Woman with Mental Status Change

Vanita Treat MD, PGY-3

An 83 year-old African-American female with a history of hypertension, frequent urinary tract infections, atrial fibrillation, and a cerebrovascular accident (CVA) on Coumadin presented two days after a fall. A family member stated she was walking normally and tripped on uneven concrete, hitting her chin on a fence. Two days after the incident, the patient was found at home slumped over in a chair with blood oozing from her chin wound. The patient was brought to the emergency department where she was lethargic but responsive. The patient denied any chest pain, shortness of breath, lightheadedness, dizziness, blurry vision, weakness or incontinence. Her speech was slightly slurred. The patient also denied nausea, vomiting, headache or fevers. She did report some pain over her jaw. There was no other significant past medical, surgical, or family history. Her home medications included 3mg of Coumadin and 12.5mg of Toprol daily. She had no known drug allergies.

On physical exam, the patient was found to have a fever of 102.7 F, a heart rate of 68 beats per minute, a blood pressure of 115/67 mm Hg, a respiratory rate of 16 breaths per minute and an oxygen saturation of 99% on 2 liters of oxygen. Her blood sugar was 26 mg/dL on admission and she was given intravenous (IV) dextrose. She was lethargic but arousable and responded to both verbal and tactile stimuli. Orientation was difficult to assess, but the patient was not at her baseline as per her family. Her extraocular movements were intact and her pupils were equal round and reactive to light. She had an area of ecchymosis and an abrasion over her chin. Her heart rate was irregularly irregular. There were no murmurs, rubs or gallops. Her lung exam was clear bilaterally. Her abdomen was soft, nontender and nondistended. She had normal active bowel sounds and was heme negative rectally. She had no edema in her extremities. Her neurological exam was difficult to assess secondary to lack of cooperation. She did have spontaneous movements in all four extremities.

Laboratory data on admission showed a white blood cell count of 4k/L (4-11), a hemoglobin of 12.4 g/dL (12.5-15), and a platelet count of 146k/L (140-400). She had a sodium of 135 mmol/L (135-146), potassium of 4.9 mmol/L (3.5-5.0), chloride of 100 mmol/L (98-109),

bicarbonate of 28 mmol/L (24-32), BUN of 8 mg/dL (12-27), creatinine of 0.9 mg/dL (0.7-1.4), and a glucose of 56 mg/dL (60-110). Her calcium was normal. Her PTT was 30 sec (22-36), her PT was 24.2 sec (13.4-15.8) and her INR was therapeutic at 2.03. In the emergency room, she had a head CT which showed no focal abnormalities and no signs of hemorrhage. Her urine drug screen was negative and a urinalysis showed no evidence of infection. Her troponins were negative times three. Blood and urine cultures were sent. She had a CXR which showed no active disease. An EKG on admission showed atrial fibrillation at a rate of 93 beats per minute. A TSH was 0.91 uIU/ml (0.4-4.8). B12 and folate levels were normal as well.

The patient started to become more confused and so a lumbar puncture was performed. The CSF findings were unremarkable. The patient defervesced and all cultures were normal. Neurology recommended an EEG which was consistent with multi-infarct dementia and metabolic encephalopathy. An MRI of the brain showed no acute infarcts or hemorrhage. There was evidence of old ischemia and a slightly enlarged pituitary gland.

Her sodium had dropped significantly during her hospital stay to a low of 119 mmol/L and her blood pressure dropped to a systolic range of 80-90mmHg. At that time, a cortisol level was checked and was found to be 1.3 mcg/dL. A repeat TSH was 0.68 uIU/mL. She had a free T4 of 0.5 ng/dL and a total T3 of 69 ng/dL, both of which were low. An endocrine consult was obtained and a dedicated pituitary MRI was recommended as well as IV steroids (hydrocortisone 100gms IV every 8 hours was used). The patient was also started on 25mcg of Synthroid daily. Her hyponatremia improved dramatically as did her mental status. An ACTH level was low at 8 pg/mL (9-52) and a somatomedin-C level was low at 19 ng/mL (71-290). A prolactin level was normal at 19 ng/mL (0-19) and an IGF-1 was ordered but not completed. The dedicated pituitary MRI showed diffuse hemorrhage involving the pituitary gland with a probable underlying macroadenoma. It was presumed that the patient had pituitary apoplexy.

Discussion

Pituitary apoplexy is a rare but serious condition caused by sudden hemorrhage or infarction of the pituitary gland. Typically, there is an underlying pituitary adenoma. A classic presentation is characterized by sudden headache, vomiting, visual impairment and meningismus. Headache is usually the most common presenting symptom. Some precipitating factors of pituitary apoplexy include anticoagulation and head trauma, both of which were present in this case. Other precipitating factors include hypertension, radiation therapy, recent surgery, bromocriptine therapy and dynamic pituitary function testing. The clinical presentation can vary greatly ranging from a mild headache to hemodynamic compromise. If blood enters the subarachnoid space, meningitis-like symptoms including fever, meningismus, vomiting and altered mental status can occur. These patients deteriorate very rapidly. The urgency of surgical decompression depends on the presentation, and many of these cases are managed conservatively.

The key to diagnosis is to first recognize the condition. Initial laboratory data should include a complete blood count, coagulation studies, electrolytes, a baseline cortisol level and thyroid function tests. Pituitary hormone deficiency is very common. Hypogonadism, growth hormone deficiency, and prolactin abnormalities occur in many cases. Acute adrenal insufficiency (AI) can be very serious and is found in 2/3 of cases. Hypothyroidism is found in about 42% of cases. Diabetes insipidus can occur from damage to the

posterior pituitary gland and is seen in only about 4% of cases. Electrolyte disturbances, especially hyponatremia, are also common. Hyponatremia can occur from inappropriate ADH secretion, secondary AI, or secondary hypothyroidism. Laboratory data to check for hypopituitarism include TSH, prolactin, LH, FSH, cortisol, GH, estradiol in females and testosterone in males. A lumbar puncture can be performed to rule out meningitis and subarachnoid bleed. Radiologic studies are used to confirm the presence of pituitary apoplexy. Imaging by CT is often used acutely, however MRI is preferred because it is more sensitive and is often used in the subacute setting. Management involves supportive care, "stress-dose" steroids, and a neurosurgical evaluation. Hydrocortisone 100mg IV every 6-8 hours is often used or dexamethasone 4mg IV every 8 hours or equivalent. Pituitary hormone replacement is used when necessary. Transsphenoidal surgery may be indicated if there are visual changes. Radiotherapy is not indicated immediately since the risk of recurrence is negligible. Careful follow-up is recommended.

References

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