

ACUTE FOCAL NEUROLOGICAL DEFICIT



1) Onset of symptoms



2) Non-contrast CT

Rules out intracerebral hemorrhage, and usually SAH



3) Consider CTA / CTP / CTV

Rules out AIS, venous sinus thrombosis



4) Consider lumbar puncture

Definitively rules out SAH, rules out neuroinfection



5) Evaluate laboratory tests

Rules out metabolic cause / hypoglycemia



6) Clinical reasoning

Migraine / functional disorder

CATEGORY	DIAGNOSIS	SIGNS / SYMPTOMS	DIAGNOSTICS	MANAGEMENT
KILLERS (Hyperacute, risk of death)	Acute ischemic stroke with large-vessel occlusion (LVO)	Sudden focal deficit — hemiparesis, gaze deviation, aphasia, neglect; posterior circulation may show vertigo, diplopia, ataxia, decreased consciousness. High NIHSS.	Non-contrast CT (rules out hemorrhage) → CT angiography head/neck to identify occlusion → CT/MR perfusion or DWI to estimate core vs. penumbra.	IV thrombolysis (alteplase/tenecteplase) within 4.5 h if eligible; mechanical thrombectomy for anterior LVO 0–24 h (with imaging selection); permissive hypertension.
	Intracerebral hemorrhage (ICH)	Sudden focal deficit with headache, vomiting, depressed consciousness, very high blood pressure; indistinguishable from ischemia clinically.	Non-contrast CT is near-100% sensitive for acute blood; CTA "spot sign" predicts expansion; check coagulation labs and anticoagulant history.	Rapid BP lowering, reversal of anticoagulation, neurosurgical consult for evacuation/EVD; ICU care.
	Subarachnoid hemorrhage (SAH)	Thunderclap headache , meningismus, transient/persistent focal deficit (e.g., CN III palsy), decreased consciousness.	Non-contrast CT (highest sensitivity <6 h); LP for xanthochromia if CT negative; CTA/DSA for aneurysm.	Aneurysm securing (coiling/clipping), nimodipine for vasospasm prophylaxis, BP control, neurosurgery/neuro-ICU.
	Basilar artery / posterior circulation occlusion	Fluctuating consciousness, crossed deficits, quadriparesis, ocular motor abnormalities, "locked-in" risk; often missed as nonspecific dizziness/confusion.	CTA/MRA of posterior circulation; CT often normal early — low threshold for vascular imaging.	Thrombolysis/thrombectomy (extended windows may apply); emergent stroke team activation.
MAIMERS (Acute, risk of long-term deficit)	Acute compressive myelopathy (epidural mass, disc, tumor, hematoma)	Bilateral limb weakness , sensory level, bladder/bowel dysfunction, saddle anesthesia, hyperreflexia (cord) or hyporeflexia (conus/cauda).	Urgent MRI of the relevant spinal segment is the reference standard; degree of compression and cord T2 signal guide surgery.	Emergent neurosurgical/spine decompression; corticosteroids for malignant compression; delay causes permanent disability.
	Spinal epidural abscess	Back pain + fever + progressive neurologic deficit (full triad in <20%); risk factors — IV drug use, diabetes, immunocompromise, recent procedure.	MRI with contrast (triggered by neuro deficit, elevated CRP/ESR, or fever + risk factors); blood cultures.	Emergent surgical drainage + prolonged IV antibiotics; neurosurgery/ID consult.
	Transverse myelitis / demyelinating myelopathy (MS, NMOSD, ADEM)	Subacute-to-acute weakness, sensory level, sphincter dysfunction; optic neuritis suggests NMOSD/MS.	MRI spine (± brain) with and without contrast; LP (oligoclonal bands), AQP4/MOG antibodies.	High-dose IV corticosteroids, plasma exchange if refractory; disease-modifying therapy per etiology.
	Spinal cord ischemia	Sudden painless para/quadriparesis with dissociated sensory loss (anterior spinal artery); often post-aortic pathology.	MRI with DWI (restricted diffusion earlier than T2); exclude aortic dissection.	BP augmentation/CSF drainage in select cases; treat underlying cause.

CATEGORY	DIAGNOSIS	SIGNS / SYMPTOMS	DIAGNOSTICS	MANAGEMENT
 URGENT (Need quick management)	Transient ischemic attack (TIA)	Transient focal deficit, typically <60 min, fully resolved; high short-term stroke risk.	MRI-DWI preferred; CTA/MRA head/neck; ECG/telemetry/troponin; glucose, CBC, chemistry, lipids, HbA1c.	Antiplatelet therapy (DAPT for high-risk minor stroke/TIA), statin, BP and AF management, urgent carotid evaluation; early neurology consult.
	Focal seizure with postictal (Todd) paralysis	Focal deficit following witnessed convulsion or aura; deficit resolves over hours; may march/spread.	History, EEG, MRI to exclude structural lesion; glucose.	Treat underlying cause, antiseizure medication as indicated; distinguish from stroke to avoid unnecessary thrombolysis.
	Giant cell (temporal) arteritis with visual loss	New headache, jaw claudication, scalp tenderness, sudden monocular visual loss, age >50.	ESR and CRP urgently; temporal artery biopsy/ultrasound.	Immediate high-dose corticosteroids before biopsy to prevent contralateral blindness.
 REST (Not immediately dangerous)	Migraine with aura / hemiplegic migraine	Gradual, spreading positive visual/sensory phenomena, then headache; younger, prior similar episodes. Diagnosis of exclusion.	Clinical; neuroimaging to exclude structural/vascular cause on first presentation.	Abortive migraine therapy, prophylaxis; avoid triptans in hemiplegic migraine.
	Peripheral facial nerve (Bell's) palsy	Unilateral facial weakness including forehead (vs. forehead sparing in central lesion); ± hyperacusis, taste change.	Clinical; imaging if atypical or other deficits present.	Early oral corticosteroids ± antivirals; eye protection.
	Peripheral vestibular disorder (e.g., vestibular neuritis, BPPV)	Vertigo with horizontal nystagmus, no other focal deficits; HINTS reassuring for peripheral.	Bedside HINTS exam, Dix-Hallpike; imaging only if central features.	Vestibular suppressants (short course), repositioning maneuvers, vestibular rehab.
	Functional (psychogenic) neurological disorder	Deficits inconsistent with anatomy; positive signs (Hoover sign, give-way weakness, distractibility).	Positive bedside signs; imaging normal; a diagnosis of inclusion, not merely exclusion.	Clear positive diagnosis, education, physiotherapy, psychological therapy.
	Toxic/metabolic causes (hypoglycemia, hyponatremia, etc.)	Focal deficit can occur (e.g., hypoglycemic hemiparesis); usually global features predominate.	Glucose, electrolytes, renal/hepatic panel, toxicology (young patients).	Correct the underlying derangement — deficit typically reverses.

☰ KEY STEPWISE ACUTE PATHWAY:

- 1. ABCs + glucose
- 2. Rapid NIHSS / focused neuro exam and onset time
- 3. Emergent non-contrast CT
- 4. If hemorrhage → BP control, reversal, neurosurgery
- 5. If ischemic and in window → thrombolysis + CTA for LVO → thrombectomy
- 6. If spinal signs → urgent spine MRI
- 7. If resolved → TIA workup with MRI-DWI, vascular and cardiac evaluation