TTP: An Unsuspecting Culprit of CVA
Laura Barba OMS-III, Kimberly Willis MD
1. Campbell University School of Osteopathic Medicine, Lillington, NC
2. Department of Medicine, Wayne Memorial Hospital, Goldsboro, NC

BACKGROUND

Highly prevalent are all causes of neurologic accidents (CVA) perioperatively in nature, and include cryptogenetic (80%), lacunar artery disease (20%), embolic (5%), and hemorrhagic (5%). CVA diagnoses are not limited to arterial causes (dissections, aneurysms, vasculitis, CVA). Common risk factors include hypertension, atrial fibrillation, diabetes, smoking, hyperlipidemia, and cardiac failure.

Clinical outcome is variable and unpredictable. However, when a patient demonstrates progressive deterioration it is critical to reassess the differential and identify new etiologies, including pathologic hypocoagulability, as are addressed.

Case description: the clinical course and diagnostic challenge of a CVA patient who suffered with recurrent CVA. Classic risk factors were initially thought to be the etiology of the CVA, but progressive neurologic deterioration revealed that initially presenting, thrombotic thrombocytopenia purpura (TTP) was the culprit. TTP is a rare etiology of CVA. Awareness of this entity yields more accurate diagnoses and prompt treatment.

LEARNING OBJECTIVES

1. Discuss its mode of presentation of TTP and CVA
2. Present the limitations of CT imaging used in diagnosing CVA
3. Differentiate for acute pathology when recovering CVA suspected
4. Discuss the use of CBC and blood smear results in diagnosing hematologic pathology

INITIAL CASE PRESENTATION

48 year old African-American female with a past medical history of tobacco abuse, GAD, HPI, hypertension, Type II diabetes, and hypercholesterolemia, presented to the ED with her husband for slurred speech, confusion, epigastric burning and high blood sugar. The patient is compliant with her anticoagulation and diabetic medications (both well controlled). She admits to smoking cigarettes 30 years but denies alcohol or illicit drug use. Family history is significant for CAD in her mother, surgical history is unremarkable, hypertension, dyslipidemia.

ROS: c/o nausea, vomiting, slurred speech, and confusion which resolved 3 days prior. Sicker, weakness, sensation or motor deficits, chest pain, GUL, incontinence, hematochezia, or skin changes.

PE: Vital WNS: AD 0.5, HR 90, Resp 20, BP 110/70, GCS 15. No focal neurologic deficit, left pronator drift, intact deep tendon reflexes, normal mouth, muscle, and speech.

Laboratory: CMP and LFTs normal, platelet count of 6, troponin I <0.1, and DIS normal. Glucose mildly elevated at 154. Head CT without contrast was negative for acute pathology. Chest X ray negative for acute pathology. Troponins and EKG normal. Glucose mildly elevated at 154. Patient is compliant with her anticoagulation and diabetic medications (both well controlled).

Patient was treated with fluids, Zofran, morphine, and GI cocktail with resolution of her blood pressure and diabetic medications (both well controlled). She admits to confusion, HA, epigastric burning and high blood sugar. The patient is compliant with her anticoagulation and diabetic medications (both well controlled).

HOSPITAL COURSE

Admitted to the ICU with complaints of slurred speech. Patient states she “just can’t think or get her words out.” Today around 10:00 her husband states patient was on the phone with her son and he noticed the slurred speech. During this time he does not think his wife was unable to make complete sentences and was incomprehensible.

- ROS: GCS 5, general weakness, difficulty with speech, throbbing headache. Decreased bilateral vision, distress, or unsteady gait.

Patient was admitted for lab/diagnostic workup:
- Further imaging and diagnostics: MRI revealed multi-focal ischemia within right frontal, left posterior, and bilateral central status. Normal TTE and EKG normal. Lower extremity venous Doppler negative.

BLOOD SMEAR RESULTS

- Blood smear shows malariform polychromatophilic cells, target cells, and Coombs positive.
- Troponin, Dimer, increased 10/17/2015.
- CBC shows platelet count of 6.
- Blood smear shows pancytopenia.
- APTT prolonged 10/17/2015. Platelets low at 64.
- Head CT without contrast was negative for acute pathology. Chest X ray negative for acute pathology.
- Labs/Imaging: no repeat CBC/CMP. Repeat head CT revealed enlargement of ischemia in the head of the right caudate and left parietal.
- Labs/Imaging: normal.

DIAGNOSTIC IMAGING

FLAIR MRI Results 10/20/2015
- FLAIR MRI Results 10/22/2015

FLAIR MRI Results 10/20/2015
- FLAIR MRI Results 10/22/2015

CONCLUSIONS AND CONSIDERATIONS

Conclusions:
- MRI may play a more sensitive role in diagnosing CVA when clinical symptoms are present but CT is negative.
- Coagulation studies should be included early in differentiating recurrent TTP/CVA are present without classic etiology.
- TTP is a rare etiology of CVA.
- Would assessment of peripheral smears have led to a diagnosis of TTP earlier?
- Should physicians include daily basic labs such as CBC and CMP in their routine management of a CVA patient with a history of non-specific thrombocytopenia?

Always remember that anamnestic pathways may not present commonly.

BIBLIOGRAPHY