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CT Perfusion Findings of Cerebral Venous Thrombosis; Potential Mimicker for Acute Arterial Stroke

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Abstract

We are presenting a case in which cerebral venous thrombosis presented to our hospital with an acute aphasia which clinically mimicking acute ischemic stroke, prompting activation of stroke code alert and showed unusual CT perfusion findings. Although correct diagnosis was made by brain CT angiography, possibility of arterial ischemic stroke was carried on giving the presenting symptom and altered CT perfusion parameters.

Introduction

Cerebral venous thrombosis is an important etiology of stroke in young patients. Its clinical manifestations are variable and based on different involved venous or dural sinus. Cerebral venous thrombosis could mimic ischemic arterial infarction and is easy to misdiagnose. Although many patients have

favourable outcomes, delayed or incorrect diagnosis due to atypical symptoms may lead to a poor prognosis. [1] The underlying cause of CVT is variable. Recognized risk factors for CVT include pregnancy, recent childbirth, oral contraceptive (OC) drug use, prothrombotic states and disorders of coagulation; consequently, the majority of patients with CVT are female. The clinical course of CVT is highly variable. [2] CT venography is one of the most often used imaging modalities for the diagnosis of CVT because of its widespread availability and cost-effectiveness. [3] Compared to CT venography, MRI is more sensitive for the detection of small parenchymal lesions and cerebral edema and has the advantage of not exposing the patient to ionizing radiation. [3, 4,5]

Acute disease management includes anticoagulation with heparin and supportive treatment. Supportive therapy includes hydration, anticonvulsants, antibiotics and neurologic interventions to reduce intracranial pressure. [2]

Case Report

A 53-year-old male, Pakistani, non-Arabic and non-English speaker brought by his friend to the emergency department after being found to have difficulty with speech, confused just one hour from last time when they were together. Upon arrival, Stroke code Activated six hours from the onset of the symptoms. Initial clinical assessment struggled by language barrier. However, he was aphasic, expressive more than receptive aphasia, dysarthria with right visual field defect. Otherwise, no focal motor, sensory or ataxic finding. His Initial NIHS was 8. Hemodynamically stable blood pressure was 140/70 mmhg. Heart rate was 70 beat per minutes. Respiratory rate was 21 per minutes. Oxygen saturation 95% in room air and temperature 37.5 C. Electrocardiogram showed normal sinus rhythm.

No enhanced brain CT scan showed no acute established territorial infarction or hemorrhage, hyper dense superior sagittal sinus was seen retrospectively (Figure 1. A&B). CT angiogram showed no arterial flow limiting stenosis or occlusion but thrombosed superior sagittal, right transverse and right sigmoid venous sinuses with filling defects extending into the right internal jugular vein (Figure 1. C, D, E &F).

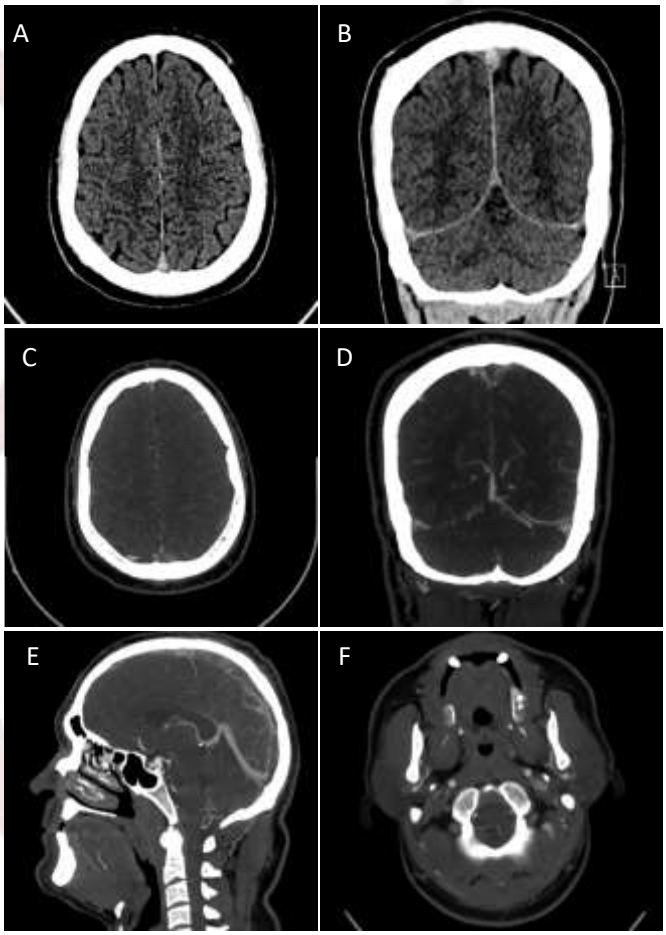

Figure 1: (A&B) No enhanced brain CT scan depicted hyper dense SSS. (C&D) corresponding cuts of brain CT angiography showed empty delta sign of SSS and engorged cortical vein in the right parietal region. (E) Sagittal brain CTA showed extensive SSS filling defects that was extending through the right transverse and sigmoid sinus down to (F) the right internal jugular vein.

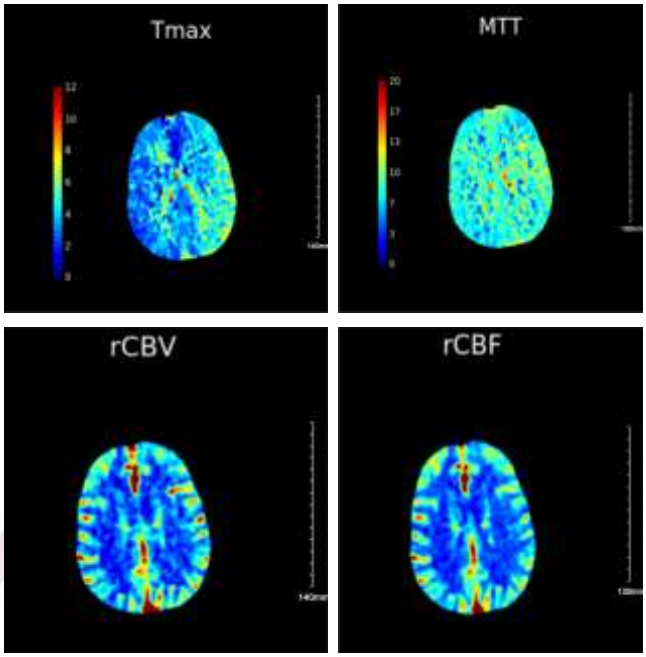

Figure 2: CT perfusion images showed prolonged Tmax and MTT in the contralateral side with normal rCBF and rCBV suggesting venous congestion. The right cerebral hemisphere showed normal CT perfusion imaging appearance.

CT perfusion showed prolonged Tmax and MTT in the left cerebral hemisphere with normal/ near normal CBV and CBF; however, no appreciated perfusion changes in the ipsilateral right side. Those changes in Tmax and MTT were not following an arterial territory (Figure 2). Diagnosis of cerebral venous thrombosis was made, although the CT perfusion changes were not fully understood at that time. Giving that beside the clinical presentation, brain MRI was done in emergency department and showed normal left cerebral hemisphere on DWI images without acute infarction or hemorrhage. MR findings of dural vein thrombosis were seen (Figure 3).

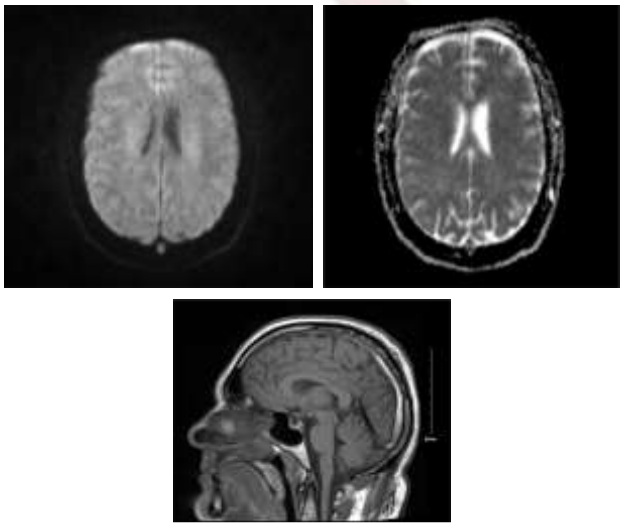

Figure 3: (A) DWIs and ADC map showed normal left cerebral hemisphere without acute infarction. Note the restricted dot in the SSS. (B) Sagittal T1WIs depicted hyperactive intense SSS related to the previously seen thrombosis on CT and CTA.

Clinical diagnosis of cerebral venous thrombosis was confirmed and started on Enoxaparin 100 mg BID. Dose adjusted to his weight.

Next morning his aphasia and confusion has improved. He was able to tell his story to a medical physician who shares the same language, telling that he had a sudden severe holocephalic headache the day before he developed his symptoms and the sudden onset of his illness. He describes his usual headache, which is mild to moderate in severity and in terms of frequency ones each six months.

He described no clear precipitants of his headache. He reported that he has been diagnosed with diabetes and taking oral medications. And he smokes. No history of hypertension, no recent infection, fever, trauma, or gastrointestinal illness nor dermatological issues.

His examination was unremarkable. His NIHSS score was 0. CT venogram done 2ed day and aimed to establish extent of cerebral venous thrombosis which shows diffuse filling defect involving the superior sagittal, right transverse and sigmoid venous sinuses, extending into the proximal internal jugular vein, consistent with cerebral venous thrombosis. The left internal jugular, left sigmoid, left transverse, straight sinus, inferior sagittal sinus, basal vein of Rosenthal, vein of Galen and internal cerebral veins were patent.

Blood workup ordered including complete blood count, renal profile, liver profile, coagulation profile and troponin were all with within normal range. Hgb A1C was 7.6%. Cholesterol Total 7.28 mmol. LDL 5.50. HDL 1.11. Patient asked for discharged and prescribed on apixaban 5 mg twice daily for 6 months with diabetes medications.

Discussion

Cerebral venous thrombosis CVT is a rare form of cerebrovascular emergency, which can be challenging to diagnose due to its variable nonspecific symptoms that may lead to misdiagnosis and devastating outcome.

Recognizing patients with this stroke mimic who are at a high risk of developing ICH prior to thrombolysis therapy prompts effective risk communication between clinician and patient. In addition, appropriate therapy with anticoagulation rather than systemic thrombolysis should be applied earlier. [6]

In our hospital, stroke code imaging protocol includes, non-enhanced CT scan, monophasic CT angiogram (CTA) and CT perfusion (CTP). The main value of monophase CTA is to evaluate the arterial system, timing of the acquisition is important for optimal arterial imaging, although the intracranial venous system can be pacified with contrast at the time of scanning, but early acquisition can hide underlying filling defects.

On non-contrast brain CT, direct signs of a thrombus, such as a dense clot sign, cord sign of the cortical vein, and dural venous sinus sign, as well as indirect signs, such as intracerebral haemorrhage, subdural haemorrhage, subarachnoid haemorrhage, cerebral edema, and venous infarction, facilitate the diagnosis. [7]

Considering the venous phase of multiphase CT angiography, and measuring haematocrit and D-dimer may help clinicians to recognize CVT. [6]

Regarding other image protocols for diagnosing CVT, the empty delta sign on contrast-enhanced CT and venous phase of multiphase CT angiography are also useful. In the absence of both time-of-flight and contrast MR venography, brain MRI with the simplest protocol still enables clinicians to identify a fat sinus sign (enlarged dural sinus with convex margin), disappearing flow void due to a blood clot, goral swelling/parenchymal hyper intensity on T2-weighted and fluid-attenuated inversion recovery imaging,

blooming on T2* imaging, and parenchymal venous ischemia on diffusion-weighted imaging. [7]

The intracranial venous system is a complex three-dimensional structure that is often asymmetric and considerably more variable than the arterial anatomy. [8]

The cerebral venous system can be divided into a superficial and a deep system. The superficial system comprises sagittal sinuses and cortical veins, which drain superficial surfaces of both cerebral hemispheres. The deep system consists of the lateral sinus, straight sinus and sigmoid sinus along with draining deeper cortical veins. Both of these systems mostly drain into internal jugular veins. Generally, venous blood drains into the nearest venous sinus or, in the case of blood draining from the deepest structures, into deep veins. The superficial cerebral veins are interlinked with anastomotic veins of Trolled and Label. Thus, the super lateral surface of the hemisphere drains into the superior sagittal sinus while the poster inferior aspect drains into the transverse sinus. [9]

Venous infarction and parenchymal haemorrhage are well known complications of CVT, few studies were made to assess the MR and CT perfusion changes in CVT. Mokin et al. in their retrospective study showed among 10 patients with acute venous sinus thrombosis (VST) included in that study, 9 had CT perfusion abnormalities. All perfusion abnormalities were localized in areas adjacent to the occluded sinus and did not match typical anterior or posterior circulation arterial territories. Bilateral perfusion deficits were seen in 4 cases. CBV abnormality was observed in 8 cases, CBF abnormality in 7 cases, MTT abnormality in 9 cases, and TTP abnormality in 9 cases. [10]

Doege et al. used MR perfusion imaging in 6 patients with acute VST and compared patterns of perfusion deficits with diffusion-weighted MR imaging changes. They concluded that an increase in MTT in the absence of changes in CBV and the MR imaging apparent diffusion coefficient was indicative of reversible parenchymal changes, analogous to the penumbra phenomenon in ischemic stroke. [11]

Gupta et al. also reported a potential role for CBV and CBF values in the prediction of clinical outcomes for patients with VST. [12]

Conclusion

CVT can mimic acute arterial stroke clinically and radiologically, high index of suspicion should lead the radiologist for the correct diagnosis. Alteration in CT perfusion can be misleading especially when the presenting symptoms are associated with focal neurological deficit. The variation in CT perfusion findings is likely attributed to the wide variation in cerebral venous anatomy, extension of the thrombosis and involvement of deep or superficial systems. Further studies are needed to study the prognostic value of CT perfusion in cerebral venous thrombosis and the diagnostic value in early detection of small cortical veins thromboses.

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