

Cerebrovascular Disease

1. Be able to differentiate clinically between cortical and subcortical strokes
2. Be able to identify common risk factors for stroke
diabetes, H1N, smoking, family hx of premature vascular disease, hyperlipidemia, atrial fibrillation, hx of TIA, recent MI, CHF (EF <25%), drugs (sympathomimetics, oral contraceptives, cocaine)
3. Know the significance of a TIA and the differential diagnosis of a brief neurologic spell. TIA = transient ischemic attack, transient neurologic deficit resulting from reduced blood flow, lasting less than 24 hrs and followed by full functional recovery, most resolve w/in 1 hr. Recent TIA w/ identical features suggest thrombosis or embolism within cerebral circulation TIA with different features. suggest recurrent embolism from cardiac source. DDX = Stroke, seizure, migraine, metabolic disturbance, syncope, infection
4. Be able to recognize the clinical signs of large vessel anterior circulation stroke, especially infarction of the anterior cerebral and middle cerebral arteries
 1. ACA - supply parasagittal cerebral cortex, includes part of motor and sensory cortex for contralateral leg and bladder inhibitory center. Clinical signs = contralateral paralysis and sensory loss in leg, voluntary control of bladder may be impaired.
 2. MCA - supply remainder of cerebral hemisphere and deep subcortical structures. (a) superior division supply motor and sensory for face, arms, and hands, along w/ Broca's (expressive language center in dominant hemisphere) (b) inferior division supply visual radiations, macular vision Wernicke's area (receptive language). (c) Lenticulostriate branches supply basal ganglia and motor fibers of face, hand, arm, leg in the genu and posterior limb of internal capsule. Clinical Signs = (a) superior division = contralateral hemiparesis of face, arms, and hand. Contralateral hemisensory loss. If dominant hemisphere, Broca's aphasia (impaired language expression, intact comprehension. (b) inferior division = contralateral homonymous hemianopia, impaired contralateral cortical sensory functions (graphesthesia, stereogenesis), neglect of contralateral side, anosognosia, Wernicke's aphasia (dominant hemisphere, impaired comprehension, fluent nonsensical speech). (c) Occlusion of bifurcation/trifurcation - combine the clinical signs of superior and inferior division strokes. (d) Occlusion of stem of MCA - block lenticulostriate as well as superior and inferior.
5. Be able to recognize the clinical signs of large vessel posterior circulation stroke, especially infarction of vertebral, posterior inferior cerebellar, basilar, and posterior cerebral arteries
 1. Vertebral
 2. PICA - Clinical signs - results in Lateral Medullary (Wallenberg's) syndrome, ipsilateral cerebellar ataxia, Homer's syndrome, facial sensory deficit, impaired contralateral pain and temp sensation, nystagmus, vertigo, nausea, vomiting, dysphagia, dysarthria, and hiccup. Motor systems spared because ventral location in brainstem.

3. Basilar - From the vertebral arteries, runs over ventral surface of brainstem, bifurcates into PCA at midbrain. Supply occipital and medial temporal lobes, medial thalamus, posterior limb of internal capsule, and entire brainstem and cerebellum. Clinical Signs (a) thrombosis - often incompatible w/ life. At level of dorsal pons, get CN VI palsy, hemiplegia or quadriplegia, coma. At ventral pons get "locked-in" syndrome where quadriplegic but conscious. (b) embolism - can effect reticular formation in midbrain and thalamus cause LOC, CN III palsy, Hemiplegia or quadriplegia possible.

4. PCA - supply occipital cerebral cortex, medial temporal lobes, thalamus, and rostral midbrain. Clinical signs - homonymous hemianopia of contralateral visual field, with occlusion near origin may get vertical gaze palsy, CN III palsy, internuclear ophthalmoplegia, vertical skew deviation of eyes. Occlusion to dominant occipital lobe causes aphasia (difficulty naming objects), alexia w/o agraphia (unable to read, but can write), or visual agnosia. Bilateral infarction results in cortical blindness, memory impairment, inability to recognize familiar faces (prosopagnosia)

6. Know the clinical characteristics that differentiate between thrombotic and embolic stroke
Thrombotic - 2/3 of ischemic strokes, occlude large cerebral arteries (ICA, MCA, basilar), small penetrating arteries (lacunar strokes), cerebral veins, and venous sinuses. Symptoms evolve over minutes to hrs. Often preceded by TIA in same territory causing similar deficits.

Embolic - 1/3 of ischemic strokes, from thrombus in heart, aortic arch, or large cerebral artery. In anterior circulation usually effect MCA, in posterior circulation usually effect branch point of basilar or PCA. Produce maximal neurological deficit at onset. When TIAs precede, symptoms vary because emboli lodge in different places.

7. Know how to manage blood pressure during an acute stroke.

HIN occurs after stroke as nonspecific response to cerebral injury. Increase perfusion to areas which are marginally perfused and have lost auto-regulation (ischemic penumbra). Aggressive BP lowering in acute stroke can increase area of infarction and neurologic deterioration. Treat HIN if systolic > 220 and/or diastolic > 120. Want to maintain BP in 150-180 mm Hg range. To lower BP use labetalol IV, if that doesn't work use nitroprusside IV.

8. Be able to recognize the common locations and clinical signs of hypertensive intracerebral hemorrhage.

Locations = Caudate/Putamen - branches of MCA; Pons - branches of basilar; Thalamus - branches of PCA; Cerebellum - branches of SCA; White matter (parietooccipital and temporal lobes)

Clinical Signs = (a) deep cerebral- putamen and thalamus most common, separated by posterior limb of internal capsule (descending motor, ascending sensory, optic radiations). Pressure produces contralateral sensorimotor deficit. Putamen gives more severe motor deficit, Thalamus gives more severe sensory. Transient homonymous hemianopia in thalamic, persistent in putamen. Aphasia may occur. (b) Lobar hemorrhage - subcortical white matter, symptoms depend on lobe being affected, include HA, vomiting, hemiparesis, hemisensory deficit, aphasia, and visual field abnormality. Seizures more likely. (c) Pontine hemorrhage - coma within seconds/minutes, death

within 48 hrs. Pinpoint pupils, horizontal eye movements lost, quadriparetic and decerebrate posturing. Often rupture into 4th ventricle and spread into midbrain. (d) Cerebellar hemorrhage - headache, dizziness, vomiting, inability to stand/walk begin suddenly, leads to coma within 12-24hrs. Impaired gaze to side of lesion, pupils small and reactive, ipsilateral facial weakness of LMN type

9. Be able to recognize the clinical presentation of subarachnoid hemorrhage

Pathophys - Rupture elevates intracranial pressure and distorts pain sensitive structures to produce headache. Increase intracranial pressure may acutely decrease cerebral blood flow and result in LOC. Intracranial pressure may cause subhyaloid retinal hemorrhages. Clinical-classically sudden onset, worst headache ever. Often LOC and vomiting and stiff neck. Headache is new, milder, similar headaches in weeks prior from small sentinel hemorrhages. Headache not as severe w/ A VM rupture. BP may rise, meningeal irritation may cause increase temp. Rare to have focal neurologic signs on exam.

10. Know the common etiologies of spontaneous subarachnoid hemorrhage

Usually due to ruptures cerebral arterial aneurysm or A VM, rupture berry aneurysm most common (75%), acute elevation of BP may cause rupture. Other causes of blood in subarachnoid space are intracerebral hemorrhage, embolic stroke, and trauma.

11. Know the common locations of saccular aneurysms

Berry aneurysms result from developmental weakness of vessel wall, especially at branching sites. Often involve circle of Willis. Associated with polycystic kidney and coarctation of aorta. Infections may lead to mycotic aneurysms.

Sites: Middle Cerebral Artery (29%), Anterior Communicating Artery (15%), Internal Carotid (16%), Basilar Artery (14%). .

12. Know the common complications following subarachnoid hemorrhage

Recurrence of Hemorrhage (20% over 2 wks), Intraparenchymal extension of hemorrhage, arterial vasospasm (lead to parenchymal ischemia), Acute/subacute hydrocephalus (impaired CSF absorption in subarachnoid space), Seizures, SIADH

13. Be able to recognize common lacunar stroke syndrome

- . Small penetrating arteries occluded due to changes in vessel walls induced by chronic HTN. Affect deep nuclei of brain (putamen, thalamus, caudate), pons, and posterior limb of internal capsule. Usually small size and located in silent locations, not recognized clinically. Onset is gradual, no HA, and no change in consciousness. Treat HTN to prevent future lacunar strokes. 4 classic lacunar syndromes: (a) Pure motor hemiparesis affect face, arm, and leg equally, without disturbance of sensation, vision, or language. Usually lesions in contralateral internal capsule or pons. (b) Pure sensory stroke hemisensory loss, lacunar infarction of thalamus. (c) Ataxic hemiparesis - aka ipsilateral ataxia and crural (leg) paresis. Pure motor hemiparesis combined with ataxia of hemiparetic side. Lesion in contralateral pons, internal capsule, or subcortical white matter. (d) Dysarthria-clumsy hand syndrome - dysarthria, facial weakness, dysphagia, and mild weakness of hand on side of facial involvement. Lesion in contralateral pons or internal capsule.

14. Be able to recognize the signs of arterial dissection

Hemorrhage into vessel wall, which can occlude the vessel or predispose to thrombus formation and embolization. Underlying path often cystic medial necrosis. Prodromal transient hemispheric ischemia or monocular blindness may precede stroke. Carotid

dissection - pain in jaw/neck, visual abnormalities (similar to migraine), or Horner's syndrome. Vertebral/Basilar dissection - headache, posterior neck pain, sudden brainstem dysfunction. Treatment controversial: do nothing, remove intramural hematoma, measures to prevent embolization (aspirin, anticoagulants)

15. Appreciate the differential diagnosis of stroke in younger patient

Migraines, arterial dissection, drugs (cocaine, heroin, contraceptives), Premature atherosclerosis, postpartum angiopathy, Cardiac factors (ASD, PFO, mitral prolapse, endocarditis), Hematologic factors (deficiency, DIC, TIP), Inflammatory factors (lupus, polyarteritis nodosa, neurosyphilis, cryoglobulinuria), Fibromuscular dysplasia

16. Appreciate the general indication and value of thrombolysis in an acute ischemic stroke

t-P A converts plasminogen to plasmin which can lyse fibrin clots. IV t-Pa within 3 hrs of onset of symptoms reduces disability and mortality from ischemic stroke. Give approximately 1 mg/kg. Major complication is hemorrhage, little benefit after 3 hr window. Establish onset of symptoms w/ accuracy. CT should not show large ischemic stroke or signs of hemorrhage. If patient is anticoagulated (warfarin/heparin), thrombocytopenic, seizures, prior intracranial hemorrhage, surgery within 14 days, GI bleed, or marked HIN should NOT receive IV t-P A. Intraarterial t-P A can be used 3-6 hrs after onset, especially for MCA strokes.

17. Know the treatment options designed to prevent infarction in patients with atrial fibrillation

and hemodynamically significant carotid stenosis.

Afib - afib w/ valvular heart disease increases yearly stroke risk 17x, anticoagulation w/ warfarin (INR between 2 to3). Afib w/o valvular disease and no other risk factors can be placed on aspirin 325 mg/day. Prosthetic valves require long-term anticoagulation. Bacterial endocarditis patients should not receive warfarin because risk of cerebral hemorrhage from septic emboli.

Carotid Stenosis -If stenosis < 60% then treat w/ anti-platelet agents, if stenosis >70% can do carotid endarterectomy to reduce risk. For patients w/ moderate risk (60-70% stenosis) decide on case by case basis.