

Patient: [REDACTED]

Date of Admission: 11/28/06

CC: Increased weakness and slurred speech

HPI:

The patient is a 61 yo gentleman with h/o CVA in September 2005, hypertension, hyperlipidemia, type 2 diabetes, seizure d/o, and cocaine abuse who presents with increased global weakness, slurred speech, and altered mental status. At approximately 4:00 AM, the patient was found by wife sitting on the couch, unable to stand up, respond appropriately to questioning, or speak clearly. The room was in disarray, possibly indicating a fall of some kind, and there was also evidence of urinary incontinence with a wet area on the couch. On presentation to the ED, the patient was found to have right upper and lower extremity weakness, slurred speech, and disorientation. The patient was unable to explain most of the night's events but did state that he fell on his buttocks. He also thought he was speaking funny despite knowing what he wanted to say. Although patient did have some residual right sided weakness from the previous stroke that requires the use of a cane, wife noted that this episode was markedly worse in terms of lower extremity strength and ambulation. Patient endorses significant cocaine use and noncompliance with medications since May 2006. When seen in ED at 11:30 AM, patient at baseline per wife. Patient denies head trauma, headache, change in vision, nausea, and vomiting. No dizziness, SOB, chest pain, or palpitations. Patient denied urinary incontinence despite evidence described above.

PMHx:

- CVA: September 2005
 - MRI w/ and w/o contrast: acute lacunar strokes in anterior basal ganglia and internal capsule on left and in the left central pons; nonspecific small area of enhancement in right basal ganglia possibly from subacute strokes; old encephalomalacic changes in both frontal lobes.
 - Bilateral carotid u/s: negative study – no atherosclerotic plaque seen in either bifurcation region
 - On d/c, started on baby aspirin x 2 per day; discontinued by patient
- HTN: Long standing h/o uncontrolled hypertension, currently untreated
- Hyperlipidemia: last fasting lipid panel in 1/06; total cholesterol at 178, triglycerides at 127, HDL at 42, LDL at 111; now untreated due to medication noncompliance. Current guidelines of ATP III place goal LDL at <100 with DM as CHD equivalent risk factor and optimally less than 70.
- DM type 2: diet controlled. Last HbA1c in 1/06 at 5.6. No known neuropathy/retinopathy/nephropathy. No previous labs seen for urinary microalbumin, last ophthalmology appt “a while ago”.
- Seizure d/o:
 - Diagnosed 2001 as tonic-clonic seizure with postictal state lasting approx 45 min. Suspected posttraumatic etiology, although cocaine abuse could have contributed.
 - Last seizure per wife approx 3 years ago. Currently not on antiepileptic medications.

- Previous EEG done at initial presentation of seizure event demonstrated some right frontotemporal slowing and spiked discharges.
- H/o MVA as child with subsequent frontal lobe injury

PSHx: None

Previous Medications (none currently – last taken prior to 5/06, due to lack of finances)

- ASA 162 mg po qday (2 baby aspirin)
- HCTZ 25 mg po qday
- KCl 20 mEq po qday
- Lotrel (amlodipine/benazepril) 10/20 mg po qday
- Zocor (simvastatin) 40 mg po qHS
- Colace (docusate) 100 mg po BID prn constipation
- No herbals, OTCs

Allergies: NKDA

FHx:

- Mother: alive and well
- Father died in late 60s of cancer, unknown type
- Otherwise FHx unknown, no relationship with rest of family

SHx:

The patient currently lives with wife and grandchild in own home in [REDACTED], NC. They have 4 children but do not have much contact with them. He has previously had some medical insurance issues with discontinuation of Medicaid and could not afford to see his regular physician or go to physical therapy for previous CVA rehab. He is now in process of applying for disability, having quit his job as a [REDACTED]. Because of the lack of finances and motivation, the patient has not been taking his prescribed medications since May 2006. Educational level is at 10 years of schooling, with no GED. ADLs are limited. Wife makes meals and bathes patient, but he can still do the essential tasks such as feeding himself and brushing his teeth. The patient walks with a cane to compensate for right sided residual weakness. He smokes a half pack per day, but denies alcohol use. He also has a significant cocaine habit, smoking approximately once per week for at least 2 years, although per wife has been using for much longer.

ROS:

- Constitutional: some generalized fatigue; denies fevers, chills, lightheadedness, change in appetite, or wt change
- HEENT: denies vertigo, rhinorrhea, congestion, sore throat, hearing loss, diplopia
- CV: denies racing heart, orthopnea, PND, edema
- Respiratory: denies wheezing, cough, hemoptysis
- GI: denies abdominal pain, constipation, diarrhea, hematochezia, melena, change in frequency or consistency
- Urinary: denies dysuria/polyuria, hematuria, hesitancy, urgency
- GU: denies penile discharge, sores, testicular pain, hernias

- MSK: right hand stiffness; denies swelling, tenderness in joints
 - Neurologic: unsteadiness in gait; denies numbness, tingling
 - Skin: denies rashes, bruises
 - Psych: denies depressed or anxious mood, change in mood
- Otherwise as indicated in HPI

PE:

- Vitals:
 - T = 97.0 (oral)
 - P = 90
 - RR = 18
 - % sat = 96% (RA)
 - BP = 197/104
- General: Patient is an overweight man in NAD, lying comfortably in bed.
- Eyes: PERRL. EOMI. Sclera clear. Mild right sided ptosis.
- ENT: Nares without any discharge. Mucous membranes moist. Oropharynx without erythema or exudate. Cerumen obstructing view of TMs bilaterally.
- Neck: Neck supple, trachea midline. No thyromegaly. No carotid bruits. Pulses 2+ bilaterally.
- Lymph Nodes: No lymphadenopathy (cervical, axillary, and inguinal)
- Cardiovascular: RRR, Normal S1, S2; no murmurs, rubs, or gallops; no prominent neck veins or JVD noted. 2+ pulses bilaterally (radial, femoral, dorsalis pedis)
- Lungs: Chest wall motion symmetric with no accessory muscle use. Resonant to percussion. CTA bilaterally. No wheezes, rhonchi heard.
- Skin: Warm to touch; no rashes/scars; dry skin noted on lower legs bilaterally.
- Abdomen: Soft, nontender, nondistended with active bowel sounds. Liver width at 10 cm to percussion along midclavicular line, nonpalpable. Rectal exam deferred due to lack of GI or prostatic associated complaints, no s/sx of overt or occult bleeding, no complaints of numbness/tingling/paresthesias. Will perform if heparin gtt needed.
- Extremities: No cyanosis, clubbing, or edema.
- Musculoskeletal: Normal ROM throughout. Contractures of right hand, with resistance to manual spreading/straightening of fingers. No joint swelling or tenderness noted.
- Neurological:
 - Alert and oriented to person, place ("hospital", not CMC), time (11/28/2006), and situation ("possible stroke").
 - Slowed mentation and responded with limited speech.
 - No dysarthria, no dysphasia.
 - CN II-XII grossly intact. Face symmetric with no mouth droop.
 - 4/5 strength in right deltoid, biceps, triceps, hip flexor, quadriceps, anterior tibialis, gastrocnemius. 5/5 strength in left. Grip strength equal bilaterally. Normal tone and bulk bilaterally.
 - Cerebellar function with finger-to-nose and shin-heel testing normal. Mild right side intention tremor. Rapid alternating movements minimally slowed.
 - Sensation to light touch slightly decreased bilaterally on ankles.
 - Gait with mild right foot drag. Negative Romberg.

- Right brachioradialis, biceps, triceps, patellar DTRs at 3+, right Achilles at 2+, left DTRs at 2+ throughout. Upgoing Babinski on right, equivocal on left.

Diagnostic tests:

- CBC: WBC = 3.8, Hgb = 13.6, Hct = 39.0, plts = 218, MCV = 89.1; differential: N = 57.3, L = 31.8, M = 6.8, E = 2.8, B = 1.3
- CMP: Na = 141, **K = 3.2**, Cl = 109, bicarb = 28, BUN = 13, Cr = 1.1, gluc = 91, Ca = 8.4, alb = 3.2, total pro = 6.0, bili = 0.9, alk phos = 34, AST = 12, ALT = 14
- UA: glucose = neg, bile = neg, ketone = trace, Hgb = neg, **alb = trace, urobil = 1.0**, nitrite = neg, leukocyte = neg
- Umicro: WBC = 0-1, RBC = 3.5, bacteria = rare, **mucus = present**, squamous epith = rare, hyaline cast = 1-2
- **Utox: negative**

Imaging: preformed in the ED, prior to assessment

- CT head w/o contrast:
 - Old anterior right frontal lobe tissue loss (9/05)
 - Bilateral basal ganglia with multiple infarcts; left sided infarcts appear old (9/05), **right side infarct of indeterminate age**
 - No hemorrhage or mass lesion
- EKG: Sinus bradycardia with rate of 59. Normal intervals (PR = 0.14, QRS = 0.08 Qtc = 0.450) and normal axis. No ventricular hypertrophy or atrial enlargement. No signs of ischemia/infarct. No change from previous EKG (9/05).

Problem List:

- Dysarthria, right sided weakness, AMS, possible incontinence
- CT of head with right side infarct of indeterminate age
- Low K at 3.2
- HTN, uncontrolled
- Hyperlipidemia
- DM type 2
- Cocaine, marijuana, and tobacco abuse
- Residual right sided weakness requiring the use of a cane
- Financial situation limiting ability to attain medications, med noncompliance

Assessment:

This is a 61 yo gentleman with h/o CVA, HTN, hyperlipidemia, seizure d/o, cocaine abuse, and medication noncompliance who presents with increasing right sided weakness and dysarthria of unknown duration less than one day, concerning for TIA versus CVA. Given the patient's history of previous stroke and well as noncompliance with medications, either of these would be most likely. Although less likely, other possibilities to explain this presentation include seizure, hypertensive urgency versus emergency, or intoxication versus withdrawal of drugs. With history of seizures, previous brain injury (CVA), and chronic cocaine abuse, recurrence of seizure could explain his presentation. However, his wife reports previous seizure episodes were

unlike these recent symptoms, and his last seizure was approx 3 years ago. The patient also has a history of uncontrolled hypertension and noncompliance with medications. His significantly elevated BP probably is chronically high but could be elevated due to acutely increased intracranial pressure. He has no complaints of headache, changes in vision, or nausea/vomiting. Finally, although his drug usage probably contributed to these recent symptoms through mechanisms of cerebral ischemia via vasoconstriction, the presentation would be very atypical for acute intoxication or withdrawal.

Plan:

1. Weakness, dysarthria: CT head shows infarct of indeterminate age, which radiologist indicated could be old and correspond with prior MRI findings. TIA more likely given resolving symptoms over short time frame of less than 24 hours.
 - a. Admit to telemetry/9A.
 - b. Will obtain MRI/MRA to assess acuity of brain infarcts. Will obtain TTE to assess wall motion abnormalities in heart, given that patient has never had one in the past. Mural and valvular thrombi better visualized with TEE, but more invasive test.
 - c. Will restart ASA at 325 mg po qday, given patient has technically not failed aspirin therapy due to medication noncompliance.
 - d. Neuro checks q2hr on floor.
 - e. NPO until swallow evaluation.
 - f. PT/OT consults to assess rehab needs, cane usage, and work with residual weakness.
2. HTN: Long history of poorly controlled hypertension.
 - a. Will restart prescribed home medications: HCTZ 25 mg po qday and Lotrel 10/20 mg po qday. Currently beta blocker contraindicated due to recurrent cocaine use.
 - b. Add Clonidine 0.1 mg po q4hr prn SBP > 180 for increased coverage.
3. Hyperlipidemia: Currently not taking Zocor for elevated lipids. Previously not at goal of LDL < 100 as indicated in PMHx.
 - a. Will order fasting lipid panel to assess status of hyperlipidemia.
 - b. Will restart Zocor 40 mg po qHS.
4. DM: previously diet controlled. Begin ADA diet and cover with Novolog SSI as needed (give 2 Units for every 50 above glucose of 100) Will order HgbA1C to assess status of recent glycemic control.
5. FEN/GI: Patient given KCl 40 mg po x 1 in ED for hypokalemia. Restarted KCl 20 mEq po qday. Will recheck BMP in AM. Started 1800 kcal ADA diet. Restarted previous home bowel regimen – Colace 100 mg po BID prn constipation.
6. Prophylaxis: Will begin Lovenox 40 mg sc qday for DVT prophylaxis. Added Nitroglycerin 0.4 mg sublingual q5 min prn chest pain (may repeat x 2).
7. Dispo: Full code. Will consult social work to assist with Medicaid. Patient will return home with wife when medically cleared.

Discussion:

The classic definition of a transient ischemic attack (TIA) is a clinical diagnosis characterized by the sudden onset of temporary, focal neurological symptoms lasting for less than 24 hours, consistent with how this patient presented. However, most TIAs have a much shorter duration of less than 1 hour.^{1,2} Pathophysiologic mechanisms of TIAs include large artery low-flow TIA, embolic TIA, or lacunar or small penetrating vessel TIA.³ A large artery low-flow TIA is brief and recurrent, associated with a stenotic atherosclerotic lesion at the internal carotid artery origin, an atherosclerotic stenosis in middle cerebral artery stem, or an atherosclerotic lesion in the intracranial portion of the internal carotid artery when collateral flow is impaired. An embolic TIA is most often a discrete event, usually more prolonged with focal neurological deficits. The embolus could arise from an artery (most often extracranial) or from the heart (mural thrombus). Determination of source is important given that recurrent emboli would not be rare, increasing risk of more devastating ischemic damage. A lacunar or small vessel TIA is due to transient cerebral ischemia within the intracerebral penetrating vessels stemming from the middle cerebral artery, the basilar artery, or the vertebral artery or within the circle of Willis.³ Patients presenting with TIAs are at significantly increased risk of stroke during the next 5 years without adequate risk reduction therapy with an antiplatelet agent, one possibility of which is aspirin, initially given to this patient on previous stroke presentation.¹ Regrettably, this patient was noncompliant with this recommendation.

Unfortunately, this classic definition of a TIA is inadequate. It implies that the injury is only temporary, resolved by restoration of blood flow to the ischemic area. However, even after an hour of ischemia, the brain can be permanently damaged.

More recently, the definition of a TIA is being challenged by findings in diffusion weighted neuroimaging (DWI). Diffusion weighted imaging has been shown to have a good sensitivity (small false negative rate, especially during very acute phase of TIA/stroke) and a higher specificity (very low rate of false positives) in the diagnosis of ischemic stroke.⁴ This type of imaging has proved to be better than conventional imaging to detect presence of infarction. In patients with clinically diagnosed TIAs, the incidence of ischemic lesions found in CT scans and conventional MR studies was anywhere from 12% to 48%. In comparison, DWI scans detected acute changes in anywhere from 35% to 66% of patients.¹ Therefore, MR imaging using diffusion weighted images reveals ischemic lesions in approximately half of all clinically diagnosed TIAs.⁵ In addition, DWIs can distinguish acute from chronic lesions as well as pick up small infarcts associated with recent TIAs. This imaging can define the size, number, and location of the lesion (and the vascular territory involved), perhaps guiding therapy and helping clarify prognostic factors.¹ In the 2004 article published in *Stroke*, researchers found that even within 6 hours after presentation of symptoms, TIA and stroke could be differentiated via DWI by analyzing signal intensity of lesions.⁶ In addition, a study recently published in 2006 in the *American Journal of Neuroradiology* found that most patients with positive DWI TIAs (especially those with markedly decreased apparent diffusion coefficients and large volume changes) have lesions on MRI that reflect permanent tissue injury similar to stroke patients.⁵ Given this advancement in technology, a new definition for TIA has been proposed by the TIA Working Group – “a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms lasting < 1 hour and without evidence of acute infarction.”⁵ Therefore if the subsequent MRI study showed an acute infarct (as it eventually did in this patient), the new findings would indicate a CVA rather than a TIA, contradicting the classical definition of TIA.

References:

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