Case Study 1

Mr. James Brown*, a 68-year-old retired plumber, came to the emergency room on a Sunday afternoon. He had been at church in the morning and while looking at the choir had noticed trouble with his vision. Over the course of seconds, it appeared that curtains were coming down on the choir and he could only see their waists, legs, and feet. This lasted for about 10 minutes or so and then the curtains rose and he could see the choir in its entirety again. After church, he and his wife went out for lunch and another episode occurred while he was reading the menu. He could see only the bottom half of the menu. He covered his right eye and the problem did not resolve. Then he covered his left eye and he could see the entire menu. He also noticed that the words on the menu were not making sense to him and that his right face and his right hand felt numb and dead as though he was feeling the menu through thick cotton gloves. His wife saw the alarm in his face and asked him what was wrong, but he could not say anything. When the symptoms persisted for 15 minutes, she took him to the emergency room nearby. By the time they got there, his symptoms were gone, having lasted about 35 minutes altogether. He could see normally, his speech was normal, and his sensation was normal.

(*not an actual patient)

1. What happened to Mr. Brown?
   a. He had one TIA and one completed stroke
   b. He had 2 TIA’s
   c. He had 2 seizures, one with speech arrest.
   d. He had 2 panic attacks

2. What would you expect to find on his general physical examination?
   a. Poor pulses in the right upper extremity
   b. A right carotid bruit
   c. A Hollenhorst plaques in a retinal artery branch of the left eye
   d. A blood pressure of 138/72

3. Now that Mr. Brown is feeling better, what should the ER physician do?
   a. Tell him to take an aspirin, go home, and make an appointment for follow-up with his primary care physician within the next 2 weeks.
   b. Have him admitted to the hospital for further testing to determine the cause of his symptoms.
   c. Give him TPA right away
   d. Get an EEG and see if he is having non-convulsive status epilepticus.

4. The test that is most likely to show the cause of Mr. Brown’s symptoms is:
   a. An MRI of the brain
   b. Carotid Doppler/Ultrasound
   c. A Holter monitor to check for atrial fibrillation
   d. An Electroencephalogram

5. Other information that might shed light on the likely diagnosis is the fact that
   a. Mr. Brown’s mother died from a stroke at the age of 86
   b. Mr. Brown’s HDL cholesterol is 60
   c. Mr. Brown quit smoking 12 years ago
   d. Mr. Brown’s last blood pressure was 164/98
1. Mr. Brown had 2 TIA’s referable to the left internal carotid artery. In both TIA’s he had an ocular phenomenon called “transient monocular blindness” or “amaurosis fugax”. This occurs when small fragments of atherosclerotic plaque from a diseased internal carotid artery break off and enter the retinal artery through the ophthalmic artery (the first branch off of the internal carotid artery). The left eye was affected (when he closed it, he could see normally out of the right eye). This means that the left internal carotid artery was affected. With his second TIA, Mr. Brown also had symptoms referable to the left hemisphere. He likely had ischemia in the middle cerebral artery territory (which is supplied by the internal carotid artery) causing face and hand numbness briefly.

2. “Hollenhorst plaques” are the small atherosclerotic debris that might be seen in the retinal arteries of patients with significant carotid disease. These are usually shiny and golden and lodge in the bifurcations of small retinal branches. They may be asymptomatic if there is good retinal collateral blood flow. A bruit may or may not be present with significant carotid stenosis. In the case of Mr. Brown, since his LEFT carotid was symptomatic, a LEFT carotid bruit would be a more significant finding. Usually during and acute stroke or TIA, the blood pressure may be very high.

3. Mr. Brown should be admitted to the hospital for further testing. The studies should be focused at identifying his risk for subsequent TIA and stroke and observing him for early recurrence of stroke. TPA can be given to patients whose stroke symptoms are not rapidly improving within the first three hours of onset. Although a TIA is defined as an ischemic stroke event that resolves in less than 24 hours, most TIA’s do not last much longer than 30 minutes. It is wise to have a patient with an ischemic TIA or stroke take an aspirin (if they are not a TPA candidate) while you are trying to figure out what is going on. Aspirin has been shown to acutely reduce the mortality associated with acute stroke.

4. Carotid Dopplers can quickly identify significant stenosis that warrants carotid endarterectomy. An MRI of the brain may be normal in a patient with a TIA. It is still useful, however, in ruling out prior “silent” strokes or other lesions that might complicate the management of this patient. The patient has had 2 TIA’s in the same arterial distribution. Usually embolic events due to entities such as atrial fibrillation occur in different arterial territories. It is particularly rare to see ocular TIA’s occur as a result of atrial fibrillation. Cardiac monitoring in this case would be unlikely to add significant useful information.

5. Treating hypertension is the single most effective means of reducing the risk of ischemic stroke in all populations. Stage II hypertension (systolic BP >160) such as Mr. Brown has is associated with a 6 to 8 fold risk of stroke compared to the general population. Having a first-degree relative with an early stroke (occurring at an age of 55 or younger) is associated with an increased risk of stroke. Remote smoking (quitting for more than 10 years) results in a risk for disease states that is very similar to that of someone who has never smoked. HDL cholesterol of 60 or greater is considered to be protective against atherosclerotic disease, a so-called “negative risk factor.”

Case Study 2
Ms. Ann Smith* a 63-year-old schoolteacher, presented to the emergency room after several days of episodes of numbness of the left leg and the left arm. The episodes would last for about 20 minutes and then get better. She had never experienced anything like this before. She was not dizzy, she did not have difficulty with her speech or vision or swallowing, she did not experience unsteadiness in her walking. She had been a diabetic for the past five years and last year, she was started on an oral hypoglycemic agent because her diet was not fully effective in controlling her blood glucose. She was also hypertensive, but in the last year had had improved control of her blood pressure with most readings in the 140’s over 70’s. She was also taking a “statin” to improve her cholesterol which was 230 the last time it was checked. Finally, she has been taking aspirin 81 mg daily for the past 3 years. She came to the emergency department because this last episode had not gotten better for more than 7 hours. When she was examined, she was found to have normal speech and memory, normal cranial nerves except she had reduced sensation to pin prick and cool temperature on her entire left face. She had full strength in the extremities. There was reduced appreciation of the pin and cool temperature over the left side of her neck, her left shoulder, down her left arm, her left chest, left abdomen and over the entire left leg. Vibration sensation was normal on both sides. Reflexes were slightly brisker on the left. Plantar responses were neutral (Babinski sign absent).

(*not an actual patient)

Questions:
1. What happened to Ms. Smith?
   a. Ms. Smith is malingering so that she can get disability and take early retirement from teaching “those little horrors.” It is impossible to have numbness on just one side of the body with an otherwise nearly normal examination.
   b. Ms. Smith has had a pure sensory stroke due to small vessel occlusion of thalamic penetrators off of the right posterior cerebral artery.
   c. Ms. Smith has had a pure sensory stroke due to ischemia in the territory of the right thalamic penetrators as a result of severe basilar artery stenosis (narrowing) with posterior cerebral artery hypoperfusion.
   d. Ms. Smith is having a migraine variant and sooner or later she is going to experience numbness associated with a profound headache.

2. The diagnosis can be confirmed by:
   a. An MRI of the brain showing a small infarct in the right thalamus
   b. A CT scan of the brain showing a large infarct in the right parietal lobe
   c. A Carotid Doppler study showing a right internal carotid with >70% stenosis
   d. An MRA showing a tight stenosis of the basilar artery

3. Ms. Smith is at higher risk for stroke because of
   a. Diabetes
   b. Hypertension
   c. Hyperlipidemia
   d. All of the above

4. Ms. Smith’s doctor should
   a. Have her continue taking her aspirin and warn her not to miss any doses.
   b. Change her aspirin to ticlopidine (Ticlid)
   c. Add clopidogrel (Plavix) to Aspirin
   d. Change her aspirin to aspirin 25mg/extended release dipyridamole200mg (Aggrenox) twice a day.
1. A pure sensory stroke occurs most frequently as a result of small vessel ischemia (a “lacunar stroke”) in the thalamus. This type of stroke tends to “stutter” along with waxing and waning symptoms before the stroke is “completed.” Because only the small penetrating arteries are involved, the syndrome is very focal and other “neighborhood signs” such as vertigo, diplopia, slurred speech, or difficulty swallowing, or a visual field deficit as might be seen with basilar stenosis with or without PCA involvement (PCA involvement would cause a field deficit) is not expected. Acephalgic (painless) migraines are occasionally seen as stroke mimickers, but usually, the patient has a strong prior history of more typical migraines.

2. An MRI (especially with diffusion weighted imaging early in the course) will usually show a distinct lesion in the thalamus. These strokes almost always cause significant sensory loss almost “splitting the midline” from head to foot on one side of the body. A parietal lobe stroke will often be associated with not only reduced primary sensation, but also with cortical sensory loss such as agraphesthesia or astereognosis or extinguishing of double simultaneous stimuli. A parietal stroke large enough to cause face, arm, and leg involvement would also cause associated weakness as a result of the proximity of the sensory cortex to the motor cortex.

3. Diabetes, hyperlipidemia, and particularly hypertension have all been shown to be associated with higher rates of stroke. Patients with diabetes show an increased rate of small vessel strokes compared to strokes due to other mechanisms.

4. Most physicians would assume that aspirin has not had an adequate protective effect in reducing Ms. Brown’s likelihood of a stroke and now that she has had a stroke, her risk for another stroke is even higher. Something “stronger” than aspirin is warranted. Ticlopidine is about as effective as Aggrenox in preventing stroke recurrence, but is associated with a significant risk for bone marrow suppression, thrombocytopenia, and liver dysfunction. It is generally a last choice in recurrent stroke prevention. There is no data to support the addition of clopidogrel to aspirin to prevent strokes at this time. A corollary study (adding aspirin to clopidogrel) showed no significant benefit and a greater chance of bleeding. Of the choices given, adding Aggrenox would be the most reasonable next step.