FINAL MBBS · GENERAL MEDICINE · LONG CASE

A E T C M

Hemiplegia Case Sheet

A Complete Neurology Case — from History to Viva

A fully worked case sheet of right hemiplegia with Broca's aphasia, written the way a final-year MBBS student should record and present it in the university examination — with the reasoning behind every question asked and every sign elicited.

History of presenting illness › | Relevant negatives & risk factors › | General examination ›

Higher mental functions & aphasia › | Cranial nerves › | Motor system — tone, power, reflexes ›

Sensory & cerebellar › | Localisation of the lesion › | Differential diagnosis › | Investigations ›

Thrombolysis & secondary prevention › | Rehabilitation › | Model presentation › | Viva questions ›

TAP A TOPIC TO JUMP TO IT

HISTORY onset · progression · risk › **EXAMINATION** UMN signs · speech › **LOCALISATION** site · territory › **DIAGNOSIS** & management

By

Dr. Gireesh Kumar K.P.

For final-year MBBS students, interns and teachers of clinical medicine

Hemiplegia Case Sheet

A complete neurology long case for final MBBS preparation and presentation

Dr. Gireesh Kumar K.P.

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How to use this case sheet

Hemiplegia is the commonest neurology long case in the final MBBS General Medicine examination, and the case in which examiners most clearly see whether a student can **think** — from a symptom, to a sign, to the site of the lesion, to the cause. This booklet presents one complete, fully worked case exactly as it should be written in the case record and presented at the bedside.

Each section has three layers: the **case record** itself (what you write), **green boxes** that summarise and interpret (what you conclude), and **blue boxes** with examiner's tips (what you are judged on). Tables of localisation, aphasia, stroke syndromes and differential diagnosis turn the single case into a revision of the whole topic. The book ends with a **model presentation** to practise aloud and **35 viva questions** with short answers.

The patient described is a composite teaching case; details have been chosen to illustrate the commonest examination scenario. Examine your own patient carefully and record only what you find.

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SECTION A · HISTORY

Case Sheet — Hemiplegia (Long Case, Neurology)

Final MBBS General Medicine · Case-taking and presentation format

1. Particulars of the patient

Name	Mr. K. R. (initials only — always respect confidentiality)
Age / Sex	62 years / Male
Handedness	**Right-handed** (always ask — it tells you the dominant hemisphere for language)
Occupation	Retired bus conductor (31 years); now runs a small provision shop
Address	Aluva, Ernakulam district, Kerala
Religion / Education	Hindu / Studied up to 10th standard
Informant	Patient (partly, by gestures and short phrases) and his wife — **reliable**
Date of admission / examination	Day 42 after the onset of illness

2. Chief complaints (in chronological order, in the patient's own words)

- Weakness of the **right upper and lower limbs** — 6 weeks
- **Difficulty in speaking** — 6 weeks
- **Deviation of the angle of the mouth** to the left side — 6 weeks
- Stiffness of the right arm and leg — 4 weeks

> **Examiner's tip**
> Write the chief complaints as symptoms with duration — never as a diagnosis. ‘Right hemiplegia × 6 weeks’ is wrong; ‘weakness of the right upper and lower limbs × 6 weeks’ is right.

3. History of presenting illness

The patient was apparently normal 6 weeks ago. He went to bed at about 10 p.m. after his usual dinner. On waking up at about 6 a.m. the next morning, he found that he was **unable to move his right arm and leg** and could not get out of bed. The weakness was **sudden in onset** and was **maximal at the onset**; it did not progress afterwards (a ‘completed stroke’). He was last seen normal at 10 p.m. — this is therefore a **wake-up stroke**.

The weakness was **more in the right upper limb than in the lower limb**. He could not lift his right arm above the bed, hold a tumbler, button his shirt or write. He was unable to stand or walk without support from two persons. Both proximal and distal muscles were affected — he could not lift his arm (proximal) and could not grip or pick up objects (distal).

At the same time, his wife noticed that he **could not speak properly**. He appeared to understand everything that was said to him and obeyed commands, but he could produce only a few single words, with great effort and frustration — ‘*he knew what he wanted to say but could not bring the words out*’. There was no slurring of speech of the type seen with a drunk person. He could not write even with his left hand.

The **angle of his mouth deviated to the left** and saliva dribbled from the right angle of the mouth. He could close both eyes fully and wrinkle his forehead. During meals, food collected between the right cheek and gums. For the first 3 days he coughed while drinking water; this resolved spontaneously and he now swallows solids and liquids normally.

He was taken to a nearby hospital and reached there at about 9 a.m. (11 hours after he was last seen normal). A CT scan of the brain was done; he was told that there was ‘*no bleeding in the brain*’, and he was treated with tablets (aspirin, atorvastatin) and physiotherapy for 7 days. He was **not** given a ‘clot-dissolving injection’ — the family was told he had come too late.

Course since onset: there has been **gradual improvement**. He started moving the right leg after 1 week and now walks short distances with one-person support, dragging the right leg in a semicircle. The right upper limb has improved less — he can lift it partly but cannot use the hand for fine work. Speech has improved; he can now say short sentences of 2–3 words. About 2 weeks after the onset, the right arm and leg became **stiff**; the arm tends to stay bent at the elbow and the fingers curled.

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History of presenting illness — relevant positives and negatives

In every case of hemiplegia, the history must answer four questions: **(1) Is it a stroke?** (sudden onset) **(2) Is it ischaemic or haemorrhagic? (3) Which vascular territory? (4) What is the cause / risk factor?** Ask the following specifically and record positives and negatives.

Question asked	Answer in this patient	Why it is asked
Any previous similar episode of transient weakness, numbness or speech loss?	**Yes** — 2 months ago, sudden weakness of the right hand with difficulty in speaking lasting **about 15 minutes**, recovered completely. Not evaluated.	**Transient ischaemic attack (TIA)** — warning sign of carotid atherosclerosis
Transient loss of vision in one eye ('curtain coming down')?	No	Amaurosis fugax — ipsilateral carotid disease
Loss of consciousness at onset?	No	Large haemorrhage, brainstem stroke, massive infarct
Headache, vomiting at onset?	No	Intracerebral / subarachnoid haemorrhage; raised ICP
Seizures / abnormal movements?	No	Todd's palsy, cortical venous thrombosis, embolic cortical infarct, tumour
Fever, ear discharge, neck stiffness?	No	Brain abscess, meningitis with vasculitis, tuberculoma
Head injury in recent weeks or months?	No (no falls)	Chronic subdural haematoma — especially in elderly and alcoholics
Gradual progression over weeks? Early-morning headache?	No — maximal at onset	Space-occupying lesion (tumour, abscess)
Double vision, vertigo, unsteadiness, facial numbness, difficulty swallowing?	Transient coughing on liquids for 3 days only	Brainstem (posterior circulation) involvement
Visual field loss — bumping into objects on one side?	No	Homonymous hemianopia — PCA or large MCA infarct
Numbness, tingling, loss of sensation on the weak side?	No	Sensory cortex / thalamic involvement
Palpitations, chest pain, breathlessness, recent MI?	No	Cardioembolism — AF, recent MI with LV thrombus
Neck pain or neck manipulation?	No	Carotid / vertebral artery dissection (young stroke)
Bladder and bowel disturbance?	Urgency for first 3 days; now normal	Bilateral or frontal lesions; spinal cord lesions
Mood changes, crying spells, sleep disturbance?	Occasional crying spells, feels low	Post-stroke depression, emotional lability
Pain in the right shoulder?	**Yes** — on lifting the arm, since 3 weeks	Hemiplegic shoulder pain / subluxation

4. Past history

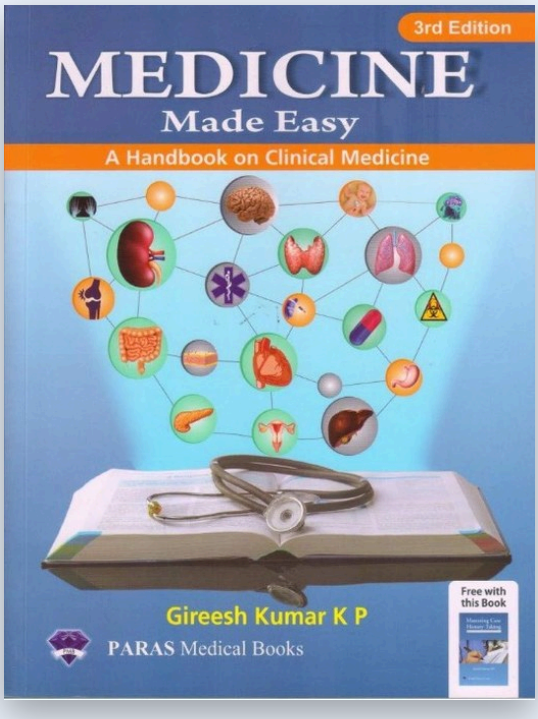
- **Hypertension** — diagnosed 8 years ago; on amlodipine 5 mg OD, **irregular** intake; BP not monitored.
- **Type 2 diabetes mellitus** — 5 years; on metformin 500 mg BD, irregular; no follow-up HbA1c.
- No history of coronary artery disease, rheumatic fever, heart disease, previous stroke, seizures, tuberculosis, syphilis, HIV, bleeding disorder or migraine.
- No history of previous surgery or blood transfusion.

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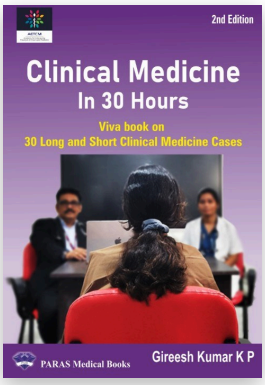
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5. Personal history

Diet	Mixed; high-salt (pickles, dried fish), coconut-oil cooking
Appetite / sleep	Normal / disturbed for 6 weeks
Bladder / bowel	Normal at present
Smoking	**20 cigarettes/day for 30 years = 30 pack-years**; stopped after the stroke
Alcohol	Occasional — about 90 mL of spirit weekly for 20 years
Physical activity	Sedentary for the last 5 years

6. Family history

Father died of a stroke at 65 years. Mother had diabetes. Younger brother had a myocardial infarction at 55 years. Wife and son are healthy. → **Positive family history of premature atherosclerotic vascular disease.**

7. Drug, treatment and socio-economic history

- **Current drugs:** aspirin 75 mg OD, atorvastatin 40 mg HS, telmisartan 40 mg + amlodipine 5 mg OD, metformin 500 mg BD, pantoprazole 40 mg OD. No known drug allergy. No oral anticoagulant.
- **Socio-economic status:** lower-middle class (modified Kuppuswamy scale); lives with wife and son in a single-storey house; covered under a state health-insurance scheme. Toilet is Indian-style — relevant for rehabilitation.

Stroke risk factors in this patient

Non-modifiable	Modifiable — present	Modifiable — to be checked
Age >55 years · male sex · family history of premature stroke and MI	Hypertension (most important) · diabetes · smoking · sedentary lifestyle · high-salt diet · previous TIA	Dyslipidaemia · atrial fibrillation · carotid stenosis · obesity · obstructive sleep apnoea · hyperhomocysteinaemia · alcohol excess

Mode of onset — the single most useful clue to the cause

Onset and course	Likely pathology
Sudden, maximal at onset, often during activity, with palpitations	**Embolism** — cardiac (AF, MS, IE, recent MI) or artery-to-artery (carotid plaque)
Sudden, noticed on waking or stepwise progression over hours, often preceded by TIAs	**Thrombosis** — large-artery atherosclerosis or lacunar (small-vessel) infarct
Sudden during exertion with severe headache, vomiting, early drowsiness	**Intracerebral haemorrhage** (hypertensive) or subarachnoid haemorrhage
Transient, complete recovery within minutes to hours	TIA; Todd's palsy (after seizure); hemiplegic migraine; hypoglycaemia
Subacute — days, with fever, headache or seizures	Brain abscess, tuberculoma, encephalitis, cerebral venous thrombosis, demyelination (ADEM, MS)
Gradual — weeks to months, with headache, vomiting, seizures, personality change	Space-occupying lesion — glioma, metastasis, meningioma; **chronic subdural haematoma** (elderly, alcohol, minor fall)

> **Examiner's tip — 'last seen normal'**
> For a wake-up stroke the time of onset is taken as the time the patient was **last seen normal** (here, 10 p.m.), not the time he woke up. This decides eligibility for thrombolysis (≤4.5 h) and thrombectomy (≤24 h).

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8. Summary of history

Summary (say this before you start the examination)

A **62-year-old right-handed man**, a known **hypertensive** (8 years) and **diabetic** (5 years) on irregular treatment, a **chronic smoker** (30 pack-years) with a family history of premature vascular disease, presented with **sudden-onset weakness of the right upper and lower limbs (upper > lower), noticed on waking up**, associated with **non-fluent speech with preserved comprehension** and **deviation of the angle of the mouth to the left**, 6 weeks ago. There was a **transient episode of right-hand weakness with speech difficulty 2 months earlier**. There was no loss of consciousness, headache, vomiting, seizures, fever, trauma, visual or sensory symptoms, or palpitations. The weakness was maximal at onset and has been **gradually improving**; spasticity developed after 2 weeks.

9. Provisional diagnosis from history

Right hemiparesis with right facial weakness and motor (Broca's) aphasia, of sudden onset — probably due to an ischaemic stroke in the territory of the left middle cerebral artery, in a patient with hypertension, diabetes and smoking, preceded by a TIA.

How the history leads to the diagnosis — the reasoning

Clue from history	Inference
Sudden onset, maximal at onset, improving afterwards	**Vascular** lesion (stroke). Tumours and abscesses progress gradually; demyelination evolves over days.
Noticed on waking, no headache/vomiting/LOC	**Ischaemic** rather than haemorrhagic (haemorrhage often occurs during activity with headache, vomiting, altered sensorium) — but only **CT** can confirm
Right-sided weakness + language disturbance in a right-handed person	Lesion in the **left (dominant) cerebral hemisphere — cortical involvement** (aphasia is a cortical sign)
Upper limb and face more than lower limb	**Lateral surface of the hemisphere — MCA territory** (leg area lies medially, in the ACA territory)
Non-fluent speech, comprehension preserved	**Broca's area** — inferior frontal gyrus → **superior division of the MCA**
No sensory loss, no field defect	Parietal and temporal lobes and optic radiation spared — consistent with superior division of MCA
Previous TIA in the same territory, smoker, HTN, DM	**Large-artery atherosclerosis** — suspect stenosis of the **left internal carotid artery**
No palpitations, no valvular heart disease	Cardioembolism less likely (still needs ECG, Holter, echo)
Stiffness after 2 weeks	Natural course of an **upper motor neuron** lesion — flaccid (cerebral shock) phase followed by spastic phase

Examiner's tip — what the examiner looks for in the history

(1) The exact **mode of onset** and time last seen normal (decides thrombolysis eligibility). (2) **Handedness**. (3) Whether you asked about **TIA, AF, headache/vomiting**. (4) Risk factors with **duration, drugs and compliance**. (5) **Functional status** — walking, self-care, swallowing, bladder.

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SECTION B · EXAMINATION

General Physical Examination

Look for the risk factors, the source of embolism and the complications of immobility

Consciousness	Conscious; responds appropriately by gestures and short phrases; cooperative
Orientation	Oriented to time, place and person (tested with yes/no questions and pointing)
Built / nourishment	Moderately built and well nourished
Height / weight / BMI	168 cm / 76 kg / **BMI 26.9 kg/m²** (overweight — Asian cut-off ≥23)
Waist circumference	98 cm (central obesity — >90 cm in Indian men)
Decubitus	Lying supine; right upper limb flexed at elbow, wrist and fingers; right lower limb extended (**Wernicke–Mann posture**)

Sign	Finding	Relevance
Pallor	Absent	Anaemia — aggravates ischaemia
Icterus	Absent	—
Cyanosis	Absent	Cyanotic heart disease → paradoxical embolism
Clubbing	Absent	Infective endocarditis, cyanotic heart disease, lung cancer (metastasis)
Lymphadenopathy	Absent	TB, lymphoma, malignancy with brain secondaries
Pedal oedema	Absent; mild **oedema of the dorsum of the right hand**	Dependent oedema of a paralysed limb; shoulder–hand syndrome
Xanthelasma / arcus	**Arcus** present bilaterally; no xanthelasma or xanthomas	Dyslipidaemia
Skin	No neurocutaneous markers, no purpura, no livedo	Neurofibromatosis, vasculitis, APLA syndrome
Pressure areas	**No bed sores** over sacrum, heels, trochanter	Complication of immobility — always examine
Calf	Non-tender, no swelling	Deep vein thrombosis in the paralysed leg
Right shoulder	Painful on abduction; **one finger-breadth gap** between acromion and humeral head	Inferior subluxation of the hemiplegic shoulder

Vital signs

Vital	Finding	Interpretation
Pulse	84/min, **regular**, normal volume and character; no radio-radial or radio-femoral delay; vessel wall not thickened; all peripheral pulses felt	Regular rhythm — AF not clinically evident (paroxysmal AF still possible)
Blood pressure	Right arm 158/94 mmHg, left arm 154/92 mmHg (supine); standing 152/90 mmHg	**Uncontrolled hypertension**; no significant inter-arm difference (>10 mmHg suggests subclavian stenosis / dissection); no postural drop
Respiratory rate	16/min, thoraco-abdominal	Normal; no Cheyne–Stokes breathing
Temperature	36.8 °C (afebrile)	Fever → aspiration pneumonia, UTI, DVT
SpO_2	97 % on room air	—
Capillary blood glucose	182 mg/dL (random)	Hyperglycaemia; always exclude hypoglycaemia in acute stroke

Neck and fundus

- **Carotid arteries:** both pulsations felt; **a bruit is heard over the left carotid** at the angle of the jaw (bell of the stethoscope, breath held). → suggests **left ICA stenosis**, ipsilateral to the infarct (symptomatic carotid stenosis). JVP not raised. Thyroid not enlarged.
- **Fundus:** arteriolar narrowing with **arteriovenous nicking** — hypertensive retinopathy grade II (Keith–Wagener–Barker); no haemorrhages or exudates; no papilloedema; no cholesterol emboli (Hollenhorst plaques); mild background diabetic changes absent.

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SECTION B · CENTRAL NERVOUS SYSTEM

Higher Mental Functions

Examine the CNS in this order: HMF → cranial nerves → motor → sensory → cerebellum → skull & spine → meninges → autonomic

Handedness	Right-handed (writes, eats and throws with the right hand) → left hemisphere dominant
Level of consciousness	Fully conscious, alert. GCS — E4 $V_{aphasic}$ M6 (record verbal score as 'aphasic' rather than a low number)
Appearance and behaviour	Well groomed with help; cooperative; frustrated when unable to express himself; occasional inappropriate crying (emotional lability)
Orientation	Oriented to time, place and person (tested non-verbally)
Memory	Immediate, recent and remote memory intact (tested by recognition and pointing)
Intelligence / calculation	Appropriate for education; simple calculations by pointing to numbers — correct
Mood	Low mood — screen for post-stroke depression
Praxis	No ideomotor apraxia with the left hand (can show how to comb, salute)
Neglect	No visual or sensory neglect (neglect is mainly a right-hemisphere sign)

Speech and language — test the six components

Component	How to test	Finding in this patient
1. Fluency	Listen to spontaneous speech — ask about his job, family	**Non-fluent**: slow, effortful, 2–3-word phrases, 'telegraphic', with omission of small words (agrammatism); prosody poor
2. Comprehension	Simple → complex commands: 'close your eyes'; 'touch your nose and then your left ear'; yes/no questions	**Preserved** for simple and two-step commands; mild difficulty with complex grammar
3. Repetition	'No ifs, ands or buts'; 'The boy went to the market'	**Impaired**
4. Naming	Name pen, watch, button, parts of a watch	**Impaired** (anomia) — improved with phonemic cues
5. Reading	Read aloud and obey a written command — 'CLOSE YOUR EYES'	Comprehension of written command preserved; reading aloud impaired
6. Writing	Write a sentence with the non-paralysed (left) hand	**Impaired** (agraphia) — not explained by left-hand clumsiness alone
Articulation	'Pa-pa-pa', 'ta-ta-ta', 'ka-ka-ka'; 'West Register Street'	Mild **dysarthria** (UMN facial and lingual weakness)

Conclusion — speech

Non-fluent aphasia with relatively preserved comprehension and impaired repetition = Broca's (expressive / motor) aphasia, with mild UMN dysarthria. Localisation: posterior part of the left inferior frontal gyrus (Brodmann areas 44, 45) — **superior division of the left MCA**.

Classification of aphasia (learn this table)

Type	Fluency	Comprehension	Repetition	Site
Broca's (motor)	Non-fluent	Intact	Impaired	Inferior frontal gyrus (dominant)
Wernicke's (sensory)	Fluent (jargon, paraphasia)	Impaired	Impaired	Posterior superior temporal gyrus
Conduction	Fluent	Intact	**Severely impaired**	Arcuate fasciculus / supramarginal gyrus
Global	Non-fluent	Impaired	Impaired	Entire perisylvian area (MCA stem occlusion)
Transcortical motor	Non-fluent	Intact	**Intact**	ACA–MCA watershed (frontal)
Transcortical sensory	Fluent	Impaired	**Intact**	MCA–PCA watershed (parieto-temporal)
Anomic	Fluent	Intact	Intact	Angular gyrus / any part; recovery stage

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Cranial nerves

Nerve	Test	Right	Left
I — Olfactory	Smell of coffee, soap — each nostril	Normal	Normal
II — Optic	Visual acuity (bedside chart); colour vision; confrontation fields; pupillary reflexes; fundus	6/9, fields full, fundus — HTN retinopathy grade II	6/9, fields full, same
III, IV, VI	Ptosis; pupils (size, shape, direct and consensual light reflex, accommodation); extra-ocular movements; nystagmus; conjugate gaze	Pupils 3 mm, round, reacting; full EOM; no nystagmus; **no gaze deviation**	Same
V — Trigeminal	Sensation over V1, V2, V3; corneal reflex; masseter/temporalis; jaw jerk	Sensation normal; corneal reflex present	Normal; jaw jerk not exaggerated
VII — Facial	Wrinkle forehead; close eyes tightly; show teeth; blow cheeks; nasolabial fold; taste (anterior 2/3)	**Flattened nasolabial fold, angle of mouth drawn to the left on showing teeth; forehead wrinkling and eye closure normal**	Normal
VIII — Vestibulocochlear	Finger rub; Rinne and Weber tests	Normal hearing; Rinne positive	Same; Weber central
IX, X	Palatal movement on saying 'aah'; uvula; gag reflex; voice; swallowing (water swallow test)	Palate moves symmetrically, uvula central, gag present	Same
XI — Accessory	Shrug shoulders against resistance; turn head against resistance	Trapezius 4/5 (mild UMN weakness)	Normal; SCM normal
XII — Hypoglossal	Tongue at rest (wasting, fasciculations); protrusion; side-to-side movements	**Tongue deviates to the right** on protrusion; no wasting or fasciculation	—

Conclusion — cranial nerves

Right upper motor neuron (supranuclear) facial palsy and **right UMN hypoglossal weakness**; all other cranial nerves normal. Cranial nerve involvement on the **same side** as the limb weakness → lesion is **above the brainstem** (cerebral hemisphere / internal capsule).

Upper motor neuron vs lower motor neuron facial palsy

Feature	UMN (supranuclear) — this patient	LMN (nuclear / infranuclear, e.g. Bell's palsy)
Part of face affected	Lower half only; upper face (forehead) spared	Whole half of face, including forehead
Why	Upper face has **bilateral** cortical representation	Final common pathway to all facial muscles damaged
Eye closure / Bell's phenomenon	Normal / not seen	Incomplete closure; Bell's phenomenon visible
Emotional movements (smiling)	Often preserved (emotional–voluntary dissociation)	Lost
Associated features	Hemiplegia, aphasia on the same side (cortical/capsular lesion)	Hyperacusis, loss of taste, ear vesicles (Ramsay Hunt), crossed hemiplegia if pontine
Side of lesion	Contralateral cerebral hemisphere	Ipsilateral

Common viva question

Why does the tongue deviate to the paralysed side in UMN lesions? The genioglossus of each side pushes the tongue forward and to the opposite side. The left cortical lesion weakens the right genioglossus, so the normal left genioglossus pushes the tongue towards the **right (weak) side**. In LMN lesions, the tongue also deviates to the side of the lesion, but with wasting and fasciculations.

Gaze deviation — a useful localising sign

In an acute **hemispheric** stroke, the eyes deviate **towards the side of the lesion** (away from the hemiplegia) because the frontal eye field of the opposite side is unopposed — 'the patient looks at the lesion'. In a **pontine** lesion, the eyes look **away** from the lesion (towards the hemiplegia). Gaze deviation usually recovers within days, as in this patient.

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Motor system

a) Nutrition / bulk

Site of measurement	Right	Left
Arm — 10 cm above the olecranon	26 cm	27 cm
Forearm — 10 cm below the olecranon	23 cm	24 cm
Thigh — 18 cm above the patella	44 cm	45 cm
Calf — 10 cm below the tibial tuberosity	32 cm	33 cm

Inspection: no wasting, no fasciculations, no involuntary movements. Minimal (1 cm) reduction on the right is due to **disuse**. Significant wasting in a hemiplegic limb should make you think of an LMN lesion, a parietal lesion (parietal wasting) or a long-standing lesion.

b) Tone

Limb	Right	Left
Upper limb	**Increased — spasticity**, more in **flexors and pronators**; **clasp-knife** type (resistance at the beginning, then sudden give); spastic catch on rapid supination	Normal
Lower limb	**Increased — spasticity**, more in **extensors and adductors**; clasp-knife type	Normal

Grade spasticity with the **Modified Ashworth Scale**: right elbow flexors grade 2, right knee extensors grade 1+.

c) Power (MRC scale 0–5)

Joint	Movement	Right	Left
Shoulder	Abduction · adduction · flexion · extension	2 · 3 · 2 · 3	5
Elbow	Flexion · extension	3 · **2**	5
Wrist	Flexion · extension	3 · **2**	5
Hand	Grip · finger extension · finger abduction	2 · 1 · 1	5 (grip 100 %)
Hip	Flexion · extension · abduction · adduction	**3** · 4 · 3 · 4	5
Knee	Flexion · extension	**3** · 4	5
Ankle	Dorsiflexion · plantar flexion · eversion	**2** · 4 · 2	5
Trunk	Sit up from lying without hand support	Able with difficulty	—

Interpretation — the pyramidal pattern of weakness

In UMN weakness the **extensors of the upper limb** (shoulder abductors, elbow, wrist and finger extensors) and the **flexors of the lower limb** (hip flexors, knee flexors, ankle dorsiflexors) are weaker than their antagonists. This produces the typical posture — arm flexed and leg extended — and the hemiplegic gait. Distal muscles and fine finger movements are affected most and recover last. Here the **upper limb is weaker than the lower limb** → MCA (lateral cortical) territory.

MRC grade	Definition
0	No contraction
1	Flicker or trace of contraction
2	Active movement with gravity eliminated
3	Active movement against gravity
4	Active movement against gravity and some resistance (4−, 4, 4+)
5	Normal power

d) Bedside tests for subtle weakness

- **Pronator drift:** right arm pronates and drifts downward with eyes closed — positive.
- **Finger-tapping / rapid movements:** slow and clumsy on the right.
- **Barré's leg sign / Mingazzini:** right leg drifts down in the prone/supine test.
- **Hoover's sign:** genuine (useful to exclude functional weakness).

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e) Reflexes

Reflex	Root value	Right	Left
Superficial			
Corneal	V (aff) / VII (eff)	Present	Present
Abdominal — upper, middle, lower	T7–T9, T9–T11, T11–T12	**Absent**	Present
Cremasteric	L1, L2	**Absent**	Present
Plantar	S1	**Extensor (Babinski sign)**	Flexor
Deep tendon (0 – 4+)			
Jaw jerk	V (pons)	Not exaggerated	—
Biceps	C5, C6	**3+**	2+
Supinator (brachioradialis)	C5, C6	**3+**	2+
Triceps	C6, C7	**3+**	2+
Finger flexion / Hoffmann's sign	C8	**Hoffmann's positive**	Negative
Knee	L3, L4	**3+ with spread to adductors**	2+
Ankle	S1, S2	**3+**	2+
Clonus	—	**Ankle clonus — 5 beats (unsustained)**; patellar clonus absent	Absent

Babinski response: stroking the lateral border of the sole from heel to little toe and across the ball of the foot → **dorsiflexion of the great toe with fanning of the other toes**. Confirmatory methods: Chaddock (lateral malleolus), Oppenheim (shin), Gordon (calf squeeze).

> **Conclusion — reflexes**
> Right-sided **hyper-reflexia, clonus, extensor plantar and loss of superficial abdominal and cremasteric reflexes** = **upper motor neuron (pyramidal tract) lesion** on the right side of the body.

f) Coordination

Test	Right	Left
Finger–nose, finger–finger–nose	Cannot be tested reliably (power <3)	Normal
Dysdiadochokinesia	Cannot be tested	Normal
Heel–knee–shin	Slow, but no ataxia out of proportion to weakness	Normal
Rebound phenomenon	—	Absent

Ataxia out of proportion to weakness on the hemiparetic side suggests **ataxic hemiparesis** — a lacunar syndrome (internal capsule / pons).

g) Gait and stance

Walks with one-person support. **Hemiplegic (circumducting) gait**: the right arm is held flexed and adducted and does not swing; the right leg is stiff and extended at the knee, the foot plantar-flexed and inverted; the leg is swung outward in a semicircle (circumduction) with scraping of the toes and the outer border of the sole. Romberg's test cannot be done (cannot stand unsupported).

h) Involuntary movements

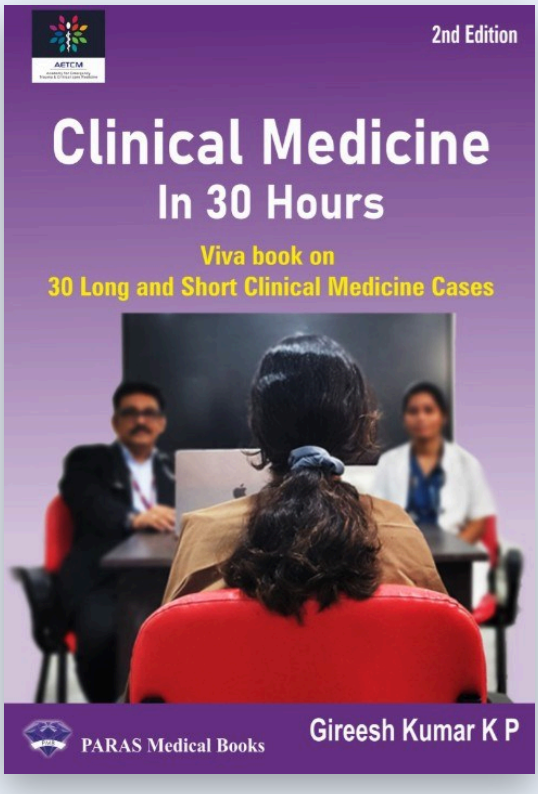
None. Note: **associated reactions** — the right arm flexes involuntarily when he yawns or makes an effort with the left hand; this is common in spastic hemiplegia.

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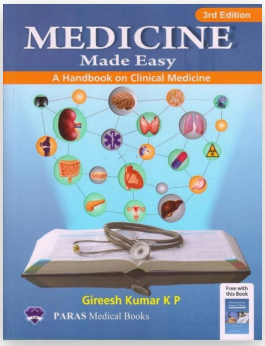
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Sensory system

Modality	Pathway	Right	Left
Touch (cotton wisp)	Anterior spinothalamic / posterior column	Normal	Normal
Pain (pin-prick)	Lateral spinothalamic	Normal	Normal
Temperature (hot/cold tubes)	Lateral spinothalamic	Normal	Normal
Vibration (128 Hz fork)	Posterior column	Normal	Normal
Joint position sense	Posterior column	Normal	Normal
Cortical sensations — two-point discrimination, stereognosis, graphaesthesia, tactile localisation, sensory extinction (inattention)	Parietal cortex	Intact (tested on the right where power permitted)	Intact

Test sensation in the dermatomes and compare both sides, from distal to proximal, with the patient's eyes closed. Cortical sensations can be tested only when primary sensations are intact. **Absence of sensory loss** spares the parietal lobe and thalamus, and argues against a large MCA-stem or thalamic lesion.

Cerebellum, skull, spine, meninges and autonomic system

Cerebellar signs	No nystagmus, no scanning speech, no intention tremor, no hypotonia, no pendular knee jerk on the left
Skull	Normal shape; no scars, tenderness or bruit
Spine	No deformity, tenderness or gibbus; straight-leg raising normal
Signs of meningeal irritation	No neck stiffness; Kernig's and Brudzinski's signs negative
Peripheral nerves	Not thickened (ulnar, common peroneal, greater auricular)
Autonomic system	Bladder and bowel control normal; no postural hypotension; no abnormal sweating; skin over the right limbs normal; no trophic ulcers
Primitive (frontal release) reflexes	Glabellar tap, snout, palmomental and grasp reflexes — absent

Examination of other systems

System	Findings	Relevance in stroke
Cardiovascular	Apex in the left 5th intercostal space just inside the mid-clavicular line, heaving (LVH); S1, S2 normal; **no murmurs**; no S3/S4; no pericardial rub	Excludes clinically obvious cardiac source of embolism — mitral stenosis, IE, prosthetic valve; LVH indicates long-standing hypertension
Respiratory	Trachea central; bilateral normal vesicular breath sounds; no crepitations	Aspiration pneumonia is the commonest infection after stroke
Abdomen	Soft, non-tender; no organomegaly; no renal bruit; bladder not palpable	Urinary retention; renal artery stenosis in atherosclerosis

Functional assessment — record it in every stroke case

Scale	Score in this patient	Meaning
NIH Stroke Scale (NIHSS) — today	8 (facial palsy 2, right arm 2, right leg 1, language 2, dysarthria 1)	Moderate stroke (initial NIHSS at admission was about 14)
Modified Rankin Scale (mRS)	**3**	Moderate disability — needs some help but walks with assistance
Barthel Index (activities of daily living)	55 / 100	Needs help with bathing, dressing, stairs and toilet

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SECTION C · DIAGNOSIS

Summary, Localisation and Final Clinical Diagnosis

Anatomical → pathological → aetiological → functional diagnosis

Summary of examination

Say this after your history summary

On examination, he is conscious, oriented and cooperative, with **BMI 26.9**, **BP 158/94 mmHg** (uncontrolled), a **regular pulse**, a **left carotid bruit**, arcus and grade II hypertensive retinopathy. Higher functions show **Broca's (non-fluent motor) aphasia** with mild dysarthria. Cranial nerves show **right UMN facial palsy** and **right UMN hypoglossal palsy**. Motor examination shows **right spastic hemiparesis** — tone increased (clasp-knife), power 1–3/5 in the right upper limb and 2–4/5 in the right lower limb in a pyramidal pattern, **upper limb more than lower limb**, brisk deep reflexes with unsustained ankle clonus, Hoffmann's positive, **right extensor plantar**, and absent right abdominal reflexes. Sensory system, cerebellar functions, visual fields and other systems are normal. He walks with a **hemiplegic gait** with support; **mRS 3**.

Localising the lesion — step by step

Question	Answer	Conclusion
Is it UMN or LMN?	Spasticity, brisk reflexes, extensor plantar, no wasting/fasciculations	**UMN** — corticospinal tract
Which side of the brain?	Right-sided limbs and face; aphasia in a right-hander	**Left** cerebral hemisphere
Above or below the brainstem?	Face (VII, XII) involved on the **same side** as limbs; no crossed signs; higher function (aphasia) involved	**Supratentorial — cerebral hemisphere**
Cortex or internal capsule?	**Aphasia** (cortical sign); upper limb ≫ lower limb (unequal — cortex is spread out); in capsular lesions face, arm and leg are equally affected and there is no aphasia	**Cortical + subcortical** (frontal lobe)
Which vascular territory?	Face and arm > leg, Broca's aphasia, no sensory or field loss	**Superior division of the left middle cerebral artery**

Localisation of hemiplegia — the master table

Site of lesion	Distinguishing features
Cerebral cortex	Unequal weakness (monoplegia or face–arm or leg predominant), cortical signs — aphasia (dominant), neglect/apraxia (non-dominant), cortical sensory loss, seizures, field defects
Internal capsule	Dense, **equal** weakness of face, arm and leg (fibres packed together); no cortical signs; pure motor stroke (lacune); may have hemisensory loss and hemianopia if posterior limb / large lesion
Midbrain (Weber syndrome)	Ipsilateral **III nerve palsy** + contralateral hemiplegia (crossed hemiplegia)
Pons (Millard–Gubler / Foville)	Ipsilateral **VI and LMN VII** palsy ± gaze palsy + contralateral hemiplegia
Medulla (medial medullary / Dejerine)	Ipsilateral **XII** palsy (LMN) + contralateral hemiplegia and loss of posterior column sensation
Cervical spinal cord (Brown-Séquard)	Ipsilateral hemiplegia (face spared) + ipsilateral posterior column loss + **contralateral** pain and temperature loss; sensory level

Final clinical diagnosis

Write it in this format

Right-sided spastic hemiparesis (upper limb > lower limb) with right UMN facial and hypoglossal palsy and Broca's (non-fluent) aphasia [*clinical / anatomical*] — due to a lesion in the **left frontal cortex and subcortical white matter, in the territory of the superior division of the left middle cerebral artery** [*site*] — most probably an **ischaemic stroke (infarct)** [*pathology*] — due to **large-artery atherosclerosis (artery-to-artery embolism from left ICA stenosis — TOAST class 1)**, preceded by a TIA [*aetiology*] — in a patient with **uncontrolled hypertension, type 2 diabetes mellitus, smoking and overweight** [*risk factors*] — **6 weeks after onset, in the recovery (spastic) phase, mRS 3**, with **right hemiplegic shoulder subluxation** [*stage, function and complications*].

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Differential diagnosis — for and against

Condition	Points in favour	Points against
Ischaemic stroke — large-artery atherosclerosis (most likely)	Sudden onset on waking; preceding TIA in the same territory; HTN, DM, smoking; left carotid bruit; cortical signs	— (to be confirmed by imaging and carotid Doppler)
Cardioembolic stroke	Sudden onset, maximal at onset; cortical (MCA branch) involvement	Regular pulse, no murmur, no recent MI; carotid bruit explains the cause — still exclude paroxysmal AF with Holter and echo
Intracerebral haemorrhage (hypertensive)	Hypertension; sudden onset	No headache, vomiting or altered sensorium; occurred during sleep; aphasia without drowsiness; CT showed no blood
Lacunar infarct (small-vessel disease)	Hypertension, diabetes	**Aphasia** (cortical sign) and unequal face–arm–leg weakness exclude a pure lacunar syndrome
Space-occupying lesion (glioma, metastasis, abscess, tuberculoma)	Hemiparesis with cortical signs	Sudden onset and improving course; no headache, seizures, fever or papilloedema
Chronic subdural haematoma	Elderly; alcohol	No trauma, no fluctuating sensorium or headache; sudden onset
Todd's paralysis	Transient hemiparesis	No seizure; deficit persisting for 6 weeks (Todd's lasts <48 h)
Hypoglycaemia (stroke mimic)	Diabetic	Deficit persisted after glucose correction; CBG normal
Demyelination (MS, ADEM)	Hemiparesis	Age 62; sudden onset; vascular risk factors
Cerebral venous thrombosis	Stroke with cortical signs	No headache, seizures, papilloedema; male without prothrombotic risk
Vasculitis (TB meningitis, syphilis, SLE)	Stroke	No fever, meningism, rash or systemic features — check VDRL, HIV

Causes of hemiplegia — classification (a favourite viva question)

Group	Causes
Vascular (commonest)	Ischaemic stroke — atherothrombosis, cardioembolism (AF, MS, IE, MI, LV aneurysm), lacunar; intracerebral haemorrhage; subarachnoid haemorrhage with vasospasm; cerebral venous thrombosis; carotid dissection
Infective / inflammatory	Brain abscess, tuberculoma, tuberculous meningitis with vasculitis, neurocysticercosis, HIV-related (toxoplasmosis, PML), syphilis, herpes encephalitis; vasculitis (SLE, PAN), APLA syndrome
Neoplastic	Glioma, meningioma, metastases (lung, breast), lymphoma
Traumatic	Subdural and extradural haematoma, contusion
Demyelinating	Multiple sclerosis, acute disseminated encephalomyelitis
Metabolic / others	Hypoglycaemia, Todd's palsy, hemiplegic migraine, hyperosmolar state, conversion disorder (functional)
In the young (<45 years)	RHD with MS/AF, IE, cardiomyopathy, PFO (paradoxical embolism), dissection, vasculitis, APLA, sickle cell, homocystinuria, oral contraceptives, drug abuse (cocaine), moyamoya, CADASIL, peripartum CVT

Major vascular territories — stroke syndromes

Artery	Clinical features
MCA — main stem	Contralateral hemiplegia (face, arm > leg), hemisensory loss, homonymous hemianopia, gaze deviation; global aphasia (dominant) or neglect/anosognosia (non-dominant)
MCA — superior division	Face and arm weakness and sensory loss; Broca's aphasia (dominant) — **this patient**
MCA — inferior division	Wernicke's aphasia (dominant) or neglect (non-dominant); upper quadrantanopia; little weakness
ACA	Contralateral **leg > arm** weakness, urinary incontinence, abulia, grasp reflex, transcortical motor aphasia
PCA	Contralateral homonymous hemianopia with macular sparing; alexia without agraphia (dominant); thalamic sensory loss and pain
Lacunar (penetrating arteries)	Pure motor, pure sensory, sensorimotor, ataxic hemiparesis, dysarthria–clumsy hand — no cortical signs
Vertebrobasilar	Crossed signs, vertigo, diplopia, dysarthria, dysphagia, ataxia, bilateral signs, coma (basilar), lateral medullary (Wallenberg) syndrome

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SECTION D · INVESTIGATIONS

Investigations — Plan, Rationale and Results

Confirm the pathology, define the vascular territory, find the cause and screen for risk factors

Investigation	Why	Result in this patient
Capillary blood glucose (immediately)	Exclude hypoglycaemia — the commonest stroke mimic; a must before thrombolysis	182 mg/dL
NCCT brain (immediately, at admission)	Differentiates infarct from haemorrhage; excludes mimics; decides thrombolysis. Early signs: hyperdense MCA sign, loss of insular ribbon, obscured lentiform nucleus, sulcal effacement (ASPECTS score)	No haemorrhage; subtle hypodensity in the left frontal operculum and insula; ASPECTS 8
MRI brain with DWI, FLAIR, GRE/SWI	DWI shows infarct within minutes; DWI–FLAIR mismatch helps in wake-up stroke; SWI shows microbleeds	Infarct in the left inferior frontal gyrus, insula and corona radiata — **superior division of left MCA**; old small-vessel changes
Carotid Doppler / CT or MR angiography (head and neck)	Large-vessel stenosis or occlusion; plan for endarterectomy / stenting / thrombectomy	**Left ICA 75 % stenosis** with a heterogeneous (ulcerated) plaque; right ICA 30 %
ECG	AF, LVH, old MI	Sinus rhythm 82/min; LVH by voltage criteria with strain
24–72-h Holter monitoring	Paroxysmal AF (changes treatment to anticoagulation)	No AF
2D echocardiography (TTE; TEE if young/cryptogenic)	LV thrombus, valve disease, vegetations, PFO, LA size	Concentric LVH, EF 58 %, no clot, normal valves
Complete blood count, ESR	Anaemia, polycythaemia, thrombocytosis, infection	Hb 13.8 g/dL, TC 8400/µL, platelets 2.6 lakh/µL, ESR 18 mm/h
FBS, PPBS, HbA1c	Diabetes control	FBS 148, PPBS 236 mg/dL, **HbA1c 8.4 %**
Fasting lipid profile	Dyslipidaemia — target for statin	TC 236, **LDL 162**, HDL 34, TG 210 mg/dL
Renal function, electrolytes	Before contrast, ACEi/ARB; hyponatraemia	Urea 32, creatinine 1.0 mg/dL; Na 138, K 4.2 mmol/L
Liver function tests	Before statin; baseline	Normal
Coagulation profile (PT/INR, aPTT)	Before thrombolysis/anticoagulation	Normal
Urine microalbumin; chest X-ray	Nephropathy; aspiration, cardiomegaly	UACR 48 mg/g (microalbuminuria); CXR normal
Swallow assessment (bedside water-swallow test)	Aspiration risk — before giving oral food/drugs	Normal now
Selected cases: VDRL, HIV, TSH, homocysteine, APLA antibodies, ANA, sickle test, CSF	Young or cryptogenic stroke, suspected vasculitis/infection	VDRL and HIV non-reactive

Imaging pearl — learn stroke imaging online

CT is the first investigation in acute stroke because it is fast and reliably detects haemorrhage; it may be normal for the first 6–12 hours in infarction. **MRI DWI** is the most sensitive test for acute infarction. See real CT and MRI images of infarcts on Radiopaedia (free):

▶ Stroke imaging — basics for MBBS

▶ Ischaemic stroke — detailed article

Scan to open

TOAST classification of ischaemic stroke (aetiology)

TOAST class	Key evidence
1. Large-artery atherosclerosis	>50 % stenosis of the relevant artery; cortical or large subcortical infarct — **this patient**
2. Cardioembolism	AF, MS, prosthetic valve, recent MI, LV thrombus, IE
3. Small-vessel occlusion (lacune)	Lacunar syndrome, infarct <1.5 cm, HTN/DM
4. Other determined cause	Dissection, vasculitis, hypercoagulable state, CVT
5. Undetermined (cryptogenic)	Two or more causes, or none found despite evaluation

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SECTION E · MANAGEMENT

Management of Ischaemic Stroke

Acute treatment · secondary prevention · rehabilitation · complications

A. Acute management (what should be done in the first 24–48 hours)

Step	Action
Stroke code / ABC	Airway, breathing (O_2 only if SpO_2 <94 %), circulation; CBG; note the time last seen normal; NIHSS; urgent NCCT ± CT angiography
IV thrombolysis	Within **4.5 hours** of last-seen-normal: **alteplase 0.9 mg/kg** (max 90 mg) — 10 % as IV bolus over 1 min, the rest over 60 min; or **tenecteplase 0.25 mg/kg** single bolus (max 25 mg). BP must be <185/110 before and <180/105 for 24 h after. Wake-up stroke: eligible if MRI shows DWI–FLAIR mismatch
Mechanical thrombectomy	Large-vessel occlusion (ICA, M1, basilar) within 6 h, and up to 24 h with favourable perfusion imaging (DAWN, DEFUSE-3)
Antiplatelet	Aspirin 150–325 mg within 24–48 h (24 h after thrombolysis). **Minor stroke (NIHSS ≤3) or high-risk TIA**: dual antiplatelet — aspirin + clopidogrel (300–600 mg load, then 75 mg) for 21 days, then single agent (CHANCE, POINT)
Blood pressure	If not thrombolysed: permissive hypertension — treat only if >220/120 mmHg (lower by 15 % in 24 h). Labetalol or nicardipine infusion when needed
Glucose, temperature	Keep glucose 140–180 mg/dL; treat fever (paracetamol) and look for infection
Swallow screen	Nil by mouth until swallow test passed; Ryle's tube feeding if unsafe
DVT prophylaxis	Intermittent pneumatic compression; LMWH in immobile patients after 24–48 h if no haemorrhage
Position and nursing	Head end 30° in large infarcts; 2-hourly turning, air mattress, bladder care (avoid catheter), early mobilisation after 24 h
Malignant MCA infarct	Decompressive hemicraniectomy within 48 h in patients ≤60 years — reduces mortality

In this patient, he arrived 11 hours after he was last seen normal, so thrombolysis was not possible without an MRI mismatch; thrombectomy was not done (branch occlusion). He received aspirin, a statin and early physiotherapy.

B. Secondary prevention — the long-term prescription for this patient

Target	Drug / measure	Goal
Antiplatelet	Aspirin 75 mg OD (or clopidogrel 75 mg OD) — lifelong. Anticoagulation only if AF/cardioembolism is found	Prevent recurrent atherothrombotic stroke
Carotid stenosis	**Left carotid endarterectomy** (symptomatic 70–99 % stenosis) — ideally within 2 weeks of the event; carotid stenting if surgical risk high	Absolute risk reduction ~16 % over 5 years (NASCET)
Lipids	Atorvastatin 40–80 mg HS ± ezetimibe 10 mg	LDL <70 mg/dL (<55 in very high risk)
Blood pressure	Telmisartan 40–80 mg + amlodipine 5–10 mg (add chlorthalidone 12.5 mg if needed); home BP monitoring	<130/80 mmHg
Diabetes	Metformin 1 g BD + add SGLT2 inhibitor or GLP-1 receptor agonist (cardiovascular benefit); pioglitazone also reduces stroke recurrence in insulin resistance	HbA1c <7 %
Smoking	Complete cessation — counselling ± nicotine replacement / varenicline	Stop permanently
Lifestyle	Salt <5 g/day, DASH-type diet, weight reduction, 150 min/week activity as able, limit alcohol	BMI <23, waist <90 cm
Others	Influenza and pneumococcal vaccination; screen for sleep apnoea and depression	—

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C. Rehabilitation — multidisciplinary, starts on day 1

Problem	Management
Motor weakness	Physiotherapy — passive then active range-of-motion exercises, task-specific training, constraint-induced movement therapy for the right arm, gait training, balance exercises; walking aids (quadripod stick); ankle–foot orthosis for foot drop
Spasticity	Positioning (arm in extension, leg in flexion), daily stretching, splints. Drugs: **baclofen** 5 mg TID → up to 20 mg TID; **tizanidine** 2–8 mg TID; dantrolene. Focal spasticity: **botulinum toxin** injection into elbow/wrist flexors
Hemiplegic shoulder (subluxation, pain)	Arm support / sling while upright, avoid pulling on the arm, correct positioning, functional electrical stimulation, analgesics, intra-articular steroid if needed
Aphasia	Speech and language therapy — intensive, early; communication boards; involve family; most recovery occurs in the first 3–6 months
Dysphagia	Swallow therapy, modified consistency diet; avoid aspiration
Activities of daily living	Occupational therapy — one-handed techniques, adaptive devices; western commode / raised toilet seat and grab bars at home
Post-stroke depression, emotional lability	Screen (PHQ-9); counselling; **SSRI** such as escitalopram 5–10 mg or fluoxetine 20 mg
Bladder and bowel	Timed voiding; avoid indwelling catheter; laxatives for constipation
Return to work and driving	Vocational rehabilitation; no driving for at least 1 month and until fit (right-sided weakness)

D. Complications of stroke — prevent, detect and treat

- **Neurological:** cerebral oedema and herniation (days 2–5), haemorrhagic transformation, recurrent stroke, post-stroke seizures, post-stroke dementia, central post-stroke pain.
- **Infections:** aspiration pneumonia (commonest cause of death after the first week), urinary tract infection.
- **Venous thromboembolism:** DVT and pulmonary embolism.
- **Immobility:** pressure sores, contractures, frozen shoulder, osteoporosis, falls and fractures.
- **Cardiac:** arrhythmias, myocardial infarction, neurogenic stunning.
- **Others:** depression, malnutrition, dehydration, hyponatraemia (SIADH), constipation, shoulder–hand syndrome (complex regional pain syndrome).

E. Prognosis

Most motor recovery occurs within the first **3 months** and continues more slowly up to 6–12 months; the leg recovers earlier and better than the arm, and proximal before distal. Language may continue to improve for a year or more. **Good prognostic factors** in this patient: early return of leg movement, preserved comprehension, intact sensation and visual fields, absence of neglect, and good family support. **Poor prognostic factors**: dense hand weakness at 6 weeks, age, diabetes, and uncontrolled risk factors. Risk of recurrent stroke is highest in the first weeks after a TIA or stroke, especially with uncorrected carotid stenosis.

F. Prescription at discharge (sample)

Drug	Dose	Remarks
Tab. Aspirin	75 mg OD after food	Lifelong
Tab. Atorvastatin	80 mg HS	Check LFT and lipids at 6–8 weeks
Tab. Telmisartan + amlodipine	40/5 mg OD	Titrate to BP <130/80; check creatinine and K^+
Tab. Metformin	1 g BD	With meals
Tab. Empagliflozin	10 mg OD	Hydration; genital hygiene
Tab. Baclofen	10 mg TID	Drowsiness; do not stop abruptly
Tab. Escitalopram	5 mg OD	Review in 4 weeks
Physiotherapy, speech therapy, occupational therapy	Daily	Home programme for caregivers
Referral	Vascular surgery — left carotid endarterectomy	Urgent

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SECTION F · PRESENTATION

How to Present This Case in the Examination

A 5-minute presentation that impresses the examiner

Structure of the presentation

- **Introduce** — name (initials), age, sex, handedness, occupation, place.
- **Chief complaints** with duration.
- **History of presenting illness** — onset, progression, distribution, associated features, relevant negatives, treatment received, current status.
- **Risk factors** — past, personal, family, drug history.
- **Summary of history** and what you think from the history.
- **General examination** — positives and relevant negatives, vitals, carotids, fundus.
- **CNS examination** in order — HMF, cranial nerves, motor, sensory, cerebellum, others.
- **Other systems** briefly.
- **Summary and diagnosis** — in the four-part format; then give differential diagnoses only if asked.

Model presentation (read aloud and practise until you can say it in 4–5 minutes)

Sir / Madam, I would like to present the case of…

"…Mr K. R., a **62-year-old right-handed retired bus conductor** from Aluva, who presented with **weakness of the right upper and lower limbs, difficulty in speaking and deviation of the angle of the mouth to the left for 6 weeks**, and stiffness of the right limbs for 4 weeks.

He was apparently normal 6 weeks ago when, **on waking up**, he noticed sudden weakness of the right upper and lower limbs, more in the upper limb, maximal at onset, with inability to express himself although he could understand what was said. The angle of the mouth deviated to the left with drooling, but eye closure was normal. He had **transient right-hand weakness with speech difficulty lasting 15 minutes, 2 months earlier**. There was **no loss of consciousness, headache, vomiting, seizures, fever, trauma, visual, sensory or bladder symptoms, and no palpitations**. He was treated elsewhere with aspirin and a statin, but not thrombolysed as he presented late. Since then, the leg has improved more than the arm, speech has partially improved, and stiffness developed after 2 weeks.

He is a known **hypertensive for 8 years and diabetic for 5 years on irregular treatment**, a **smoker of 30 pack-years**, with a family history of stroke in his father and MI in his brother.

On general examination he is conscious and oriented, overweight (BMI 26.9), pulse 84/min regular, **BP 158/94 mmHg**, with a **left carotid bruit**, arcus and grade II hypertensive retinopathy.

On CNS examination, he has **Broca's aphasia** with mild dysarthria; **right UMN facial and hypoglossal palsy**; **right spastic hemiparesis**, upper limb more than lower limb, with power of 1–3/5 in the upper limb and 2–4/5 in the lower limb in a pyramidal pattern, **exaggerated reflexes, unsustained ankle clonus, Hoffmann's positive and an extensor plantar** on the right. Sensory, cerebellar and other systems are normal. He walks with a hemiplegic gait with support.

So my diagnosis is **right spastic hemiparesis with right UMN facial palsy and Broca's aphasia due to an infarct in the territory of the superior division of the left middle cerebral artery, probably due to large-artery atherosclerosis of the left ICA, in a hypertensive, diabetic smoker, 6 weeks after onset, in the recovery phase, mRS 3**."

Do's and don'ts during presentation

Do	Don't
Speak confidently, in chronological order	Read from your notes
State relevant negatives — they show your thinking	Present every normal finding at length
Mention handedness and the time last seen normal	Call it 'CVA' without saying ischaemic or haemorrhagic and the territory
Give the diagnosis in the anatomical–pathological–aetiological–functional format	Jump to investigations before you are asked
Be ready to demonstrate any sign you have reported (plantar, clonus, aphasia)	Report a sign you have not elicited yourself

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SECTION G · VIVA VOCE

Frequently Asked Viva Questions with Answers

Short, exam-ready answers

Q1 Define stroke.

A rapidly developing clinical syndrome of focal (or global) disturbance of cerebral function lasting **more than 24 hours** or leading to death, with no apparent cause other than vascular origin (WHO). The tissue-based definition (AHA/ASA 2013) defines stroke as an episode of neurological dysfunction caused by focal cerebral, spinal or retinal **infarction**, regardless of duration.

Q2 What is a TIA? Why is it important?

A transient episode of neurological dysfunction caused by focal ischaemia **without acute infarction** on imaging; most last <1 hour. About 10 % of patients have a stroke within 90 days, half within 48 hours — it is a medical emergency. Risk is assessed by the **ABCD² score** (Age ≥60, BP ≥140/90, Clinical features — weakness 2, speech 1, Duration ≥60 min 2 / 10–59 min 1, Diabetes).

Q3 Define hemiplegia, hemiparesis, monoplegia, crossed hemiplegia.

Hemiplegia — paralysis of one half of the body (face, arm, leg). **Hemiparesis** — partial weakness of one half. **Monoplegia** — weakness of one limb (cortical, spinal or peripheral). **Crossed hemiplegia** — ipsilateral cranial nerve palsy (LMN) with contralateral hemiplegia — a **brainstem** lesion.

Q4 Why is the initial phase flaccid?

'**Cerebral (neural) shock**' — sudden loss of descending supraspinal input depresses the spinal motor neurons and stretch reflexes for days to weeks. Tone and reflexes then increase as the spinal segmental reflexes become disinhibited (spastic phase).

Q5 What is spasticity? Differentiate it from rigidity.

Spasticity is a **velocity-dependent** increase in tone due to UMN lesion — **clasp-knife**, more in UL flexors and LL extensors, with brisk reflexes. Rigidity (extrapyramidal) is **velocity-independent**, present equally in flexors and extensors throughout the range (**lead-pipe**, or **cog-wheel** with tremor); reflexes normal.

Q6 Why are superficial abdominal reflexes lost in UMN lesions?

Superficial reflexes are **polysynaptic** reflexes with a cortical (transcortical) component; the corticospinal pathway facilitates them, so they are lost in pyramidal lesions while deep reflexes, which are released from inhibition, become brisk.

Q7 What is the Babinski sign? Where else is it seen?

Dorsiflexion of the great toe with fanning of the other toes on plantar stimulation — sign of a **pyramidal tract lesion**. Also seen normally in infants (<1–2 years), deep sleep, deep anaesthesia, coma, post-ictal state, hypoglycaemia and after an overdose.

Q8 Where is the lesion if the face, arm and leg are equally weak with no cortical signs?

Internal capsule (posterior limb) — usually a lacunar infarct from a lenticulostriate artery or a capsular haemorrhage.

Q9 Which artery supplies the internal capsule?

Mainly the **lenticulostriate branches of the MCA** ('artery of cerebral haemorrhage' — Charcot), the anterior choroidal artery (posterior limb, inferior part) and the recurrent artery of Heubner (anterior limb, from ACA).

Q10 What is the commonest site of hypertensive intracerebral haemorrhage?

Putamen (most common) — then thalamus, pons, cerebellum and subcortical white matter; due to rupture of Charcot–Bouchard micro-aneurysms of penetrating arteries.

Q11 How do you differentiate infarct and haemorrhage clinically?

Haemorrhage favours: onset during activity, severe headache, vomiting, early altered sensorium, very high BP, neck stiffness, rapid worsening. However, **clinical features are unreliable — CT is mandatory**. Siriraj or Allen scores were used before CT but are not reliable enough to start antithrombotics.

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Q12 What are the eligibility criteria and contraindications for IV thrombolysis?

Eligible: clinical diagnosis of ischaemic stroke with measurable deficit, onset (last seen normal) **≤4.5 h**, age ≥18, CT excludes haemorrhage. Contraindications: intracranial haemorrhage on CT, previous ICH, ischaemic stroke or severe head injury within 3 months, intracranial neoplasm/AVM, recent intracranial or spinal surgery, GI bleeding within 21 days, platelets <1 lakh, INR >1.7, therapeutic LMWH within 24 h or DOAC within 48 h, BP persistently >185/110 despite treatment, glucose <50 mg/dL, aortic dissection, infective endocarditis.

Q13 What is the dose of alteplase in stroke? How is it different from MI?

Stroke: **0.9 mg/kg** (max 90 mg), 10 % as bolus and 90 % over 60 min. Tenecteplase: **0.25 mg/kg** bolus (max 25 mg). MI uses higher, front-loaded doses (alteplase up to 100 mg; tenecteplase 30–50 mg).

Q14 What is the 'time is brain' concept?

In a typical large-vessel ischaemic stroke, about **1.9 million neurons** are lost every minute without treatment. The **ischaemic penumbra** — hypoperfused but viable tissue around the core — can be saved by early reperfusion.

Q15 Why should BP not be lowered aggressively in acute ischaemic stroke?

Cerebral autoregulation is impaired in the ischaemic area; perfusion of the penumbra becomes pressure-dependent. Rapid lowering may extend the infarct. Treat only if >220/120 mmHg (or >185/110 if thrombolysis planned).

Q16 When is anticoagulation indicated in stroke? When should it be started?

Cardioembolic stroke — AF, mechanical valve, LV thrombus, CVT. Timing after AF-related stroke (ELAN trial, '1-3-6-12 rule' simplified): early start (within 48 h for minor/moderate, day 6–7 for major stroke) is safe in most patients after imaging excludes significant haemorrhagic transformation. DOACs are preferred over warfarin, except in mechanical valves and moderate–severe MS.

Q17 What are the indications for carotid endarterectomy?

Symptomatic carotid stenosis **70–99 %** (benefit also in 50–69 % in selected men) in a patient with non-disabling stroke or TIA in that territory, ideally **within 2 weeks**. Not indicated for complete occlusion or <50 % stenosis. Asymptomatic stenosis ≥60–70 % — selected patients.

Q18 What is a lacunar infarct? Name the lacunar syndromes.

A small (<15 mm) subcortical infarct from occlusion of a single penetrating artery, mainly due to lipohyalinosis in hypertension and diabetes. Syndromes: **pure motor hemiparesis** (posterior limb of internal capsule — commonest), **pure sensory stroke** (thalamus), **sensorimotor stroke**, **ataxic hemiparesis** (pons/internal capsule), **dysarthria–clumsy hand** (pons/genu).

Q19 What is Weber syndrome? Millard–Gubler syndrome?

Weber: lesion of the ventral midbrain — ipsilateral III nerve palsy + contralateral hemiplegia. **Millard–Gubler**: ventral pons — ipsilateral VI and LMN VII palsy + contralateral hemiplegia. **Benedikt**: midbrain tegmentum — III palsy + contralateral tremor/ataxia.

Q20 What is the lateral medullary (Wallenberg) syndrome?

Occlusion of the **vertebral artery / PICA** → ipsilateral facial pain and temperature loss, Horner's syndrome, cerebellar ataxia, dysphagia and hoarseness (IX, X), vertigo and nystagmus; **contralateral** loss of pain and temperature over the body. **No hemiplegia**.

Q21 What is anosognosia? In which lesion is it seen?

Unawareness or denial of one's own hemiplegia — seen with **non-dominant (right) parietal** lesions, along with hemi-neglect, dressing apraxia and constructional apraxia.

Q22 What is Gerstmann syndrome?

Lesion of the **dominant angular gyrus** (parietal lobe): finger agnosia, acalculia, agraphia, right–left disorientation.

Q23 What is a watershed (border-zone) infarct?

Infarction at the junction of two arterial territories (ACA–MCA, MCA–PCA) due to **hypoperfusion** — cardiac arrest, severe hypotension, or severe carotid stenosis. ACA–MCA watershed gives 'man-in-a-barrel' (proximal arm and shoulder weakness) and transcortical aphasia.

Abnormal gaits — differentiate from the hemiplegic gait

Gait	Cause
Hemiplegic (circumduction)	UMN lesion — stroke
Scissoring	Bilateral UMN — spastic paraplegia, cerebral palsy
High-stepping	Foot drop — common peroneal palsy, L5 radiculopathy, peripheral neuropathy
Festinant / shuffling	Parkinsonism
Broad-based, reeling	Cerebellar ataxia
Stamping	Sensory ataxia — posterior column disease
Waddling	Proximal myopathy
Magnetic / apraxic	Normal-pressure hydrocephalus, frontal lobe disease

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Q24 What is the most common cause of death in stroke?

In the first week: the stroke itself — cerebral oedema with herniation (large infarcts, haemorrhage). After the first week: **aspiration pneumonia**, followed by cardiac causes (MI, arrhythmias) and pulmonary embolism.

Q25 What is a malignant MCA infarct?

A large MCA-territory infarct (>50 % of the territory) causing massive oedema and herniation on days 2–5, with high mortality (~80 %). **Decompressive hemicraniectomy** within 48 hours in patients ≤60 years reduces mortality by about half.

Q26 How do you assess the severity of stroke?

NIH Stroke Scale (0–42): level of consciousness, gaze, visual fields, facial palsy, motor arm and leg, ataxia, sensation, language, dysarthria, extinction. 1–4 minor, 5–15 moderate, 16–20 moderate–severe, >20 severe. Outcome: **modified Rankin Scale** (0 = no symptoms to 6 = dead) and Barthel Index.

Q27 What are the stroke mimics?

Hypoglycaemia (commonest), post-ictal Todd's palsy, migraine with aura, functional (conversion) disorder, sepsis with recrudescence of an old deficit, brain tumour, subdural haematoma, encephalitis, hyponatraemia, Wernicke's encephalopathy, peripheral vertigo, Bell's palsy, drug toxicity.

Q28 Why does the upper limb recover less than the lower limb?

The leg has more **bilateral** cortical representation and is supported by brainstem pathways (reticulospinal, vestibulospinal) that control proximal and axial muscles; fine distal hand movements depend almost entirely on the crossed corticospinal tract.

Q29 What is the posture in hemiplegia? Why?

Wernicke–Mann posture: upper limb adducted at the shoulder, flexed at the elbow, wrist and fingers, forearm pronated; lower limb extended at hip and knee, foot plantar-flexed and inverted. Due to unopposed antigravity muscle activity (UL flexors, LL extensors) mediated by vestibulospinal and reticulospinal pathways when the corticospinal tract is damaged.

Q30 What are the causes of stroke in a young Indian patient?

Rheumatic heart disease (MS with AF), infective endocarditis, cardiomyopathy, tuberculous meningitis with vasculitis, cerebral venous thrombosis (puerperium, OCP), arterial dissection, APLA syndrome, SLE, homocystinuria, sickle cell disease, Takayasu arteritis, moyamoya disease, illicit drugs, PFO with paradoxical embolism.

Q31 What is cerebral venous thrombosis? How is it treated?

Thrombosis of the dural sinuses/cortical veins; presents with headache, papilloedema, seizures, focal deficits, altered sensorium; common in the puerperium and with OCPs, dehydration and thrombophilia in India. Diagnosed by MR venography; treated with **anticoagulation (LMWH, even in the presence of haemorrhagic infarct)**, then warfarin or dabigatran for 3–12 months.

Q32 Name drugs used for spasticity.

Baclofen (GABA-B agonist), **tizanidine** (α2 agonist), **dantrolene** (acts on muscle — ryanodine receptor), diazepam, gabapentin; **botulinum toxin** for focal spasticity; intrathecal baclofen for severe cases.

Q33 What is the role of statins in stroke?

High-intensity atorvastatin (80 mg) reduced recurrent stroke by 16 % after stroke/TIA (**SPARCL**). Target LDL <70 mg/dL (Treat Stroke to Target trial). Statins also stabilise carotid plaque.

Q34 What is the difference between a stroke and a stroke-in-evolution?

Completed stroke: deficit maximal at onset and stable. **Stroke-in-evolution (progressing stroke)**: deficit worsens over hours to days — seen in thrombotic occlusion, lacunar 'capsular warning syndrome', haemorrhage expansion or oedema.

Q35 List the components of the ABCD² score and the CHA_2DS_2-VASc score.

ABCD² (TIA): Age ≥60 (1), BP ≥140/90 (1), Clinical — unilateral weakness (2) or speech disturbance alone (1), Duration ≥60 min (2) / 10–59 min (1), Diabetes (1). **CHA_2DS_2-VASc** (AF): CHF, HTN, Age ≥75 (2), DM, Stroke/TIA (2), Vascular disease, Age 65–74, Sex category (female).

Last-minute checklist before you go to the examiner

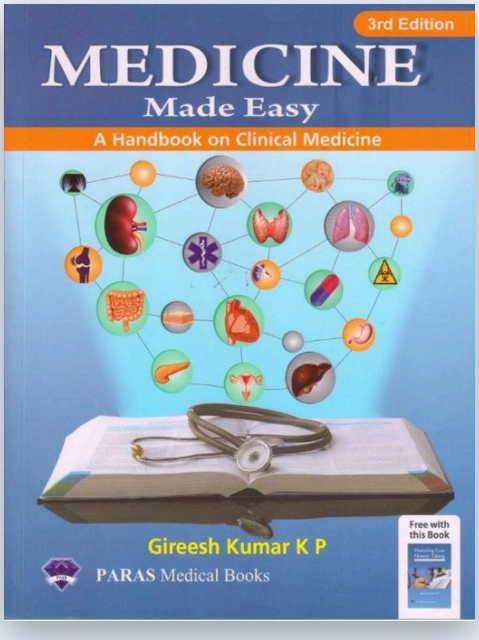
✔ Handedness · ✔ time last seen normal · ✔ TIA history · ✔ BP both arms · ✔ pulse rhythm · ✔ carotid bruit · ✔ fundus · ✔ six components of speech · ✔ UMN vs LMN face · ✔ tone, power table, reflexes with plantar · ✔ sensory and visual fields · ✔ gait · ✔ mRS · ✔ four-part diagnosis · ✔ management in three parts — acute, secondary prevention, rehabilitation.

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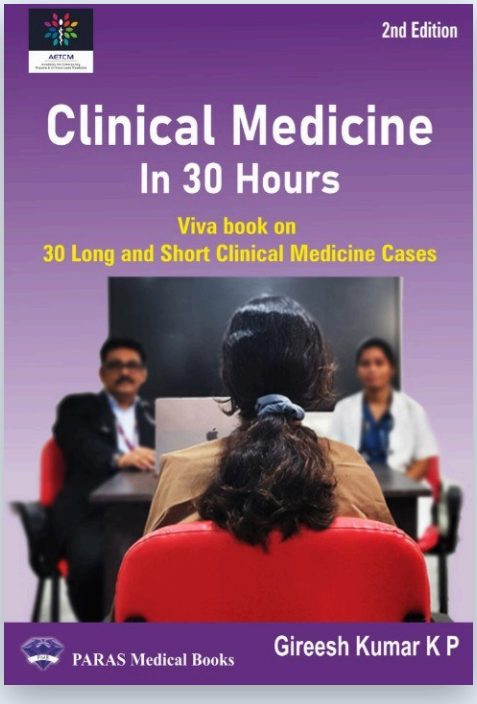
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