmonary oedema; cobra — local necrosis.
- **Samples**: skin and tissue around the bite, blood and urine, preserved for venom detection by ELISA in forensic laboratories; fang-mark spacing measured in mm (gives an idea of snake size).
- Look for treatment marks — tourniquet grooves, incisions, herbal applications.

Snakebite as homicide or suicide

Rarely, venomous snakes have been used for homicide in India; a widely reported 2020 case from Kerala, in which a woman was killed with a cobra bite while asleep, ended in conviction on the basis of **forensic herpetology** — the improbability of the snake's behaviour, the bite marks' location and spacing, venom detection, and the procurement of the snake. Clues: bites at unusual sites in a sleeping or sedated person, repeated envenoming events, no snake habitat, a snake found in a locked room, prior sedation in toxicology.

Medico-legal duties of the doctor

Situation	Action
Death due to snakebite or any bite/sting	Unnatural death — intimate police; post-mortem as per law; issue cause-of-death certificate after PM
Brought dead	Do not certify; inform police
Suspected foul play	Preserve bite-site tissue, blood and urine; document photographs with scale
Snakebite as notifiable condition	Report through IDSP/IHIP where notified (e.g. Karnataka)
Compensation	Several States treat snakebite death as a natural calamity or under wildlife/agricultural schemes — post-mortem report and FIR are usually required; inform families
Occupational bite (farm workers, snake rescuers, forest staff)	Document as occupational injury for insurance/compensation
Wildlife law	Most Indian snakes are protected under the Wildlife (Protection) Act, 1972 — killing or keeping them is an offence; involve forest department for rescue
Dog bites	Municipal reporting of stray dog bites as locally required; rabies is notifiable in many States

Good documentation protects everyone

Time of bite, time of arrival, clinical findings, 20WBCT times and results, ASV batch and vials, reactions and their treatment, referral time and reason — written contemporaneously and signed.

CHAPTER 16 · MASTER ALGORITHM

Snakebite — One-page Management Algorithm

Print and pin in every Emergency Medicine Department

SUSPECTED SNAKEBITE — reassure · remove tourniquet only after preparations · ABC · IV line · admit for 24 h

Baseline: **20WBCT**, CBC, PT/INR, creatinine, K^+, urine, ECG, group & cross-match · Examine: bite, ptosis, neck lift, SBC, bleeding, BP, urine

Is there systemic or severe local envenoming? (bleeding / unclotted 20WBCT / neurotoxic signs / shock / AKI / swelling > ½ limb or rapidly spreading / tender nodes)

NO
Observe 24 h: 20WBCT hourly × 3 then 6-hourly; neuro checks hourly; tetanus; analgesia (no NSAIDs)

No signs at 24 h → dry/non-venomous bite → discharge with advice

YES → ASV (no test dose; adrenaline 0.25 mg SC premedication per STG unless contraindicated; adrenaline 0.5 mg IM drawn up)

Neurotoxic
10 vials over 30–60 min + AN trial
Reassess at 1 h → repeat 10 vials if not better
Max 20 · ventilate early

Haemotoxic
10 vials (RV) / 6 vials (SSV) over 30–60 min
20WBCT at 6 h → repeat 2–6 (or 5–10) vials 6-hourly till clotted
Reassess at **30**

Reaction? Stop ASV → adrenaline 0.5 mg IM (0.01 mg/kg) → fluids, chlorpheniramine 10 mg IV, hydrocortisone 200 mg IV → restart slowly

Respiratory failure? Neck lift lost / SBC < 20 / SpO_2 < 90% → bag-mask → intubate → ventilate; do not declare brain death

Kidney? Urine < 1 mL/kg/h, rising creatinine, K^+ > 7 → fluids, treat K^+, early dialysis

Bleeding persists after adequate ASV → FFP 10–15 mL/kg, cryoprecipitate, whole blood; **no heparin, no tranexamic acid**

Local necrosis → wound care, antibiotics if infected, debride after demarcation; fasciotomy only if pressure > 40 mmHg + coagulation corrected

Refer: no ASV, ventilator or dialysis needed, ongoing bleeding — with escort, ASV given, AN dose given

Discharge after ≥ 24 h (≥ 48 h if ASV): warn about serum sickness (5–12 days), wound infection, urine output; follow up creatinine and pituitary function (Russell's viper)

CHAPTER 16 · QUICK REFERENCE

Snakebite Drug and Dose Card

Adult doses with paediatric equivalents (verify against current guideline and product insert)

Drug	Adult	Child	Use / comment
Polyvalent ASV	Neuro: 10 vials, repeat 10 at 1 h (max 20). Haemo: 10 (RV) / 6 (SSV), repeat 2–6 vials 6-hourly (WHO 5–10)	Same as adult	Reconstitute each vial in 10 mL; dilute in 5–10 mL/kg NS/5% D; over 30–60 min; or ≤ 2 mL/min IV push
Adrenaline — premedication	0.25 mg (0.25 mL of 1:1000) SC	0.005 mg/kg SC	STG; omit in hypertension/IHD/ BP > 140/90
Adrenaline — anaphylaxis	0.5 mg (0.5 mL of 1:1000) IM, repeat 5–15 min	0.01 mg/kg IM (max 0.5 mg)	Infusion 0.05–0.5 µg/kg/min if refractory
Chlorpheniramine	10 mg IV (late reactions 2 mg 6-hourly × 5 days)	0.2 mg/kg IV (late 0.25 mg/kg/day)	Urticaria, itching, serum sickness
Hydrocortisone	100–200 mg IV	2–4 mg/kg IV	Anaphylaxis (slow); adrenal crisis 100 mg 6–8-hourly
Prednisolone	5 mg 6-hourly × 5 days	0.7 mg/kg/day divided	Serum sickness not settling with antihistamine
Atropine	0.6 mg IV before neostigmine; infusion 0.6 mg over 8 h	0.02 mg/kg (min 0.1 mg); STG 0.05 mg/kg	Also for hypotension with bradycardia
Neostigmine	1.5–2 mg IV/IM; then 0.5 mg every 30 min (× 5 doses, then taper)	0.025–0.08 mg/kg (STG 0.04 mg/kg); then 0.01 mg/kg every 30 min	Positive if ≥ 50% improvement within 1 h; stop after 3 doses if no response
Calcium gluconate 10%	10 mL IV over 5–10 min 6-hourly (krait, STG); 10 mL over 2–5 min for hyperkalaemia	0.5–1 mL/kg diluted (STG 1–2 mL/kg of 1:1)	ECG monitoring
Insulin–dextrose	10 units soluble insulin + 50 mL of 50% dextrose IV	0.1 unit/kg + 0.5 g/kg dextrose	Hyperkalaemia
Sodium bicarbonate 8.4%	40 mL slow IV (acidosis/ hyperkalaemia)	1–2 mL/kg diluted	Watch for hypocalcaemia
FFP / cryoprecipitate	FFP 10–15 mL/kg; cryo 1 unit/10 kg	Same per kg	Only after adequate ASV
Paracetamol	500–1000 mg 4–6-hourly (max 4 g/ day)	10–15 mg/kg/dose	Analgesia, pyrogenic reaction
Tramadol	50 mg orally/IV 6–8-hourly	1 mg/kg (> 12 years)	Severe pain; avoid NSAIDs
Tetanus toxoid (Td)	0.5 mL IM	Age-appropriate vaccine	After coagulopathy corrected
Amoxicillin–clavulanate	1.2 g IV 8-hourly	100 mg/kg/day IV ÷ 3	Infected/necrotic wounds
Ceftriaxone + metronidazole	1 g IV 12-hourly + 400–500 mg IV 8-hourly	100 mg/kg/day ÷ 2 + 30 mg/kg/day ÷ 3	Alternative; adjust in AKI

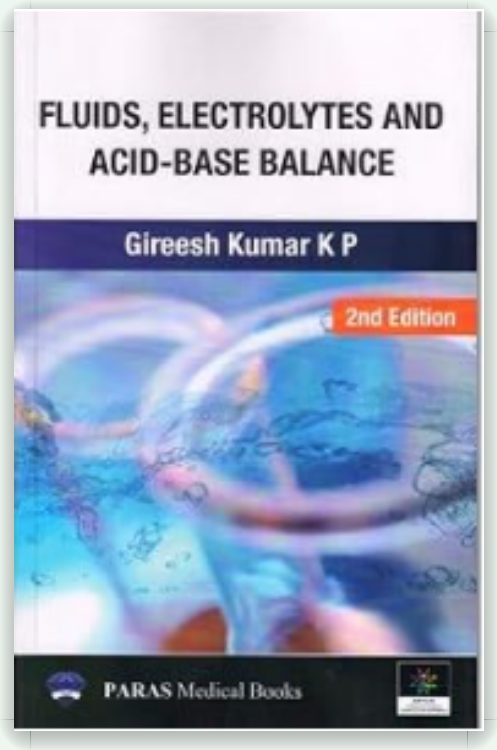
Ten things never to do in snakebite

Tourniquet · cut and suck · ASV test dose · "two vials to see" · IM or local ASV · heparin · tranexamic acid · NSAIDs/aspirin · early fasciotomy without measuring pressure or correcting clotting · declaring brain death in a paralysed patient.

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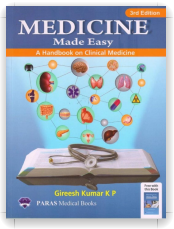
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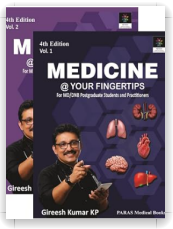
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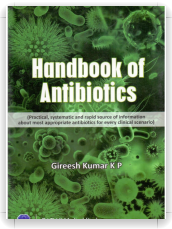
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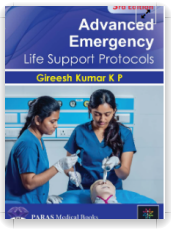
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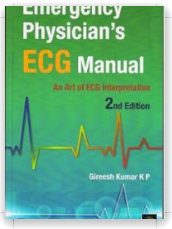
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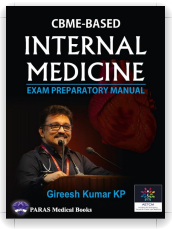
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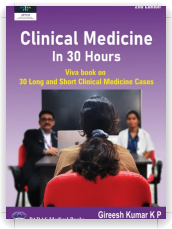
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PART B

Other Bites & Stings

Scorpions, bees, wasps and ants, spiders and centipedes, beetles and caterpillars, marine creatures, animal and human bites with rabies prophylaxis — and a few curiosities.

17. Scorpion sting
18. Bees, wasps, hornets and ants
19. Spiders, centipedes and millipedes
20. Beetles, caterpillars and blistering insects
21. Marine envenomations
22. Animal and human bites
23. Rabies post-exposure prophylaxis
24. Ticks, mites, leeches, toads and lizards
25. Anaphylaxis after bites and stings
26. Bites and stings at a glance

CHAPTER 17 · SCORPION

Scorpion Sting

An acute, time-limited, life-threatening autonomic storm — prazosin saves lives

Scorpions of medical importance in India

- Scorpions live in warm, dry regions throughout India; stings are commonest in rural homes, at night, in summer and the monsoon, while sleeping on the floor or putting on shoes and clothes.
- Of ~86–100 Indian species, the **Indian red scorpion** (*Hottentotta tamulus*, formerly *Mesobuthus/Buthus tamulus*) is the most dangerous — Maharashtra (Konkan), Gujarat, Karnataka, Andhra Pradesh, Telangana, Tamil Nadu, Rajasthan, Pondicherry.
- The **black scorpion** (*Heterometrus*, *Palamneus swammerdami*) is large and frightening but its sting causes mainly **severe local pain**, rarely systemic effects.
- Scorpions sting only when roughly handled or trodden on; children are the most severely affected (small body mass).

Indian red scorpion

▶ Images



Hottentotta (Mesobuthus) tamulus

Learn more

▶ Article Scorpion sting overview

▶ Watch — AETCM video: Scorpion sting

▶ Scorpion sting video

Venom and pathophysiology

The venom contains free amino acids, serotonin, hyaluronidase and neurotoxic peptides. The toxins **open (delay inactivation of) sodium channels** at pre-synaptic nerve terminals and **block calcium-dependent potassium channels**, causing massive release of acetylcholine and **catecholamines** — an **autonomic storm**. Early parasympathetic effects are followed by sustained sympathetic (α-receptor) stimulation: hypertension, tachycardia, myocardial dysfunction ("catecholamine myocarditis"), **pulmonary oedema** and cold, clammy extremities; later hypotension and shock. Coagulation changes may lead to DIC, and a systemic inflammatory response-like syndrome is triggered by some species. Hyperglycaemia (insulin suppression), pancreatitis and hypokalaemia may occur.

Clinical features and grading

Grade	Features	Typical timing
Grade I — local	**Severe, burning local pain** radiating along the dermatome, local paraesthesia, mild swelling; no systemic signs	Immediately; lasts hours (up to 24 h)
Grade II — autonomic storm	**Cholinergic**: vomiting, profuse **sweating**, hypersalivation, lacrimation, **priapism**, diarrhoea, bradycardia, hypotension (early, 1–2 h). **Adrenergic**: tachycardia, **hypertension**, cold extremities, mydriasis, restlessness	Within 30 min – 4 h; vomiting, sweating, salivation, priapism and bradycardia are early diagnostic signs
Grade III — cardiovascular	**Pulmonary oedema** (breathlessness, crepitations, pink frothy sputum), myocardial dysfunction, arrhythmias, hypotension, **shock**	Pulmonary oedema 30 min – 3 h after sting (up to 6–10 h)
Grade IV — multi-organ	Grade III + CNS involvement (agitation, confusion, seizures, coma, stroke), DIC, AKI, MODS	Hours

Anaphylaxis to the sting is possible but very rare. ECG: sinus tachycardia, ST-T changes, peaked T, Q waves (pseudo-infarction), conduction blocks; troponin and CK-MB may rise; echocardiography shows global hypokinesia (reversible).

CHAPTER 17 · SCORPION MANAGEMENT

Scorpion Sting — Treatment Protocol

Prazosin first line; scorpion antivenom for systemic envenoming; inodilators for pulmonary oedema

Scorpion sting — ABC, IV line, monitor pulse, BP, SpO_2, ECG; observe all children and Grade ≥ II for at least 24 h

Grade I (pain only)
Ice packs · local infiltration of **2% lignocaine** (1–2 mL, without adrenaline) or digital block · paracetamol · observe 6–12 h (children 24 h) for autonomic signs

Grade II (autonomic storm)
Prazosin 30 µg/kg/dose orally (adult ≈ 500 µg–1 mg) · repeat **3-hourly** then 6-hourly until extremities are warm and dry · ± scorpion antivenom

Grade III–IV (pulmonary oedema / shock / CNS): oxygen, CPAP/NIV or intubation · prazosin (if not hypotensive) · **dobutamine** 5–15 µg/kg/min ± **nitroglycerin** 5–20 µg/min (titrate) or **sodium nitroprusside** 0.3–5 µg/kg/min · furosemide if fluid overloaded · scorpion antivenom · ICU

Prazosin — the drug of choice

- A **competitive post-synaptic α_1-adrenoceptor blocker**; reverses the α-mediated vasoconstriction, hypertension and left-ventricular afterload, and acts as a pharmacological antidote to the catecholamine storm. It reduced mortality from scorpion sting in Maharashtra from about 30% to around 1%.
- Available as scored **1 mg / 2 mg tablets** (use plain, **not sustained-release**); crush and give orally or by NG tube.
- **Dose: 30 µg/kg/dose** (children and adults; adult usually 500 µg–1 mg). Repeat the same dose at **3 hours** if features persist, then every 6 hours until the extremities are warm, dry and sweating stops (usually 1–3 doses; maximum about 8 doses).
- Can be given irrespective of BP provided there is no hypovolaemia; give **lying down** — watch for **first-dose hypotension** (treat with IV saline).

Supportive drugs and doses

Hypotension	IV normal saline **20–30 mL/kg** bolus (careful in pulmonary oedema); dopamine/ dobutamine if refractory
Pulmonary oedema	Dobutamine 5–15 µg/kg/min; NTG or nitroprusside; furosemide (only if fluid overload); NIV/ventilation; diuretics reduce fluid overload
Pain	Ice, lignocaine infiltration, paracetamol 15 mg/kg; NSAIDs for severe local pain in Grade I only
Seizures	Midazolam/lorazepam; check glucose and BP
Hypertensive crisis	Prazosin; nitroprusside/NTG if severe

Avoid

Atropine (worsens tachycardia, hypertension and pulmonary oedema — the cholinergic phase is self-limiting), **nifedipine** (reflex tachycardia), routine steroids and antihistamines, opioids/sedatives in children with respiratory compromise, and **digoxin**.

Landmark article (J Clin Diagn Res 2025;19(1))

Efficacy and safety of high-dose versus standard-dose prazosin in paediatric scorpion envenomation: a single-blinded randomised controlled study. Abbadi AG, Bijapure HR, Doddihal CR, et al. *J Clin Diagn Res 2025;19(1)*.

Message: A recent Indian paediatric RCT (86 children, grade II–III stings) compared prazosin 30 µg/kg 3-hourly with higher loading doses (60–90 µg/kg); the authors found high-dose prazosin safe — but this is a small single-centre trial, and 30 µg/kg/dose remains the standard.

▶ Read article

Scorpion antivenom (SAV)

- Indian product: **Haffkine** monovalent, lyophilised, enzyme-refined equine immunoglobulin against *H. (Buthus) tamulus* venom; reconstitute with 10 mL sterile water; 1 mL of reconstituted serum neutralises 1.0 mg of red scorpion venom.
- **Dose** (IAP/JAPI protocols): **30 mL (3 vials)** diluted in 100 mL normal saline as IV infusion over 30 min for Grade II and above, irrespective of age and weight (the label states 1 vial initially, further doses if symptoms persist).
- An Indian randomised trial (Bawaskar, BMJ 2011) showed **prazosin + SAV** gave faster recovery than prazosin alone; SAV works best given early (within 2–4 h). It neutralises circulating venom but cannot reverse established myocardial damage.
- Keep adrenaline ready — equine product; sensitivity tests are mentioned on the label but are poorly predictive.

Remember

Cold sweaty extremities + priapism + vomiting after a sting = autonomic storm. Give prazosin early — do not wait for pulmonary oedema. Warm dry extremities signal recovery.

Pearls

Black scorpion stings need pain relief, not prazosin, unless autonomic signs appear. Hyperglycaemia is common and settles without insulin. Myocardial dysfunction usually recovers within days.

Prevention

Shake out shoes, clothes and bedding; sleep on a cot; seal cracks in mud walls; use a torch at night; keep firewood and stones away from the house.

CHAPTER 18 · HYMENOPTERA

Bees, Wasps, Hornets and Ants

Local reactions in everyone, allergy in the sensitised, and toxicity after mass attacks

Stinging insects belong to the order **Hymenoptera** (class Insecta). Major subgroups:

- **Apids** — honeybees (*Apis cerana*, rock/giant bee *A. dorsata*, *A. florea*), bumblebees. The honeybee's **barbed sting** stays in the skin with its venom sac; it has its own ganglion and continues to pump venom for several minutes.
- **Vespids** — wasps, yellow jackets and **hornets** (*Vespa* spp., including the large *V. mandarinia* and *V. tropica* of the North-East and the Ghats). Smooth stings — can sting repeatedly.
- **Formicids** — ants, including fire ants (*Solenopsis*) — bite and sting, producing sterile pustules.

Venom components	
Melittin (bee)	Membrane lysis, haemolysis, pain
Phospholipase A_2, hyaluronidase	Major allergens; tissue spread, rhabdomyolysis
Apamin, MCD peptide	Neurotoxic, mast-cell degranulation
Histamine, serotonin, kinins, acetylcholine	Pain, itching, vasodilatation
Mastoparan, antigen 5 (wasp)	Mast-cell activation; major vespid allergen

Many bees and wasps have two poison glands — one acidic, one alkaline — whose combination is strongly irritant.

▶ **Watch — AETCM video: Bee sting**

▶ Bee sting video

Types of reaction

Reaction	Features	Treatment
Local	Immediate burning pain, itching, erythema, swelling and induration up to a few cm, settling in hours	Remove sting, ice, analgesic, antihistamine
Large local	Swelling > 10 cm, contiguous with sting site, peaks at 24–48 h, lasts up to a week (late-phase IgE). **Swelling alone is not anaphylaxis**	Elevation, oral antihistamine, short course of oral prednisolone (0.5–1 mg/kg × 3–5 days)
Systemic allergic (anaphylaxis)	Generalised urticaria, angioedema, bronchospasm, laryngeal oedema, vomiting, refractory hypotension and shock — usually within 10–30 min	Adrenaline IM (see below), airway, fluids
Toxic (mass envenomation)	Usually > 50 stings (children fewer): vomiting, diarrhoea, hypotension, **intravascular haemolysis**, **rhabdomyolysis**, **AKI**, hepatic injury, myocardial injury, thrombocytopenia, DIC, seizures, ARDS — onset hours to 1–2 days	Admit; fluids, alkalinisation for pigmenturia, dialysis
Unusual	Kounis syndrome (allergic acute coronary syndrome), stroke, serum-sickness-like illness (1–2 weeks), Guillain–Barré, vasculitis; stings in the mouth/throat → airway swelling; corneal stings	Specific

Management

1. **ABC** — airway assessment for signs of allergy is the priority.
2. **Remove stingers quickly** by scraping sideways with a thin dull edge (edge of a card, blunt side of a scalpel, table knife) or by pulling — speed matters more than technique.
3. Wash; cold compress or an ice cube wrapped in cloth for pain, burning and itching.
4. Intradermal/local injection of **1% lignocaine** (with or without 1:100,000 adrenaline) for severe pain; NSAIDs/paracetamol.
5. Topical steroid cream; oral antihistamine (cetirizine 10 mg) for itching.
6. Tetanus prophylaxis; watch for local infection and ulceration later.

Anaphylaxis

Adrenaline 0.5 mg IM (0.5 mL of 1:1000) in the thigh, children **0.01 mg/kg** (max 0.5 mg), repeat every 5–15 min; oxygen; lie flat; IV fluids 20 mL/kg; chlorpheniramine 10 mg IV; hydrocortisone 200 mg IV; nebulised salbutamol; adrenaline infusion or vasopressors if refractory. Observe at least 6–12 h (biphasic reactions).

Mass stings — protocol

Count stings (estimate) · remove all stings · CBC, smear, LDH, CK, creatinine, K^+, LFT, urine for haemoglobin/myoglobin, ECG, troponin · IV fluids to urine output 2–3 mL/kg/h (adults 200–300 mL/h) if no overload · monitor 24–72 h · early haemodialysis for AKI/hyperkalaemia · plasmapheresis has been used in severe haemolysis.

After an allergic reaction

Refer to an allergist for **venom immunotherapy** (highly effective in preventing future anaphylaxis); prescribe an adrenaline auto-injector or teach drawing up 0.5 mL of 1:1000 for IM use; advise avoidance of perfumes and bright clothing near hives.

▶ Article Bee sting ▶ Images Hornet ▶ Images Fire ant

CHAPTER 19 · ARACHNIDS & MYRIAPODS

Spiders, Centipedes and Millipedes

Mostly painful, rarely dangerous — know the exceptions

Spider bites

Spider	Clinical picture	Management
Widow spiders (*Latrodectus* — Indian red-back *L. hasselti* and brown widow *L. geometricus*)	**Latrodectism**: pin-prick bite, then within 30–60 min spreading pain, sweating at the bite, regional then generalised **muscle cramps and spasms**, abdominal rigidity mimicking an acute abdomen, chest tightness, hypertension, tachycardia, nausea, restlessness; lasts 1–3 days. Venom α-latrotoxin causes massive neurotransmitter release	Analgesia (paracetamol, opioids titrated), benzodiazepines for spasms; calcium gluconate is no longer recommended; red-back antivenom (Australia) not available in India; observe for hypertension
Tarantulas (*Poecilotheria*, ornamental tarantulas)	Painful bite; rarely delayed severe muscle cramps lasting days (reports from India/Sri Lanka); urticating hairs of some species cause dermatitis and eye inflammation	Analgesia, muscle relaxants; eye exposure — irrigation, topical steroid by ophthalmologist
Recluse-type spiders (*Loxosceles* — rarely reported)	Necrotic arachnidism: painless bite → blue-grey ischaemic centre with red halo ("red, white and blue") → necrotic ulcer; rarely haemolysis, AKI	Wound care, tetanus, analgesia; delayed debridement; monitor Hb/urine in systemic cases
Other spiders (jumping, wolf, huntsman)	Local pain, redness, small papule; secondary infection	Clean, cold pack, analgesic, antihistamine

Pearl

Most "spider bites" with an expanding red necrotic lesion are actually **skin infections** (MRSA/Staphylococcal boils), Paederus dermatitis or cutaneous anthrax — examine carefully before blaming a spider. ▶ Images ▶ Images

Centipede bites

- Giant centipedes (*Scolopendra*) bite with modified front legs (forcipules) — two puncture marks in a chevron.
- Immediate **intense burning pain**, swelling, erythema, lymphangitis, local blistering and occasionally necrosis; symptoms usually settle in 1–2 days.
- Rare systemic effects: vomiting, palpitations, hypotension, **myocardial ischaemia/infarction**, rhabdomyolysis, AKI, anaphylaxis.

Treatment

Wash; **hot water immersion** (40–45 °C, as hot as tolerated, test with an unaffected hand) for 20–30 min; local lignocaine infiltration or nerve block; paracetamol/NSAIDs; antihistamine; tetanus; antibiotics only if infected; ECG if chest pain. ▶ Images

Millipedes

Millipedes do not bite; they secrete defensive fluids (benzoquinones, hydrogen cyanide) that cause **brown-black or purple staining of the skin** (often mistaken for gangrene or bruising in children's feet), burning and blistering, and severe conjunctivitis/keratitis if in the eye. **Treatment**: wash with soap and plenty of water (alcohol/ether can help remove pigment); copious eye irrigation and ophthalmology review; the staining fades over weeks. Reassure parents — circulation is normal.

CHAPTER 20 · BLISTERING INSECTS

Beetles, Caterpillars and Blistering Insects

Acid fly (Paederus) dermatitis, blister beetles and caterpillar hair dermatitis

Acid fly — Paederus beetle (rove beetle, Nairobi fly)

- Small (7–10 mm) orange-and-black beetles attracted to lights in the monsoon and post-monsoon; outbreaks in hostels, campuses and new townships in Kerala, Karnataka, Tamil Nadu, Assam and elsewhere.
- Unlike many insects, **Paederus beetles do not bite or sting**. When crushed on the skin they release **pederin** — a potent, **non-proteinaceous vesicant** that inhibits protein synthesis and mitosis.
- **Paederus dermatitis**: 12–36 h after contact, burning linear or irregular erythema, vesicles and pustules on exposed skin (neck, face, arms), "**kissing lesions**" in skin folds (elbow flexure), periorbital involvement ("Nairobi eye"); heals with hyperpigmentation in 1–3 weeks.

Treatment

- If exposed, **wash immediately with soap and water** to remove the toxin before it penetrates; do not crush or rub the beetle — brush it off.
- Cool compresses; **topical steroid** (hydrocortisone 1% for face; betamethasone/ mometasone elsewhere) to reduce inflammation.
- **Topical antibiotic** — fusidic acid or mupirocin — if blisters are infected.
- **Oral antihistamine** — cetirizine 10 mg or loratadine 10 mg daily — for itching.
- Tincture of iodine applied shortly after contact may help neutralise pederin.
- Eye involvement: irrigation, ophthalmology review.

Prevention

Use bed nets, close windows at night in outbreak seasons, use yellow/low-attraction lights, do not slap insects on the skin. ▶ Images

Blister beetles (Meloidae)

Blister beetles release **cantharidin** on contact, producing painless thin-walled blisters within hours (blister beetle dermatitis), usually healing in a week. **Ingestion** of cantharidin (historically "Spanish fly" aphrodisiac) is dangerous — oropharyngeal burns, haematemesis, haematuria, **AKI**, priapism, seizures. Treatment is supportive: wash skin, drain large blisters aseptically, topical antibiotic; for ingestion — fluids, renal monitoring, supportive care. ▶ Images

Caterpillar dermatitis (erucism) and lepidopterism

- Hairy caterpillars (tussock moths, processionary and hairy caterpillar outbreaks on castor, teak and mango trees) shed **urticating hairs** (setae) that embed in skin.
- Itchy urticarial papules and wheals, linear lesions, burning; airborne hairs cause conjunctivitis, rhinitis and wheeze ("lepidopterism").
- **Ophthalmia nodosa** — hairs migrating into the cornea and globe cause nodular conjunctivitis/ uveitis.

Treatment

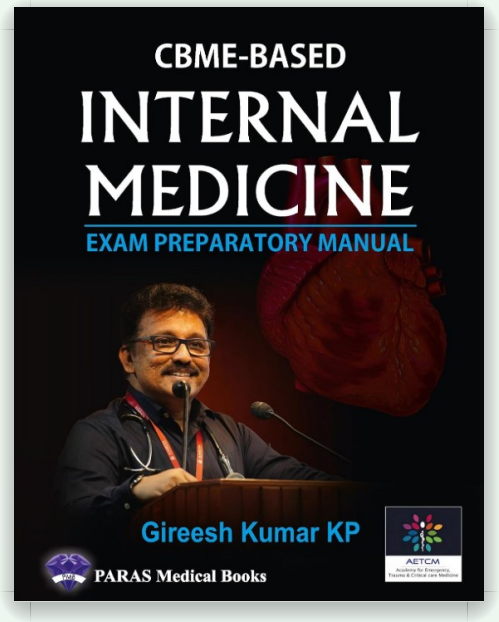
Remove hairs with **adhesive tape stripping** (repeat with fresh tape), then wash; cold compress; calamine; topical steroid; oral antihistamine; short oral prednisolone for extensive reactions; inhaled bronchodilator for wheeze; slit-lamp examination and removal of hairs for eye involvement.

▶ Article

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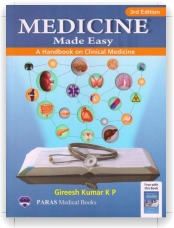
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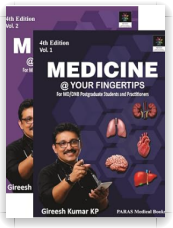
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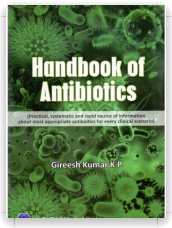
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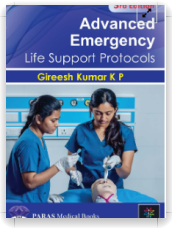
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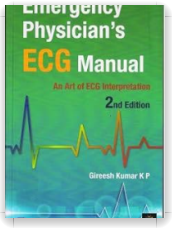
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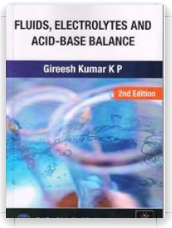
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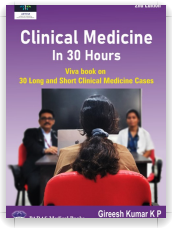
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CHAPTER 21 · MARINE

Marine Envenomations

Jellyfish, stingrays, venomous fish, sea urchins and cone snails along India's 7,500 km coastline

Jellyfish and other cnidarians

- Tentacles carry millions of **nematocysts** (stinging capsules) that fire on contact.
- **Portuguese man o' war / bluebottle** (*Physalia*) washes ashore on the west coast (Goa, Maharashtra, Kerala) and Odisha coast in the monsoon: immediate severe pain, linear red welts, rarely systemic symptoms (vomiting, muscle cramps, breathlessness).
- **Box jellyfish** (cubozoans) occur in Indian waters; severe envenoming can cause cardiovascular collapse (Irukandji-like syndrome — delayed severe back pain, hypertension, sweating, pulmonary oedema).
- Sea anemones and fire corals — local stinging dermatitis.

First aid and treatment

1. Get the victim out of the water; ABC.
2. **Do not rub**; do not rinse with fresh water first (may fire nematocysts).
3. **Box jellyfish**: flood with **vinegar** (4–6% acetic acid) for 30 s to inactivate undischarged nematocysts. **Physalia**: **rinse with sea water** — vinegar is not recommended for bluebottle.
4. Pick off remaining tentacles with gloved fingers/ forceps.
5. **Hot water immersion** (≈ 45 °C) for 20 min relieves pain; ice packs if hot water unavailable.
6. Analgesics, antihistamine, topical steroid; adrenaline for anaphylaxis; supportive ICU care for Irukandji-like syndrome.

Venomous fish, stingrays and other marine creatures

Creature	Injury and features	Management
Stingrays	Barbed, serrated tail spine — deep lacerations/punctures of foot or leg while wading; **excruciating pain**, bleeding, swelling; rarely penetrating chest/abdominal injury; retained barb fragments; infection	Irrigate, **hot water immersion** 30–90 min, analgesia/local block; X-ray or ultrasound for retained spine; explore and debride; leave wound open; tetanus; antibiotics
Catfish (*Plotosus*, marine and freshwater catfish) — common in Indian fishermen	Pectoral/dorsal spine puncture of hands: severe throbbing pain, swelling, bleeding; retained spine; infection (Vibrio, Aeromonas)	Hot water, remove spine, X-ray, debride, antibiotics; tetanus
Stonefish, scorpionfish, lionfish	Dorsal spines; agonising pain, swelling, necrosis, hypotension, arrhythmia, collapse (stonefish)	Hot water immersion, analgesia, regional block; stonefish antivenom (Australia) rarely available
Sea urchins	Brittle spines break in the skin; pain, tattoo-like discolouration, granulomas, synovitis if near a joint	Hot water; remove accessible spines; X-ray; surgical removal near joints
Cone snails (*Conus*)	Harpoon-like tooth — local numbness, then **paralysis** (conotoxins), respiratory failure	Pressure immobilisation, ventilatory support; no antivenom
Blue-ringed octopus	Painless bite; tetrodotoxin → rapid paralysis	Ventilate until recovery (hours)
Sea snakes	See Chapter 3 (myotoxic + neurotoxic)	Supportive; no Indian ASV

Marine wound infections

Wounds contaminated with sea or brackish water can be infected with ***Vibrio vulnificus*** (rapidly spreading cellulitis, haemorrhagic bullae, septic shock — especially in cirrhosis/diabetes) and *Aeromonas* (fresh water). Empirical antibiotics: **doxycycline 100 mg 12-hourly + ceftazidime/ceftriaxone** (or ciprofloxacin); early surgical debridement for necrotising infection.

▶ Images box jellyfish ▶ Images man o' war ▶ Article stingray injury ▶ Images catfish ▶ Images stonefish ▶ Images sea urchin ▶ Images cone snail

CHAPTER 22 · ANIMAL BITES

Animal and Human Bites

Wound care, infection prophylaxis and the animals that matter in India

India records several million animal bites every year, about 90% by dogs; India accounts for about one-third of global rabies deaths (WHO). Every bite needs **wound care, tetanus assessment, infection prophylaxis where indicated, and a rabies risk assessment**.

▶ **Watch — AETCM video: Dog bite**

▶ Dog bite video

Wound management — the first and most important step

1. **Wash immediately** and thoroughly with soap/detergent under running water for **15 minutes** — this alone greatly reduces rabies risk.
2. Apply an antiseptic — **povidone-iodine** or alcohol (70%).
3. **Avoid** chilli, turmeric, oil, lime, herbs, soil or other irritants; do not touch the wound with bare hands.
4. **Avoid suturing** where possible; if essential (face, gaping wounds, bleeding), suture loosely **after** infiltrating RIG; delayed primary closure for heavily contaminated wounds.
5. Avoid tight dressings; elevate injured hands.
6. Tetanus toxoid ± tetanus immunoglobulin as per status.

Antibiotic prophylaxis (3–5 days) — consider for	
High-risk wounds	Puncture wounds, cat bites, hand/foot/face/genital bites, wounds near joints or bones, crush injuries, delayed presentation (> 8 h), wounds requiring closure
High-risk hosts	Diabetes, cirrhosis, asplenia, immunosuppression, prosthetic valves/joints
Drug	**Amoxicillin–clavulanate** 625 mg 8-hourly (children 25 mg/kg/dose amoxicillin 8-hourly); penicillin allergy — doxycycline 100 mg 12-hourly (or cotrimoxazole) + metronidazole/clindamycin

Specific animals

Animal	Concerns	Key actions
Dog	Rabies; lacerations, crush; infection (*Pasteurella canis*, *Capnocytophaga canimorsus* — fulminant sepsis in asplenic/alcoholic patients), facial injuries in children	Rabies PEP by category; wound care; antibiotics for high-risk wounds
Cat	Deep punctures — **Pasteurella multocida** cellulitis within 24 h, tenosynovitis, septic arthritis; cat-scratch disease (*Bartonella*); rabies	Prophylactic amoxicillin–clavulanate for all cat bites; rabies PEP
Monkey	Rabies (category III in India), wound infection; macaque herpes B virus (very rare)	Rabies PEP with RIG; wound care
Human ("fight bite")	Clenched-fist injury over MCP joint — tendon/joint penetration, *Eikenella corrodens*, streptococci, anaerobes; HBV, HCV, HIV transmission (rare)	Explore, irrigate, do not suture, amoxicillin–clavulanate; hepatitis B/HIV risk assessment; X-ray for fracture/tooth
Rat and other rodents	**Rat-bite fever** (*Streptobacillus moniliformis*, *Spirillum minus*): fever, rash, migratory polyarthritis, endocarditis 3–10 days later; leptospirosis risk	Penicillin/amoxicillin or doxycycline; **rabies PEP not required** for rats, mice, squirrels, rabbits (national guideline)
Wild animals (jackal, fox, wolf, mongoose)	High rabies risk; severe lacerations	Category III PEP with RIG
Bats	Any bat exposure is treated as category III by WHO	PEP with RIG

Livestock (cattle, horse, pig), elephant	Crush, gore and penetrating injuries; rabies possible in rabid livestock	Trauma protocol; rabies assessment if animal sick

▶ Article dog bite ▶ Article rat-bite fever ▶ Article WHO rabies fact sheet

CHAPTER 23 · RABIES PROPHYLAXIS

Rabies Post-exposure Prophylaxis (PEP)

National Guidelines on Rabies Prophylaxis (NCDC/NRCP) in line with WHO 2018

Categories of exposure

Category	Type of contact with a suspect/confirmed rabid animal	Recommended PEP
I	Touching or feeding animals; licks on **intact** skin; contact of intact skin with secretions/excretions	**None** if history reliable (wash the area)
II	Nibbling of uncovered skin; minor scratches or abrasions **without bleeding**	**Wound care + anti-rabies vaccine** (no RIG)
III	Single or multiple **transdermal bites or scratches** (bleeding); licks on broken skin; contamination of **mucous membranes** with saliva; any bat exposure	**Wound care + RIG + vaccine** — all category III exposures should get RIG

Immunocompromised patients with category II exposure should be managed as category III. Start PEP immediately — do not wait for the outcome of animal observation; WHO allows PEP to be stopped if a dog or cat remains healthy for 10 days of observation.

Anti-rabies vaccine (cell-culture or embryonated-egg vaccines)

Intramuscular — Essen regimen (5 doses)

Schedule	Days **0, 3, 7, 14, 28** (1-1-1-1-1)
Dose	One full vial (0.5 mL or 1 mL as per product) per visit
Site	Deltoid (adults); anterolateral thigh (young children); **never gluteal**

Intradermal — updated Thai Red Cross (2-2-2-0-2)

Schedule	Days **0, 3, 7, 28**
Dose	**0.1 mL at 2 sites** per visit (left and right deltoid), using a 1 mL insulin/BCG syringe; must raise a bleb
Note	Only by trained staff at designated ARCs; an opened vial is used within 6–8 h (2–8 °C)

Rabies immunoglobulin (RIG) — day 0

Equine RIG (ERIG)	**40 IU/kg** body weight
Human RIG (HRIG)	**20 IU/kg** body weight
Rabies monoclonal antibodies	Licensed in India as an alternative; dose as per product (e.g. 3.33 IU/kg for the single-antibody product)
Technique	Infiltrate as much as possible **into and around all wounds**; dilute with saline for multiple wounds; any remainder need not be given IM (WHO 2018)
Timing	With the first vaccine dose; if unavailable, up to **day 7** after the first vaccine dose — not later
Safety	Purified ERIG: follow the product insert/ national guideline on skin testing; keep adrenaline ready

📰 **Landmark article (Clin Infect Dis 2021)**

A phase 3, randomized, open-label, non-inferiority trial evaluating anti-rabies monoclonal antibody cocktail (Twinrab) against human rabies immunoglobulin. Kansagra K, Parmar D, Mendiratta SK, et al. *Clin Infect Dis 2021.*

Message: In 308 Indian patients with WHO category III exposures, the monoclonal antibody cocktail (docaravimab + miromavimab, 40 IU/kg) was non-inferior to HRIG with the Essen vaccine schedule — supporting rabies monoclonal antibodies as an affordable alternative to RIG.

▶ Read article

Special situations

Situation	Recommendation

Previously fully vaccinated (PrEP or complete PEP)	Two booster doses on **days 0 and 3** (IM one vial, or ID 0.1 mL single site); **no RIG**
Pre-exposure prophylaxis (vets, lab staff, dog handlers, children in high-risk areas)	Days **0, 7 and 21 or 28** — IM one dose, or ID 0.1 mL (2 sites as per national schedule); antibody checks for continuous high-risk
Pregnancy, infants, elderly	No contraindication — full PEP
Delayed presentation	Start PEP regardless of interval since bite (incubation may be months)
Missed doses	Resume the schedule maintaining intervals; do not restart
Change of vaccine brand/route	Avoid where possible; if unavoidable, continue with the new product (as per national guideline)

Clinical rabies

Hydrophobia, aerophobia, agitation, hypersalivation, autonomic instability (furious) or ascending flaccid paralysis (paralytic) after a bite — no curative treatment. Provide humane palliative care with sedation and analgesia in a quiet room, barrier precautions for staff (PEP for exposed staff with mucosal/broken-skin contact), and counselling of family. Notify the case.

Official sources

▶ Guideline National Rabies Control Programme portal ▶ Guideline National Guidelines on Rabies Prophylaxis

Rabies vaccines and immunoglobulins used in India

Product type	Examples (generic)	Notes
Purified chick embryo cell vaccine (PCECV)	Cell-culture vaccine, 1 mL IM or 0.1 mL ID	Approved for ID use in India
Purified Vero cell rabies vaccine (PVRV)	0.5 mL IM or 0.1 mL ID	Approved for ID use
Human diploid cell vaccine (HDCV)	1 mL IM	Limited availability
Purified duck embryo vaccine (PDEV)	1 mL IM	Embryonated egg vaccine
Equine RIG (purified, $F(ab')_2$)	40 IU/kg	Cheaper; widely used
Human RIG	20 IU/kg	Expensive; preferred in high-risk for reactions
Rabies monoclonal antibody (RmAb)	As per product	Licensed alternative to RIG

Nerve-tissue vaccines are no longer used in India. Store all vaccines at 2–8 °C; never freeze.

CHAPTER 23 · AT A GLANCE

Rabies PEP at a Glance — Schedules and FAQs

Visual timeline for the anti-rabies clinic

Rabies vaccine schedules (numbers inside circles = IM vials or ID sites of 0.1 mL).

Question	Answer
The dog is vaccinated. Can I skip PEP?	Not on the owner's word alone in India; vaccination records may be unreliable. Start PEP; WHO allows stopping if the animal stays healthy for 10 days of observation.
The bite was 2 months ago. Is PEP still useful?	Yes — start the full course (with RIG for category III) irrespective of delay, provided no symptoms of rabies.
RIG is not available today.	Start the vaccine now; give RIG when available up to day 7 of the course. Never delay the vaccine for RIG.
Can RIG be given into the face or fingers?	Yes — infiltrate carefully; avoid compartment pressure in fingers; dilute to cover all wounds.
Should the wound be sutured?	Avoid; if unavoidable, infiltrate RIG first and suture loosely after a few hours.
Can pregnant women and infants get PEP?	Yes — rabies is fatal; there are no contraindications.
Rat, mouse, squirrel or rabbit bite?	Rabies PEP is not required under the national guideline; treat the wound and consider rat-bite fever.
Patient on steroids/immunosuppressed or HIV?	Give RIG even for category II, and full vaccine course; check antibody titre if possible.
Licks on intact skin by a pet?	Category I — wash; no PEP.
Can the patient eat normally, bathe, travel?	Yes — there are no dietary or activity restrictions during PEP.

Remember

Wash · Infiltrate · Vaccinate — fifteen minutes of soap and water, RIG into every category III wound, and a complete vaccine course.

CHAPTER 24 · MISCELLANEOUS

Ticks, Mites, Leeches, Toads and Lizards

Unusual bites and contacts that still reach the Emergency Medicine Department

Creature	Clinical problem	Management
Ticks	**Tick paralysis** — ascending flaccid paralysis (mimics Guillain–Barré) from a neurotoxin in an engorged tick, often hidden in the scalp/ear/groin of a child; recovers within hours of tick removal. Tick-borne infections: **Kyasanur Forest disease** (Western Ghats — Karnataka, Kerala, Goa, Maharashtra; haemorrhagic fever with neurological phase), Crimean–Congo haemorrhagic fever (Gujarat, Rajasthan), spotted fevers	Search the whole body; remove the tick with fine forceps close to the skin, steady traction (do not crush, burn or apply oil); supportive care; doxycycline for rickettsial fevers; KFD vaccine in endemic districts
Mites (chiggers)	**Scrub typhus** (*Orientia tsutsugamushi*) — fever, painless **eschar** (axilla, groin, waist), lymphadenopathy, pneumonitis, meningoencephalitis, AKI, MODS	**Doxycycline** 100 mg 12-hourly × 7 days (children 4.5 mg/kg/day) or azithromycin (pregnancy); IV doxycycline + azithromycin in severe cases
Leeches	Land leeches (trekkers, Western Ghats, NE) and aquatic leeches; **hirudin** causes prolonged oozing; leeches in the nose, pharynx, vagina or conjunctiva (drinking/bathing in streams) — epistaxis, haemoptysis, haematuria, airway obstruction, anaemia	Do not pull forcibly; detach with a fingernail at the oral sucker, or apply salt, vinegar or local anaesthetic; pressure for bleeding; endoscopic removal for internal sites; tetanus
Toads (*Duttaphrynus*, Asian common toad)	Parotoid gland secretion (bufadienolides — cardiac glycoside-like) — eye irritation on contact; **ingestion** (folk remedies, "toad eggs") causes vomiting, bradycardia, AV block, hyperkalaemia, arrhythmias like digoxin toxicity	Wash skin/eyes; ECG and K^+; atropine for bradycardia; **digoxin-specific Fab** if available for life-threatening arrhythmia; avoid calcium
House lizards (geckos) and garden lizards	Indian house geckos are **non-venomous**; "lizard fallen in food" — anxiety, vomiting (psychogenic or food contamination/ Salmonella), not poisoning; monitor lizard bites — painful lacerations, infection	Reassurance; observe; treat dehydration; wound care and antibiotics for bites
Frogs, earthworms, harmless insects	Non-toxic; anxiety and folk-remedy complications	Reassure; check for harmful first-aid practices

Pearl — the paralysed child with a tick

An afebrile child with rapidly ascending weakness and areflexia: before ordering nerve conduction or IVIG, part the hair and examine the scalp, ears, axillae and groin for a tick. Removal is curative. ▶ Article ▶ Article

See them

▶ Images Asian common toad ▶ Images Leech

CHAPTER 25 · UNIVERSAL PROTOCOL

Anaphylaxis After Bites and Stings

Recognition and treatment — the same steps for bee, wasp, ant, jellyfish, ASV or any antiserum

When is it anaphylaxis?

Anaphylaxis is **highly likely** when, within minutes to hours of exposure to a sting, bite or antiserum, there is acute involvement of skin/mucosa (urticaria, flushing, swollen lips or tongue) **plus** at least one of:

- **Airway/breathing** — hoarseness, stridor, wheeze, breathlessness, hypoxaemia
- **Circulation** — hypotension, collapse, syncope, incontinence
- **Persistent GI symptoms** — crampy abdominal pain, repeated vomiting

OR hypotension/bronchospasm/laryngeal involvement alone after exposure to a known allergen — skin signs may be absent in up to 10–20%.

Hypotension definitions	
Adults	Systolic BP < 90 mmHg or > 30% fall from baseline
Infants 1–12 months	SBP < 70 mmHg
1–10 years	SBP < 70 + (2 × age in years) mmHg
11–17 years	SBP < 90 mmHg

Pearl

Large local swelling after a bee sting, however impressive, is **not** anaphylaxis and does not need adrenaline. Isolated urticaria without other systems involved can be treated with an antihistamine and observation.

Treatment algorithm

Recognise anaphylaxis · **call for help** · remove the trigger (stop ASV/antiserum infusion; scrape out the sting)

▼

Adrenaline IM into the anterolateral thigh — 1:1000 (1 mg/mL): **0.01 mg/kg** (max 0.5 mg adult, 0.3 mg child) Adult/child > 12 y: 0.5 mg (0.5 mL) · 6–12 y: 0.3 mg (0.3 mL) · < 6 y: 0.15 mg (0.15 mL) · repeat every 5 min if no improvement

▼

Position: lie flat with legs raised; sit up if breathing difficulty; left lateral in pregnancy

High-flow oxygen; airway — early intubation for stridor/angioedema

IV access; crystalloid bolus 20 mL/kg (adult 500–1000 mL), repeat

▼

No response after 2 IM doses → **adrenaline infusion** 0.05–0.5 µg/kg/min (e.g. 1 mg in 100 mL NS = 10 µg/mL), titrate with monitoring; add noradrenaline/vasopressin if refractory; glucagon 1–2 mg IV (children 20–30 µg/kg) for β-blocked patients

▼

Bronchospasm: salbutamol 5 mg nebulised (repeat), ipratropium; consider magnesium sulphate 2 g IV over 20 min (adults)

Adjuncts (after adrenaline): chlorpheniramine 10 mg IV (0.2 mg/kg), hydrocortisone 200 mg IV (2–4 mg/kg) — do not delay adrenaline for them

▼

Observe at least 6–12 h (24 h if severe or needed repeat adrenaline) — biphasic reactions occur in up to 5–20%; discharge with written advice and allergy referral

Common errors

Giving antihistamine or steroid first and adrenaline late · SC instead of IM adrenaline in shock · IV bolus of 1 mg adrenaline outside cardiac arrest · not stopping the ASV infusion · discharging too early · forgetting to restart ASV in a patient who still needs it.

More

▶ Article Anaphylaxis — clinical overview

CHAPTER 26 · SUMMARY

Bites and Stings at a Glance

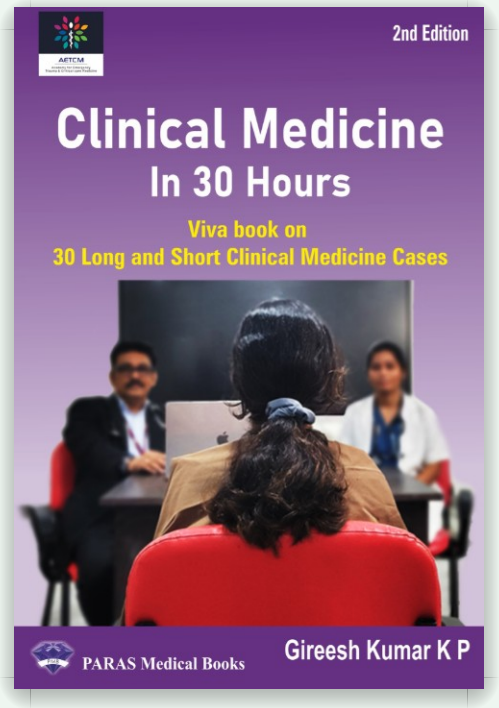
Key features, danger signs and specific treatment on one page

Creature	Typical features	Danger signs	Specific treatment
Cobra	Painful swelling, blisters, necrosis; ptosis within hours	Bulbar palsy, neck weakness, apnoea	ASV 10 (+10); AN trial; ventilation; wound care
Krait	Night bite while sleeping; no local signs; abdominal pain	Rapid descending paralysis; "coma"	ASV 10 (+10); ventilation for days; sodium
Russell's viper	Swelling, bleeding, unclotted 20WBCT	Shock, AKI, ICH, parotid swelling, chemosis	ASV 10, repeat 6-hourly by 20WBCT; fluids; dialysis
Saw-scaled viper	Painful swelling, bleeding, small snake	Severe bleeding, ICH	ASV 6–10, repeat 6-hourly
Hump-nosed pit viper	Local swelling, coagulopathy (W. Ghats)	AKI, TMA	No ASV if confirmed; supportive, dialysis
Sea snake	Painless bite at sea; myalgia	Dark urine, hyperkalaemia, paralysis	No Indian ASV; ventilation, K^+, dialysis
Red scorpion	Severe pain; sweating, vomiting, priapism, cold limbs	Pulmonary oedema, shock	Prazosin 30 µg/kg 3-hourly; SAV 3 vials; dobutamine/NTG
Bee / wasp / hornet	Pain, swelling; urticaria	Anaphylaxis; > 50 stings → haemolysis, AKI	Remove stings; adrenaline IM; fluids; dialysis
Widow spider	Spreading pain, cramps, sweating	Hypertension, abdominal rigidity	Analgesia, benzodiazepines
Centipede	Intense burning pain	Chest pain (rare MI)	Hot water, local anaesthetic
Paederus beetle	Linear burning vesicles, kissing lesions	Eye involvement	Wash, topical steroid, antihistamine
Caterpillar	Itchy wheals, linear lesions	Ophthalmia nodosa, wheeze	Tape stripping, steroid, antihistamine
Jellyfish	Linear welts, severe pain	Collapse (box jellyfish)	Vinegar (box) / sea water (Physalia); hot water
Stingray / catfish / stonefish	Puncture, agonising pain	Retained spine, infection, shock	Hot water 45 °C, X-ray, debride, antibiotics
Dog / cat / monkey	Lacerations, punctures	Rabies risk; Pasteurella cellulitis	Wash 15 min; RIG + vaccine; amoxicillin–clavulanate
Human bite	Fight bite over knuckle	Septic arthritis, tenosynovitis	Explore, no suture, amoxicillin–clavulanate
Rat	Small puncture	Fever, rash, arthritis (rat-bite fever)	Penicillin/doxycycline; no rabies PEP
Tick	Attached tick	Ascending paralysis; KFD, CCHF	Remove tick; supportive; doxycycline for rickettsiae
Chigger (mite)	Eschar, fever	Pneumonitis, meningoencephalitis, AKI	Doxycycline or azithromycin
Leech	Oozing bite; internal leech	Airway obstruction, anaemia	Salt/local anaesthetic, endoscopic removal
Toad (ingested)	Vomiting, bradycardia	AV block, hyperkalaemia	Atropine; digoxin-specific Fab

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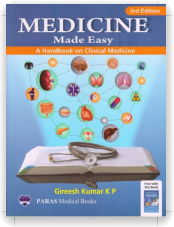
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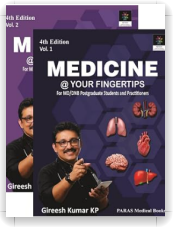
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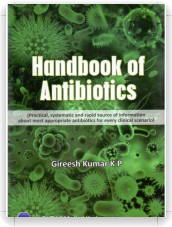
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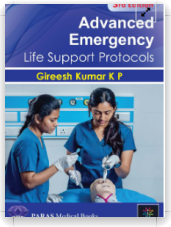
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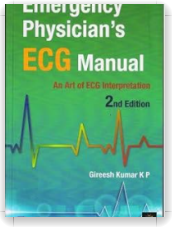
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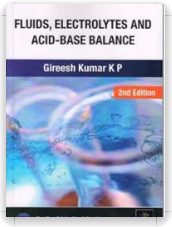
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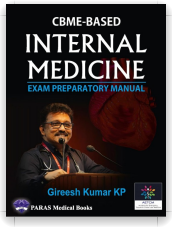
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CHAPTER 26 · DOSE CARD

Drug Dose Card — Other Bites and Stings

Adult and paediatric doses (verify with current formulary)

Drug	Adult	Child	Indication
Prazosin	30 µg/kg/dose (≈ 0.5–1 mg) orally; repeat 3 h, then 6-hourly	30 µg/kg/dose	Scorpion autonomic storm
Scorpion antivenom (Haffkine)	3 vials (30 mL) in 100 mL NS over 30 min	Same	Grade ≥ II scorpion envenoming
Dobutamine	5–15 µg/kg/min	5–15 µg/kg/min	Scorpion myocardial dysfunction
Nitroglycerin	5–20 µg/min IV, titrate	0.5–5 µg/kg/min	Scorpion pulmonary oedema with hypertension
Sodium nitroprusside	0.3–5 µg/kg/min	0.3–5 µg/kg/min	Severe hypertension/pulmonary oedema
Adrenaline (anaphylaxis)	0.5 mg IM (1:1000), repeat 5 min	0.01 mg/kg IM (0.15–0.3 mg)	Any anaphylaxis
Cetirizine	10 mg daily	2.5–5 mg (by age)	Itch, urticaria
Chlorpheniramine	10 mg IV/IM	0.2 mg/kg	Acute allergic reaction
Prednisolone	0.5–1 mg/kg/day × 3–5 days	1 mg/kg/day	Large local reactions, caterpillar dermatitis
Lignocaine 1–2%	1–5 mL local infiltration/digital block (max 3 mg/kg plain)	Max 3 mg/kg	Scorpion, bee, centipede, fish stings
Midazolam	2–5 mg IV titrated	0.1–0.2 mg/kg	Latrodectism spasms, seizures
Amoxicillin–clavulanate	625 mg 8-hourly orally (1.2 g IV)	25 mg/kg/dose 8-hourly	Animal and human bites
Doxycycline	100 mg 12-hourly	2.2 mg/kg 12-hourly	Scrub typhus, rickettsiae, Vibrio (with ceftazidime), rat-bite fever
Azithromycin	500 mg daily × 5 days	10 mg/kg/day	Scrub typhus in pregnancy/children
Rabies vaccine (IM)	1 vial days 0, 3, 7, 14, 28	Same	Category II/III
Rabies vaccine (ID)	0.1 mL × 2 sites days 0, 3, 7, 28	Same	Category II/III
ERIG / HRIG	40 IU/kg / 20 IU/kg into wounds	Same per kg	Category III
Tetanus (Td/TT; TIG)	Td 0.5 mL IM; TIG 250 IU IM for dirty wounds in unimmunised	Age-appropriate	All bites and stings
Paracetamol	1 g 6-hourly (max 4 g/day)	15 mg/kg/dose	Pain, fever

Hot water immersion — how to do it safely

Water at about 40–45 °C (tested by the rescuer's hand or a thermometer — the patient's injured limb may have impaired sensation); immerse for 20–90 minutes until pain eases; re-immerse if pain recurs. Used for stingray, catfish, stonefish, sea urchin, jellyfish (after initial measures) and centipede injuries. Never on numb limbs without checking temperature — scald risk.

APPENDICES

Tools for the Bedside

Case record proforma, ASV observation chart, myths and facts, glossary, viva questions and MCQs, visual atlas with QR codes, standard guidelines, and the subject index.

APPENDIX A

Snakebite Case Record Proforma

Adapted from the STG snakebite examination proforma — photocopy for every patient

Name: _____ Age/Sex: _____ Weight: ___ kg	IP No.: _____ Date/time of arrival: _____
Address / village: _____	Brought by: _____ Referred from: _____
Date and **time of bite**: _____ Place: field / house / bed (floor) / road / water	Activity at time of bite: _____
Site of bite: _____ Fang marks: Y / N	Snake seen: Y / N Photo: Y / N Identified as: _____
First aid: splint / tourniquet (time ___) / incision / suction / herbal / other	ASV given before arrival: Y / N — vials ___ time ___
Previous snakebite / ASV / equine serum: Y / N	Atopy / asthma: Y / N Pregnancy: Y / N LMP: _____
Tetanus immunisation: up to date / not / unknown	Drugs (anticoagulants, antiplatelets, β-blockers): _____

Symptoms (tick)

☐ Local pain	☐ Swelling	☐ Bleeding from bite	☐ Gum bleeding/epistaxis	☐ Haematuria / red urine
☐ Vomiting	☐ Abdominal pain	☐ Heavy eyelids	☐ Double vision	☐ Difficulty swallowing
☐ Breathlessness	☐ Reduced urine	☐ Dark brown urine	☐ Muscle pain	☐ Syncope/drowsiness

Examination on arrival

Pulse ___ BP ___/___ RR ___ SpO_2 ___% Temp ___	GCS ___ Pupils ___ Glucose ___
Swelling grade (0–5): ___ Edge marked at ___ (time)	Circumference: bite level ___ cm, 10 cm above ___ cm
Blisters / necrosis / lymph nodes: _____	Distal pulses: present / absent Compartment tense: Y / N
Ptosis: R ___ L ___ Ophthalmoplegia: Y / N	Neck lift: possible / not SBC: ___ Breath-hold: ___ s
Bulbar palsy / pooling of saliva: Y / N	Bleeding sites: _____
Parotid swelling / chemosis: Y / N	Urine colour: _____ Output: ___ mL/h

Investigations

20WBCT 0 h: C / NC	1 h: C / NC	2 h: C / NC	3 h: C / NC
6 h: C / NC	12 h: C / NC	18 h: C / NC	24 h: C / NC
Hb ___ TLC ___ Plt ___	PT/INR ___ aPTT ___	Fibrinogen ___ D-dimer ___	Urea ___ Creat ___ K^+ ___ Na^+ ___
CK ___ LDH ___	Urine: blood ___ RBC ___	ECG: _____	Group & X-match sent: Y / N

Assessment and plan

Syndrome: ☐ None/dry ☐ Local ☐ Haemotoxic ☐ Neurotoxic ☐ Renal ☐ Myotoxic	Probable snake: _____
ASV indicated: Y / N Reason: _____	Premedication: adrenaline 0.25 mg SC Y / N
ASV batch/manufacturer: _____ Expiry _____	AN trial: done / not Response: + / –
Doctor's name & signature: _____	Police intimation (if death/MLC): _____

APPENDIX B

ASV Administration and Observation Chart

One row per dose; observations every 5 min for 30 min, then every 15 min for 2 hours

Dose no.	Date/time started	Vials	Diluent & volume	Rate / duration	Reason (20WBCT/ neuro)	Reaction (Y/N, type)	Treatment of reaction	Sign
1								
2								
3								
4								
5								
6								
Total vials								

Observation record

Time	Pulse	BP	RR	SpO_2	Ptosis	SBC	Neck lift	Swelling edge	Bleeding	Urine mL	20WBCT	Skin/itch	Init.
0 min													
5 min													
10 min													
15 min													
20 min													
25 min													
30 min													
45 min													
1 h													
1 h 15													
1 h 30													
1 h 45													
2 h													
3 h													
4 h													
6 h													
12 h													
18 h													
24 h													

Stop the infusion and call the doctor if

Itching, rash, cough, wheeze, lip/tongue swelling, vomiting, fall in BP > 20 mmHg, pulse > 120, rigors or fever, falling SpO_2, new ptosis or difficulty swallowing, fresh bleeding.

APPENDIX C

Myths and Facts about Snakebite

For patient and community education

Myth	Fact
A tight tourniquet stops the poison spreading.	It does not stop venom and causes gangrene; release may cause sudden collapse. Splint the limb instead.
Cutting the wound and sucking out venom helps.	It removes almost no venom and causes bleeding, infection and nerve/tendon damage.
Black stones, herbs, mantras and "snake stones" cure snakebite.	Only ASV neutralises venom; delay for such practices kills.
If there is no pain or swelling, the bite is harmless.	Krait bites are painless and often invisible, but fatal.
All snakes are venomous.	About 70% of bites in India are by non-venomous snakes — but all bites need hospital observation.
Snakes take revenge if their mate is killed; snakes drink milk.	Snakes have no such behaviour; they do not drink milk.
A test dose tells you whether ASV is safe.	Test doses do not predict reactions and waste time.
Children need a smaller ASV dose.	Children need the same dose as adults.
ASV must be given within the first few hours or it is useless.	ASV is useful as long as signs of systemic envenoming persist, even days later.
A dead snake is safe to handle.	Reflex bites can occur for an hour or more after death — even from a severed head.
Fixed pupils and no response mean the patient is brain dead.	Complete paralysis from elapid venom mimics brain death; ventilate and wait.
Snakes chase people.	Snakes bite in defence when stepped on, cornered or handled.
Alcohol, painkillers (NSAIDs) or hot fomentation help.	They worsen bleeding and tissue damage.
A two-headed snake (sand boa) brings wealth.	Sand boas are harmless and protected; trading them is a crime under the Wildlife Protection Act.

The three messages for every village

Sleep on a cot under a net · Carry a torch at night · Reach a hospital with ASV immediately — no tourniquet, no healer.

APPENDIX D

Glossary of Toxinology Terms

Words used in this book, explained simply

Antivenom (antivenin)	Purified antibodies (usually equine $F(ab')_2$) that bind and neutralise venom toxins
Dry bite	A bite by a venomous snake in which no venom is injected
Envenoming (envenomation)	Clinical effects caused by venom injected into the body
Fangs	Modified teeth that deliver venom — fixed front (elapids), hinged front (vipers), grooved rear (colubrids)
Haemorrhagins	Venom metalloproteinases that damage capillary walls and cause bleeding
Loreal pit	Heat-sensing organ between the eye and nostril of pit vipers
Monovalent / polyvalent	Antivenom against one species / several species (Indian ASV is polyvalent against the Big Four)
Nematocyst	Stinging capsule of jellyfish and other cnidarians
Neurotoxin — pre-/post-synaptic	Toxin acting on the nerve terminal (pre-synaptic, e.g. β-bungarotoxin) or on the muscle ACh receptor (post-synaptic, e.g. cobra α-neurotoxin)
Paraspecific neutralisation	Partial neutralisation by an antivenom of venom from a species not used in its production
Pressure immobilisation	Firm crepe bandaging of the whole bitten limb with a splint to slow lymphatic spread of elapid venom
Serum sickness	Immune-complex reaction 1–12 days after antiserum: fever, rash, arthralgia, lymphadenopathy
Syndromic approach	Inferring the likely snake from the pattern of clinical features when the snake is not identified
Thrombotic microangiopathy	Haemolysis with fragmented red cells, low platelets and small-vessel thrombosis causing AKI
Toxidrome	A recognisable cluster of symptoms and signs caused by a toxin or class of toxins
Toxinology	The science of toxins produced by living organisms — venoms of animals, and toxins of plants and microbes
Venom vs poison	Venom is injected (bite or sting); a poison is absorbed when eaten, touched or inhaled — snakes are venomous, toads are poisonous
VICC	Venom-induced consumption coagulopathy — depletion of fibrinogen and clotting factors by procoagulant venom
Urticating hairs (setae)	Irritant barbed hairs of caterpillars and some tarantulas

APPENDIX E

Viva Questions and MCQs

Quick revision with short answers

Viva questions

1. **Name the Big Four.** Spectacled cobra, common krait, Russell's viper, saw-scaled viper.
2. **How much venom does one vial neutralise?** 10 mL × 0.6 mg = 6 mg cobra or Russell's viper; 4.5 mg krait or saw-scaled viper.
3. **Why is there a 6-hour gap between ASV doses in viper bites?** The liver needs about 6 h to restore clotting factors after venom is neutralised.
4. **What is the earliest sign of neurotoxicity?** Ptosis.
5. **Why does neostigmine help in cobra but not in krait bites?** Cobra α-neurotoxin is post-synaptic and competitive; krait β-bungarotoxin is pre-synaptic and destroys the nerve terminal.
6. **Three signs of capillary leak in Russell's viper bite?** Parotid swelling, chemosis, effusions/hypotension.
7. **Name snakes for which Indian ASV is ineffective.** Hump-nosed pit viper, green/Malabar pit vipers, sea snakes, king cobra (partial), Sind krait (poor).
8. **Criteria for fasciotomy?** Clinical compartment syndrome + pressure > 40 mmHg + corrected coagulopathy.
9. **Late endocrine complication after Russell's viper bite?** Hypopituitarism (Sheehan-like).
10. **Drug of choice in scorpion autonomic storm?** Prazosin 30 µg/kg/dose.
11. **Category III rabies exposure needs?** Wound wash, RIG infiltration and vaccine.
12. **Intradermal rabies schedule in India?** Updated Thai Red Cross 2-2-2-0-2: 0.1 mL at 2 sites on days 0, 3, 7, 28.

MCQs

1. The 20WBCT is read after: (a) 10 min (b) 20 min (c) 30 min (d) 60 min
Answer: (b)

2. Maximum recommended ASV for neurotoxic envenoming (STG): (a) 10 (b) 20 (c) 30 (d) no limit
Answer: (b) 20 vials

3. Which is contraindicated in viper coagulopathy? (a) ASV (b) FFP after ASV (c) Heparin (d) Packed cells
Answer: (c)

4. Low-dose ASV loading for confirmed saw-scaled viper (STG): (a) 2 (b) 6 (c) 10 (d) 20 vials
Answer: (b)

5. Adrenaline dose for ASV anaphylaxis in adults: (a) 0.05 mg IV (b) 0.5 mg IM (c) 1 mg SC (d) 5 mg IM
Answer: (b)

6. Krait bites typically occur: (a) In fields at noon (b) In water (c) At night in sleeping people (d) On trees
Answer: (c)

7. Dark brown urine, myalgia and paralysis after a bite on the beach suggest: (a) Cobra (b) Sea snake (c) Russell's viper (d) Pit viper
Answer: (b)

8. A positive neostigmine trial is improvement of ptosis by: (a) 10% (b) 25% (c) ≥ 50% (d) 100%
Answer: (c)

9. Drug to avoid in scorpion sting: (a) Prazosin (b) Dobutamine (c) Atropine (d) Paracetamol
Answer: (c)

10. ERIG dose: (a) 10 IU/kg (b) 20 IU/kg (c) 40 IU/kg (d) 80 IU/kg
Answer: (c)

11. Paederus dermatitis is caused by: (a) Cantharidin (b) Pederin (c) Melittin (d) Formic acid
Answer: (b)

12. First aid for a box jellyfish sting: (a) Fresh water (b) Vinegar (c) Rubbing sand (d) Urine
Answer: (b)

APPENDIX E · MORE MCQS

MCQs — Set 2

Answers below each question

13. Each mL of Indian polyvalent ASV neutralises how much common krait venom? (a) 0.25 mg (b) 0.45 mg (c) 0.6 mg (d) 1 mg
Answer: (b) 0.45 mg

14. Bilateral parotid swelling and chemosis after a snakebite suggest: (a) Krait (b) Cobra (c) Russell's viper (d) Sea snake
Answer: (c)

15. The STG criterion for fasciotomy includes intracompartmental pressure above: (a) 20 mmHg (b) 30 mmHg (c) 40 mmHg (d) 60 mmHg
Answer: (c)

16. The pre-synaptic toxin of the common krait is: (a) α-bungarotoxin (b) β-bungarotoxin (c) cardiotoxin (d) ecarin
Answer: (b)

17. Indian polyvalent ASV is NOT effective against: (a) Saw-scaled viper (b) Common krait (c) Hump-nosed pit viper (d) Spectacled cobra
Answer: (c)

18. Dialysis indication in snakebite AKI (STG) includes serum creatinine above: (a) 2 mg/dL (b) 3 mg/dL (c) 4 mg/dL (d) 6 mg/dL
Answer: (c)

19. First-line drug for late serum sickness after ASV: (a) Adrenaline (b) Chlorpheniramine (c) Furosemide (d) Atropine
Answer: (b), then prednisolone if no response

20. Rabies immunoglobulin can be given up to which day after the first vaccine dose? (a) Day 3 (b) Day 7 (c) Day 14 (d) Day 28
Answer: (b)

21. Rat bites in India require: (a) Category III PEP (b) Vaccine only (c) No rabies PEP (d) RIG only
Answer: (c)

22. Snakebite helpline number piloted under NAPSE: (a) 104 (b) 1075 (c) 15400 (d) 14567
Answer: (c)

23. Paired thin white bands and enlarged hexagonal vertebral scales identify: (a) Wolf snake (b) Common krait (c) Banded krait (d) Rat snake
Answer: (b)

24. A snake drug that came from Bothrops venom: (a) Eptifibatide (b) Captopril (c) Exenatide (d) Ziconotide
Answer: (b)

Scoring

20–24 correct — ready for the Emergency Medicine Department night shift · 14–19 — revise Chapters 9–13 · below 14 — read Part A again, slowly, with the flowcharts.

APPENDIX F · VISUAL ATLAS

See It Online — Tap or Scan

Free photographs of Indian snakes and look-alikes (Wikipedia/Wikimedia and the Indian Snakes portal)

Spectacled cobra

▶ Images

Naja naja — hood & spectacle mark

Monocled cobra

▶ Images

Naja kaouthia — single 'O' mark

King cobra

▶ Images

Ophiophagus hannah

Common krait

▶ Images

Bungarus caeruleus — paired white bands

Banded krait

▶ Images

Yellow & black bands, NE India

Sind krait

▶ Images

Bungarus sindanus — W India

Russell's viper

▶ Images

Daboia russelii — 3 chains of spots

Saw-scaled viper

▶ Images

Echis carinatus — 'bird-foot' mark

Hump-nosed pit viper

▶ Images

Hypnale hypnale — upturned snout

Malabar pit viper

▶ Images

Craspedocephalus malabaricus

Bamboo pit viper

▶ Images

Green pit viper, Western Ghats

Beaked sea snake

▶ Images

Hydrophis schistosus — paddle tail

Slender coral snake

▶ Images

Calliophis melanurus

Common cat snake

▶ Images

Boiga trigonata — mildly venomous

Red-necked keelback

▶ Images

Rhabdophis — rear-fanged

Indian rat snake

▶ Images

Ptyas mucosa — non-venomous

Common wolf snake

▶ Images

Lycodon aulicus — krait mimic

Rough-scaled sand boa

▶ Images

Russell's viper mimic

Checkered keelback

▶ Images

Water snake — non-venomous

Indian rock python

▶ Images

Python molurus

Indian Snakes portal

▶ Website

Species ID photos by state

Snake venom & fangs

▶ Article

Fang types, venom glands

Snakebite overview

▶ Article

Clinical overview

Compartment syndrome

▶ Article

5 Ps, fasciotomy

APPENDIX F · VISUAL ATLAS

Other Bites and Stings — Pictures

Scorpions, insects, spiders, marine creatures and others

Indian red scorpion

▶ Images

Hottentotta (Mesobuthus) tamulus

Scorpion sting

▶ Article

Clinical overview

Bee sting

▶ Article

Stinger, venom, allergy

Asian giant hornet

▶ Images

Vespa mandarinia

Fire ant

▶ Images

Solenopsis — pustules

Anaphylaxis

▶ Article

Recognition & treatment

Widow spiders

▶ Images

Latrodectus

Indian tarantula

▶ Images

Poecilotheria

Giant centipede

▶ Images

Scolopendra

Paederus dermatitis

▶ Images

Kissing lesions

Blister beetle

▶ Images

Cantharidin

Caterpillar dermatitis

▶ Article

Urticating hairs

Box jellyfish

▶ Images

Cubozoa

Portuguese man o' war

▶ Images

Physalia

Stingrays

▶ Article

Barb wounds, injuries

Stonefish

▶ Images

Synanceia

Striped eel catfish

▶ Images

Plotosus lineatus

Sea urchin

▶ Images

Spine injuries

Cone snails

▶ Images

Conidae — conotoxins

Tick paralysis

▶ Article

Ascending paralysis

Kyasanur Forest disease

▶ Article

Tick-borne, Western Ghats

Asian common toad

▶ Images

Duttaphrynus — bufotoxin

Leech

▶ Images

Land & aquatic leeches

Rat-bite fever

▶ Article

Streptobacillus, Spirillum

APPENDIX F · VIDEOS

AETCM Video Lectures

Tap or scan to watch on YouTube

These AETCM teaching videos complement the chapters on snakebite and anti-snake venom (videos 1–3), scorpion sting, bee sting and dog bite. Watch them before clinical postings and use them for group teaching.

AETCM video lecture 1

▶ Video

AETCM YouTube — watch

AETCM video lecture 2

▶ Video

AETCM YouTube — watch

AETCM video lecture 3

▶ Video

AETCM YouTube — watch

AETCM video — Scorpion sting

▶ Video

AETCM YouTube

AETCM video — Bee sting

▶ Video

AETCM YouTube

AETCM video — Dog bite

▶ Video

AETCM YouTube

APPENDIX G

Standard Guidelines and Further Reading

Official documents used in this book — tap or scan to open

MoHFW STG Snakebite 2016
▶ Guideline
Full Standard Treatment Guideline

STG Quick Reference 2016
▶ Guideline
Management of snake bite

NAPSE 2024
▶ Guideline
National Action Plan, NCDC

NCDC Snakebite Programme
▶ Website
National programme page

NHSRC — Management of Snake Bite
▶ Guideline
Clinician module

WHO SEARO Guidelines 2016
▶ Guideline
Management of snakebites, 2nd ed.

WHO fact sheet
▶ Article
Snakebite envenoming

WHO Snakebite Strategy 2019
▶ Guideline
Halve deaths by 2030

Million Death Study 2020
▶ Paper
eLife — 58,000 deaths/yr

Beyond the Big Four
▶ Paper
Neglected snakes & ASV gaps

Indian protocol highlights
▶ Paper
Indian Pediatrics 2007

Haffkine Snake Antivenin
▶ Product
Label, potency, packing

Rabies-free India (NRCP)
▶ Guideline
National rabies portal

National Rabies Guidelines
▶ Guideline
NCDC rabies prophylaxis

WHO fact sheet
▶ Article
Rabies

Dog bite
▶ Article
Wound care

References

1. Directorate General of Health Services, MoHFW, Government of India. *Standard Treatment Guidelines — Management of Snake Bite* (full guideline and Quick Reference Guide), 2016.
2. Centre for One Health, National Centre for Disease Control. *National Action Plan for Prevention and Control of Snakebite Envenoming (NAPSE)*, 2024.
3. Warrell DA, WHO Regional Office for South-East Asia. *Guidelines for the Management of Snakebites*, 2nd edition, 2016.
4. World Health Organization. *Snakebite envenoming: a strategy for prevention and control*, 2019.
5. Suraweera W, Warrell D, Whitaker R, et al. Trends in snakebite deaths in India from 2000 to 2019 in a nationally representative mortality study. *eLife* 2020;9:e54076.
6. Simpson ID. The new national snakebite management protocols (highlights). *Indian Pediatrics* 2007;44:173–176.
7. Senji Laxme RR, et al. Beyond the "big four": venom profiling of the medically important yet neglected Indian snakes reveals disturbing antivenom deficiencies. *PLoS Negl Trop Dis* 2019.
8. National Centre for Disease Control. *National Guidelines on Rabies Prophylaxis* (National Rabies Control Programme) and expert group review 2019; WHO Rabies Position Paper 2018.

9. Bawaskar HS, Bawaskar PH. Efficacy and safety of scorpion antivenom plus prazosin compared with prazosin alone for venomous scorpion sting: randomised open label clinical trial. *BMJ* 2011;342:c7136.
10. Indian Academy of Pediatrics. *Standard Treatment Guidelines — Scorpion sting; Snake envenomation*, 2022–23.
11. Haffkine Bio-Pharmaceutical Corporation. Snake Antivenin I.P. and Scorpion Venom Antiserum — product information.

Web links were checked at the time of writing (November 2026); official sites sometimes move documents — search the title on the NCDC or MoHFW website if a link fails.

APPENDIX G · EVIDENCE

Landmark Articles Since 2020

Recent high-impact research on snakebite, antivenoms, scorpion stings and rabies — tap a title to open

Journal	Article	Take-home message
eLife 2020;9:e54076	**Trends in snakebite deaths in India from 2000 to 2019 in a nationally representative mortality study.** Suraweera W, Warrell D, Whitaker R, et al.	India had about 1.2 million snakebite deaths in 2000–2019 (≈ 58,000 per year) — far more than official figures. Nearly half occurred in the monsoon, most at home in rural areas, and about 70% in eight high-burden States; the lifetime risk of dying of snakebite before 70 is about 1 in 250.
Trans R Soc Trop Med Hyg 2024	**National Action Plan for the prevention and control of snakebite envenoming in India: strategies and challenges.** Munshi H, Gajbhiye R.	NAPSE (2024) adopts a One Health approach to halve snakebite deaths and disability by 2030 — sustained ASV availability, training of doctors, surveillance and community education are the pillars; implementation at district level is the challenge.
J Forensic Leg Med 2020;73:101996	**Comparison of two anti-snake venom protocols in hemotoxic snake bite: a randomized trial.** Sagar BKP, Bammigatti C, Kadhiravan T, et al.	In 140 haemotoxic snakebite patients (JIPMER), the national protocol (100 mL then 100 mL 6-hourly) did not reduce mortality or morbidity compared with a lower-dose protocol (70 mL then 30 mL 6-hourly); about 12% had persistent coagulopathy until death — more ASV is not always better.
Sci Transl Med 2024;16:eadk1867	**Synthetic development of a broadly neutralizing antibody against snake venom long-chain α-neurotoxins.** Khalek IS, Senji Laxme RR, … Sunagar K, Jardine JG.	An Indian (IISc)–US team produced a fully human synthetic antibody that neutralises long-chain α-neurotoxins of many elapids and protected mice from lethal venom — a step towards monoclonal, reaction-free antivenoms.
BMJ Glob Health 2024;9:e015985	**Oral varespladib for the treatment of snakebite envenoming in India and the USA (BRAVO): a phase II randomised clinical trial.** Gerardo CJ, Carter RW, Kumar S, et al.	The first trial of a direct toxin inhibitor in snakebite (95 patients, India and USA): oral varespladib added to antivenom was safe; the primary end-point was not met, but there was a signal of benefit when treatment began within 5 hours of the bite.
Cell 2025;188:3117–3134	**Snake venom protection by a cocktail of varespladib and broadly neutralizing human antibodies.** Glanville J, Bellin M, Pletnev S, et al.	Two broadly neutralising antibodies from a hyper-immune human donor, combined with the PLA_2 inhibitor varespladib, protected mice against venoms of 19 WHO category 1–2 elapids (including cobras, king cobra, common and banded kraits) — proof of principle for a 'universal' elapid antivenom.
Clin Infect Dis 2021	**A phase 3, randomized, open-label, non-inferiority trial evaluating anti-rabies monoclonal antibody cocktail (Twinrab) against human rabies immunoglobulin.** Kansagra K, Parmar D, Mendiratta SK, et al.	In 308 Indian patients with WHO category III exposures, the monoclonal antibody cocktail (docaravimab + miromavimab, 40 IU/kg) was non-inferior to HRIG with the Essen vaccine schedule — supporting rabies monoclonal antibodies as an affordable alternative to RIG.
J Clin Diagn Res 2025;19(1)	**Efficacy and safety of high-dose versus standard-dose prazosin in paediatric scorpion envenomation: a single-blinded randomised controlled study.** Abbadi AG, Bijapure HR, Doddihal CR, et al.	A recent Indian paediatric RCT (86 children, grade II–III stings) compared prazosin 30 µg/kg 3-hourly with higher loading doses (60–90 µg/kg); the authors found high-dose prazosin safe — but this is a small single-centre trial, and 30 µg/kg/dose remains the standard.

PLoS Negl Trop Dis 2026;20(9):e0014736	**Adjunctive varespladib after antivenom administration for snakebite: a phase II randomized clinical trial (BRAVIO).** Gerardo CJ, Bhalla A, Mohanty CR, et al.	IV-then-oral varespladib given late (mean 7 h after the bite, after ASV) in 140 patients in India and the USA did not meet its primary end-points; signals in krait bites are only hypothesis-generating. ASV and ventilation remain the standard of care.

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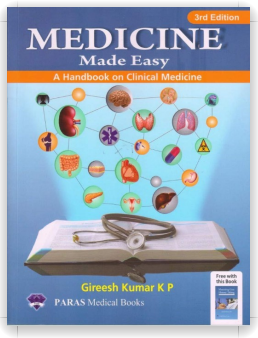
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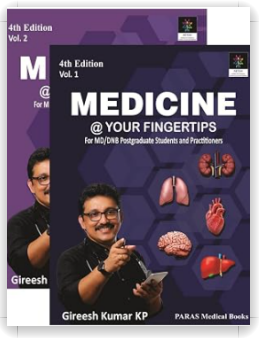
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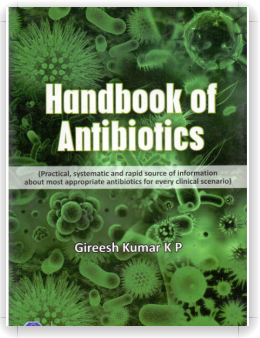
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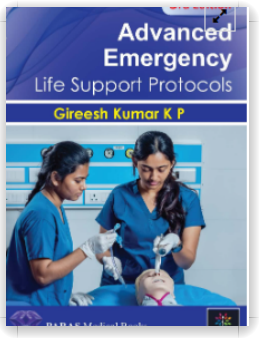
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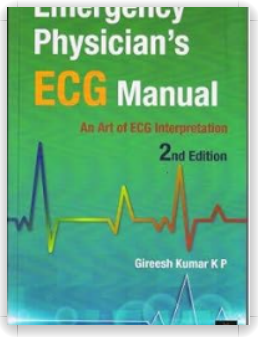
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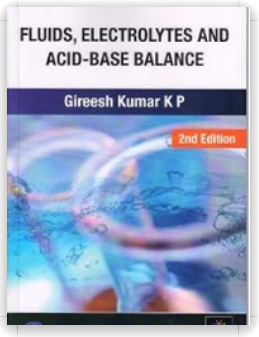
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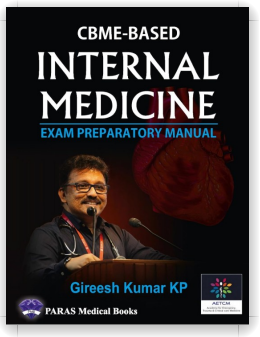
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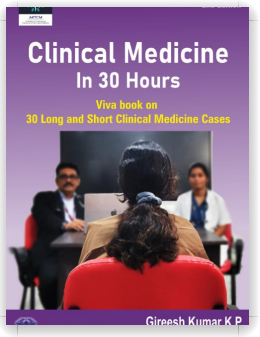
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