

STROKE CASES

CASE 1

62 year old woman presents to the emergency department with a complaint of left eye blindness that started abruptly 30 minutes ago while shopping. She denies headache, weakness, dizziness, slurred speech, or other associated symptoms. She has never had anything like this before.

PMHx: Type 2 DM, well-controlled (recent HbA1c 6.5)
HTN; hyperlipidemia (LDL 123 @ visit 2 weeks ago)
Hysterectomy ~ 20 yrs ago
(was on estrogen for ~ 5 years post-op but then stopped)

Meds: Metformin 1000 mg BID; lisinopril 5 mg QD; HCTZ 25 mg QD
NKDA

SocHx/Habits: Married x 35 yrs; middle school teacher; remote tobacco use, quit 20 yrs ago after 15-pack-year history; social alcohol use; no other drug use

Fam Hx: Mother had CVA at age 72, left her with residual left hemiparesis, resides in assisted living facility at age 83; father died in automobile accident at age 40; 2 siblings with HTN

What is the most likely cause of her symptom? What is the pathophysiology?

Transient ischemic attack; more specifically, amaurosis fugax; thrombosis or embolism to the retinal arterial supply

Her vital signs are T 98.6, P 82, BP 148/78, RR 12, O2 sat 98% on room air. By the time the ER doctor comes to see her (5 minutes after arrival), her symptoms have resolved. What is the differential diagnosis of her chief complaint & how would you rule out the other causes?

- Focal seizure- doubtful based on ocular sx alone, no prior hx of seizures
- Hypoglycemia- not a high risk with metformin rx; check STAT glucoscan on arrival
- Atypical migraine- no hx of migraines, no other aura sx
- Conversion disorder- no prior psychiatric hx or emotional stressors

What initial work-up would you suggest?

Labs to include: blood count, electrolytes, renal function, glucose, PT/PTT; could consider in certain situations: LFTs (?hepatic encephalopathy), ESR (looking for vasculitis), alcohol level, urine toxicology screen, urine/serum HCG

Chest X-ray

Electrocardiogram

Non-contrast CT of head

The ER physician calls you to evaluate the patient for admission. Her lab work was normal except for a glucose of 183. Chest x-ray & EKG are displayed below. Her head CT by report is “negative”. Does a normal CT make a stroke or TIA less likely? What is the main reason for initial imaging?

No. Ischemic damage to the brain may not be present on CT for up to 6-24 hours after the initial event. Most TIAs will not show any findings on CT. MRI is more sensitive in the acute setting, but not available or necessary in most cases (initial diagnosis should be made by history & physical and ruling out other causes). The main reason for CT is to rule out an intracranial bleed, which should be evident immediately.

After meeting the patient and revisiting her history, you move to physical exam. What specific areas do you want to evaluate?

- Funduscopic exam: ?????
- Peripheral pulses: equal, signs of PVOD
- Carotid exam for bruits
- Cardiac exam: regular, murmurs?
- Skin exam: embolic findings?, trauma?
- Neuro exam: level of consciousness, cranial nerves, visual fields, strength/tone, sensation, reflexes, gait

Listening to her neck, you hear a soft bruit over the left carotid artery. What is the significance of this finding?

Uncertain. Physical exam for carotid stenosis is difficult. Bruits have a poor specificity & sensitivity for significant stenosis. Turbulence may be from tortuous vessel or referred aortic stenosis murmur. Also, many patients with significant stenosis have no appreciable bruit. Still worth examining, but lack of bruit should not dissuade from further evaluation.

After completing your evaluation, the patient states that she would prefer to go home as she is feeling better now. Her husband asks you if she has had a stroke and if she is at risk for another event. What would you explain to the patient and her husband about her risk for stroke?

Based on the NASCET trial, the 1-year risk of stroke after a TIA could be as high as 20% (although in that evaluation, retinal TIAs were excluded). A study from JAMA in 2000 stated a 5% stroke risk within 2 days (This study has led to many ER doctors advocating for patient admission for evaluation). The risk for subsequent stroke, however, depends on the patient history, sx, and course. The best way to evaluate is with the ABCD score:

- A: Age \geq 60 1 point
- B: BP $>$ 140/90 1 point
- C: Clinical features
 - Unilateral weakness 2 points
 - Isolated speech deficit 1 point

- Other sx 0 points
- D: Duration
 - >60 min 2 points
 - 10-59 min 1 point
 - <10 min 0 points

A score of 3 or less indicates a low risk of early stroke (<1%), 4-5 indicates a moderate risk (1-12%), 6 indicates a high risk (~20%). Note that this is not a scoring system for TIA/Stroke diagnosis, just risk of another event. A patient could have a real ischemic event and a low score, but this not mean that the symptoms should be ignored or blamed on something else.

In this patient, her ABCD score is 3, which signifies a low risk of an early recurrent stroke. She may be appropriate for close outpatient follow-up rather than admission.

What is gained by admission for a TIA?

Immediate initiation of antithrombotic therapy.

Telemetry to look for evidence of paroxysmal atrial fibrillation.

Expedited further work-up of underlying problems (echocardiogram, carotid dopplers).

Close monitoring for evaluation of new neurologic sx, indicating recurrence.

In this patient's case, she seems likely to be able to follow instructions re: therapy and follow-up evaluation. Her risk of recurrent stroke is low, and she may be considered for discharge & outpatient evaluation.

What immediate treatment would you suggest?

Antithrombotic therapy for secondary prevention. I would suggest aspirin at a dose of 81 to 325 mg daily (studies do not indicate any difference between ASA doses). Furthermore, if the patient already takes aspirin, increasing the dose is not likely to have much more effect at preventing a recurrent stroke (including increases from 81 to 325 mg daily or 325 mg daily to twice daily). Aspirin with ER dipyridamole (AKA Aggrenox) or clopidogrel (Plavix) are other 1st line options, but I would suggest only for patients already on ASA (i.e. ASA-failure patients). Combination of ASA with clopidogrel or Aggrenox is not recommended.

Risk factor modification important as well. Consider increasing ACEI to improve BP control (goal <130/80) and addition of statin to get goal LDL <70. Consider modification of diet & medications to improve glucose control (goal A1C <6.0).

What follow-up tests would you suggest and why?

Transthoracic echocardiogram: Some don't advocate for this in all cases, and does not need to be repeated serially in cases of recurrent stroke TIA, but there are some helpful reasons to get it. First, you can get an evaluation of LV function (LV systolic dysfunction & low EF can be a risk factor for cardioembolic source) and looks for wall motion abnormalities that might suggest underlying vascular disease (CAD). Also, you may be able to evaluate for valvular abnormalities

(although if suspicion is high for SBE, need to get transesophageal view) and atheromatous plaques of the ascending aorta.

Bilateral carotid dopplers: Evaluate for underlying carotid stenosis.

Other labs: If not done as part of initial work-up, make sure you have checked lipid panel. Could also consider ANA, ESR, hypercoagulability studies in young patients to r/o inflammatory vasculitis or coagulopathy as a cause.

Your patient returns to the office in 3 days. She has been symptom-free and taking her aspirin as prescribed. She had a normal echocardiogram. Her carotid study revealed a 50-69% stenosis of the left internal carotid artery. How would you proceed?

This is a controversial subject. Based on the NASCET studies, it is agreed that 70-99% stenosis in a symptomatic patient should be treated with early carotid endarterectomy (i.e. within 2 weeks of the event if possible). This is with the assumption that the patient does not have other underlying disease that affects 5-year survival and that the surgeon/surgical center has a low rate of complications (<6%). Some argue that this should be extended to the finding of 50-69% stenosis, but studies have only shown a benefit in male, but not female, patients.

In this case, I would suggest a referral to a trusted vascular surgeon for discussion of risks & benefits of CEA. If the patient & surgeon agree that watchful waiting is best, I would continue ASA & risk factor modification (including aggressive lipid control with high dose statin therapy) with plan for repeat carotid studies in 4-6 months.

CASE 2

A 75 year old man with a history of atrial fibrillation is brought to the ER by ambulance. He was unresponsive to his wife this morning when she awoke at 6 AM. He went to bed without complaints at 11 PM last night and did not get up during the night. The EMS provider reports the patient has spontaneous eye opening, but is aphasic with a left facial droop and not moving his left side. He does not seem to be in any distress otherwise.

**PMHx: Hypertension, chronic atrial fibrillation
h/o peptic ulcer disease s/p upper GI bleed 3 years ago**

Meds: Warfarin 5 mg QD, atenolol 100 mg QD, multivitamin QD, Prilosec 20 mg QD

Allergies: PCN

SocHx: Married x 42 years, retired auto mechanic, smoked 1 PPD until quitting 5 years ago, 2-3 beers a week.

What is the most likely cause of his symptoms? What other possibilities are there?

Ischemic stroke, likely to the right middle cerebral artery.

Differential diagnosis includes a hemorrhagic stroke, seizure with post-ictal paralysis, metabolic encephalopathy (hypoglycemia, hyponatremia, hepatic), intoxication.

His vital signs are T 99.0, BP 172/90, HR 95 & irregular, RR 12, O2 sat 99% on 2L nasal cannula. His lab work is notable for normal chemistries & CBC, blood glucose is 123, INR is 4.1.

EKG reveals atrial fibrillation with a controlled ventricular response. Chest x-ray is unremarkable. A head CT is normal by report of the on-call radiologist.

It is now 8 AM. The family has asked the ER physician about giving a “clot-busting drug” for the patient’s stroke, but he wanted to call you, the admitting resident, first to discuss. Do you think this patient is a candidate for thrombolytic therapy?

No. The main issue in this case is the timing of the stroke. Thrombolytics are only approved for use in acute ischemic stroke when they can be given within 3 hours of symptom onset. In this case, the patient arrived in the ER & had the work-up done expeditiously, but the patient was last seen as normal at 11 PM last night, 9 hours ago. The clock for thrombolysis starts at that point, not at the time of recognition of symptoms.

In addition, the patient has another contraindication to thrombolytics: a coagulopathy with an INR of 4.1. An INR of >1.7 was a cause for exclusion from the NINDS study.

The prior history of GI bleed is not a CI (only recent GI bleed within 21 days) nor is the hypertension, unless severe (>185/110) and not able to be improved with IV antihypertensives.

You come to the ER to see the patient. He opens his eyes to your voice, but does not follow any commands or speak. The nurse notes that he did wince with pain and pull back his hand when an IV was placed on his right side. His exam is notable for normal pupillary responses, intact extraocular movements, and intact gag reflex. He has no movement in his left arm or leg, with flaccid paralysis and 3+ reflexes at the biceps, triceps, brachioradialis, knee, and ankle. Toes are downgoing on the left & upgoing on the right. Strength, sensation, and tone seem to be intact on the right side, although the patient is somewhat difficult to examine because of his apraxia.

What is his Glasgow Coma Score (GCS)?

8 points. 3 points for eye opening to voice, 1 point for lack of verbal response, 4 points for withdrawal to pain.

What admitting orders would you consider for this patient?

Admit to telemetry.

Routine vital signs with regular neuro checks to observe for improving or declining status.

Activity: Bedrest initially with advancement as per PT/OT evals & neuro changes over next 24 hours.

Diet: NPO until swallow evaluation to rule out dysphagia. In some cases, a bedside nursing evaluation can be done on admit to allow for oral intake.

INT or consider maintenance IV fluids if NPO.

Meds: DVT prophylaxis (see below). ASA 81-325 mg daily unless contraindication. Consider IV metoprolol for atrial fibrillation (only if pt has rapid ventricular response, see BP discussion below).

Should this patient receive an unfractionated heparin drip?

I don't think so. Heparin drips have been an area of controversy in acute stroke management. Theoretically, they make sense; if a CVA is a "brain attack" from a thrombus-related blockage of an artery, then treating that coagulation problem with heparin should make sense in the same way that it is used for an acute coronary syndrome. The problem is that brain cells are not the same as cardiac muscle cells.

Studies have looked at this issue and the results have shown that IV heparin (and the effects have been assumed for LMWH) may reduce the short-term risk of recurrent ischemic stroke (within 2 weeks), but that comes with the increased risk of conversion to a hemorrhagic stroke. Overall morbidity and mortality are unchanged. Therefore, IV heparin has been suggested (although not clinically proven) for only a few specific causes of stroke:

- Arterial dissection, particularly vertebral dissection with posterior cerebral infarcts
- Proven cardioembolic source, i.e. intra-atrial clot with atrial fibrillation or severe LV dysfunction due to AMI with a ventricular clot in the affected area
- Controversial: "stroke-in-evolution"- repeated, stuttering symptoms to suggest a showering of emboli from an unstable "up-stream" source. No proven utility and difficult to diagnose (as stroke sx can change particularly over the 1st 24 hours)

Particularly in atrial fibrillation, it is not recommended to treat with IV heparin in the acute phase (unless there is a proven atrial clot on echo), because of the concern for intracerebral hemorrhage. Aspirin is the proven therapy, with a plan to restart warfarin once the patient is stable (usually in 1-2 weeks or at the time of discharge).

Despite the lack of evidence for IV heparin or standard-dose LMWH, there is strong evidence for antithrombotic therapy with aspirin (or clopidogrel in ASA-allergic patients) in the acute phase (within 48 hours, usually on admission). Studies have shown decreased mortality and recurrent stroke within the 2-4 week time period, with only a slight increase in hemorrhage risk.

This patient was on appropriate stroke prophylaxis (warfarin) for his chronic atrial fibrillation. Why did he have a stroke?

Atrial fibrillation is a strong risk factor for ischemic stroke, and even with appropriate prophylactic therapy, the annual risk is not zero. In fact, the average annual stroke risk associated with chronic atrial fibrillation is about 5% (may be higher or lower based on risk factors, including prior TIA/CVA, age, HTN, and CHF), and that risk can be lowered to less than 1% with appropriate therapy (but not to zero). For most patients, appropriate therapy is warfarin titrated to an INR of 2-3. In selected patients, under 75 years old without associated hypertension, CHF, or prior CVA/TIA, aspirin monotherapy can be considered because the baseline annual stroke risk is <1% anyway.

In this patient's case, his annual risk for recurrent stroke is >10% after his first episode, and I would certainly resume warfarin therapy once the stroke symptoms have stabilized and the patient is able to take oral medications.

What is the most appropriate choice for DVT prophylaxis?

Stroke patients have a high risk of venous thromboembolism because of immobility and, in some cases, hypercoagulability. DVT prophylaxis is an important component of care in the subacute phase of the illness. The best choice is some form of subcutaneous anticoagulant: unfractionated heparin 2-3 times daily, low molecular weight heparin (enoxaparin once daily), or fondaparinux. TED stockings or sequential compression devices (SCDs) may be used in addition, but should only be used as monotherapy in patients with active bleeding or at very high risk for bleeding.

What should you do for the patient's elevated INR?

In the setting of an elevated INR without active bleeding, I would elect to hold the patient's warfarin. While vitamin K and/or fresh frozen plasma (FFP) could be used to reverse the coagulopathy, I would have some concern about worsening a hypercoagulable state in the setting of the acute stroke. I would expect the INR to trend down over the course of 3-5 days, and would likely resume the warfarin therapy prior to discharge.

The nurse calls 2 hours after admission because the patient's blood pressure is 190/100. She is concerned that the patient will have another stroke. What should you do?

Provide reassurance to the nurse & continue close monitoring. Severe hypertension is common in acute ischemic stroke, due to autoregulation in the brain (the blood flow in dilated, post-stroke intracranial vessels is BP-dependent). Lowering blood pressure in the acute phase is actually associated with increased morbidity & mortality. You should avoid treating blood pressures in the acute phase of stroke except in these situations:

- Critically severe HTN (>220/120)
- Associated acute coronary syndrome, acute heart failure, or aortic dissection
- Pts who have received thrombolysis (goal BP <180/105)

This does not apply to hemorrhagic stroke, where BP control is more warranted.

If the patient's BP rose to >220/120, I would use intermittent doses of labetalol (10-20 mg IVP, if using frequently can switch to IV drip). Hydralazine, nitroglycerin, & nitroprusside are other choices. Avoid sublingual nifedipine (used to be a frequent choice in acute severe hypertension) because the rapid drop in BP can precipitate worsening stroke symptoms.