Clinical Image

Cerebral venous thrombosis presenting with headache only and misdiagnosed as subarachnoid hemorrhage

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INTRODUCTION

Patients with headache constitute up to 4.5% of emergency department (ED) visits.^[1] Cerebral venous thrombosis (CVT) is an important cause of the headache that is more common than once suspected. The diagnosis of CVT is often missed or delayed because of non-specific clinical manifestations, and brain computerized tomography (CT) may easily be misinterpreted.^[2] Herein we report a patient with CVT who presented with headache only and was misdiagnosed with subarachnoid hemorrhage (SAH).

CASE

A 78-year-old man who had headaches off and on for one year presented to our emergency department with a sudden-onset, severe (thunderclap) headache. The current headache was different from previous headaches. His pain was not associated with nausea, vomiting, fever, photophobia, phonophobia, or lethargy. Recently he had no viral illness or toxic exposure, but a stroke one year

ago. He was treated with clopidogrel for one year since the onset of the stroke. He had no history of malignancy, hematologic disorders, or recent infection.

Vital signs of the patient included temperature 36 °C (98.6 °F, temporal), pulse 91 beats/minute, blood pressure 124/80 mmHg, and respiration 20 breaths/minute. Physical examination showed nothing abnormal, with normal cranial nerve, muscle tone, and cerebellar function. The neck of the patient was supple. His right optic disc could not be evaluated because of a cataract. His left optic disc was normal (no papilledema). Laboratory tests including blood count, C-reactive protein, basic chemistries, and D-dimer were normal. SAH was diagnosed and non-contrast CT was performed.

Non-contrast CT (Figure 1) revealed blood in the suprasellar cistern, along the straight sinus, in the posterior interhemispheric fissure and tentorium. These findings were suspicious for CVT; thus, a contrast CT scan of the head (Figure 2) showed filling defects in the transverse and sigmoid sinuses bilaterally, and in the posterior superior sagittal sinus.

Figure 1. The non-contrast CT scan of the head.

Figure 2. The contrast CT scan of the head.

Figure 3. A brain magnetic resonance venography.

The day after admission, brain magnetic resonance venography (MRV) showed venous thrombosis involving the rectus sinus, superior sagittal sinus, and transverse and sigmoid sinuses bilaterally (Figure 3). Heparin infusion was started. When tested for a possible hypercoagulable state, the following were found to be normal (or negative): antithrombin III, protein C, protein S, homocysteine, factor V Leiden mutation, prothrombin gene mutation, lupus anticoagulant, antinuclear antibody (ANA), anti-DNA, anti-neutrophil cytoplasmic antibody (ANCA), anticardiolipin, and antiphospholipid antibody. The patient was discharged after therapeutic anticoagulation and had no complaints after a two-week follow-up, indicating the disappearance of headache.

DISCUSSION

CVT is less common than other types of cerebrovascular catastrophe and presents with a wide variety of signs and symptoms. Common presentation includes isolated intracranial hypertension, focal neurological abnormalities, seizures, and encephalopathy.^[3] Predisposing causes include oral contraceptives, pregnancy, malignancy, infection, head injury, and prothrombotic conditions like protein C/S deficiency and antithrombin deficiency.^[4] Headache is the most common presenting complaint in CVT patients, with most reporting a gradual onset of the pain. Rarely, as in our patient, headache can mimic the "thunderclap" headache of SAH. Additionally, headache may be the only symptom of CVT, and other symptoms and signs of intracranial hypertension, subarachnoid hemorrhage, meningitis, or an intracranial mass may be absent.^[5,6] In our patient, no predisposing factor was identified and the neurological examination was completely normal.

CVT which can have similar clinical features to aneurysmal SAH, rarely can be also a reason for nonaneurysmal SAH. The mechanism of development of SAH in isolated CVT is not certain. The possible mechanisms include: (1) rupture of venous paraneurysmal hemorrhagic infarcts into the subarachnoid space; (2) increased vascular permeability as a local inflammatory response and extravasation of the blood into the subarachnoid space; and (3) rupture of cortical veins due to venous hypertension.^[7]

Unenhanced head CT or MRI remains the modality of choice for imaging patients with nonspecific clinical presentation who may have intracranial bleeding. Although these techniques may detect alternative diagnoses or demonstrate venous infarcts or hemorrhages, they do not rule out CVT. A venographic study should be performed if CVT is suspected.^[4] Signs of acute CVT on unenhanced CT include hyperdensity in the area of a sinus or cortical vein (dense clot sign and cord sign, respectively). A hyperdense appearance of the thrombosed cortical veins may be confused with hemorrhage. On contrast CT, filling defects, especially in the superior sagittal sinus ("empty delta sign"), support the diagnosis of acute or chronic CVT. However, this finding may vanish with time, as the thrombus enhances as it becomes more organized. In the subacute and chronic stages, thickening of the dura and enhancement of the cortical veins occur due to congestion from circulatory failure.^[3,8] In our patient, no acute thrombosis was found. Also, the typical "empty delta sign" was not present in the superior sagittal sinus, probably due to partial recanalization. Thickening

of the dura and cortical veins, as well as filling defects in the posterior superior sagittal sinus (with partial recanalization) and transverse and sigmoid sinuses bilaterally on MRV supported the final diagnosis of CVT.

In conclusion, CVT may present with a wide variety of non-specific clinical manifestations. Images, especially those of non-contrast CT, can easily be misinterpreted. Morbidity and mortality increases if initially misdiagnosed and treatment is delayed. Typical radiological findings may be absent in the chronic phase; in such patients in whom CVT is suspected, MRV should be performed to rule in or out the diagnosis.

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