HEMORRHAGIC CEREBRAL VENOUS INFARCTION IN A PATIENT WITH ATRIAL FIBRILLATION: CASE REPORT OF AN UNUSUAL MRI FINDING

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ABSTRACT
 Venous sinus thrombosis is an uncommon cause of stroke. Magnetic resonance imaging and magnetic resonance venography are sensitive and specific non-invasive tools for the diagnosis. This is a report of patient who presented with atrial fibrillation and heart failure. The course of the disease was complicated by left-sided transverse sinus thrombosis leading to hemorrhagic stroke. There was normal looking transverse sinus upon contrast injection with gadolinium despite the lack of flow on magnetic resonance venography. It is postulate that this apparently normal transverse sinus appearance on post gadolinium T1-weighted imaging was a result of thrombus enhancement in the acute stage of the venous occlusion. This finding could mislead the diagnosis if it was read in isolation without the confirmation of the occlusion in magnetic resonance venography.

Keywords: Venous sinus thrombosis, MRI, MRV, Enhanced thrombus, AF, Enhancement.

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INTRODUCTION

Cerebral venous sinus thrombosis (CVSS) is a challenging condition due to its variability in clinical presentations, and the need of high index of suspicion to reach and confirm the diagnosis\[^{1,2}\]. It can be complicated with hemorrhagic infarction and subsequently increases mortality rate\[^{2,3}\]. Early and accurate diagnosis is required to prevent complications and decrease this mortality\[^{4,5}\]. Magnetic resonance imaging (MRI) and magnetic resonance venography (MRV) are useful tools for the diagnosis\[^{6,7}\].

CASE REPORT

A previously healthy 53-years-old female, presented to emergency department with shortness of breath for two days. The patient was hypotensive with rapid atrial fibrillation (AF) and signs of heart failure (HF). She was stabilized and then admitted to Cardiac Care Unit (CCU) for close monitoring and management. Two days later, she became confused with evolving headache and double vision. The patient was afebrile with normal blood pressure. Cranial nerves examination revealed: papilledema and the right 6th nerve palsy. Otherwise, the rest of neurological examination was unremarkable.

Initial unenhanced computed tomography (CT) of the brain (Fig. 1) showed a massive left occipital and temporal lobe bleeding with cytotoxic edema and mass effect. This CT scan finding was not compatible with the diagnosis of embolic infarction secondary to atrial fibrillation with hemorrhagic transformation as the amount of hemorrhage was disproportionally larger than the size of infarction. Unenhanced T1-weighted MRI confirmed the presence of occipital and temporal lobe hemorrhage with loss of the left transverse sinus flow void (Fig. 2). Gadolinium-enhanced T1-weighted imaging (Fig. 3) showed hyperintensity in the left transverse sinus. MRV (Fig. 4) documented the absence of flow signals in the left transverse sinus. Based on MRI/MRV finding, the diagnosis of left transverse sinus venous thrombosis was made. The patient was started on unfractionated Heparin infusion with partial thromboplastin time range of 1.5X to 2X. Subsequently, the patient underwent serial unenhanced CT examination of the brain over the week of her hospital stay, which revealed no expansion of the lobar hematoma. Indeed, her clinical status has improved. The patient was then started on Warfarin with an adjusted dose to maintain an international normalized ratio (INR) between 2 to 2.5. Unfractionated heparin was subsequently discontinued.

DISCUSSION

This is a case of left transverse sinus thrombosis with hemorrhagic infarction secondary to hypercoagulable state as a result of HF and dehyadratio\[^{8-10}\]. The initial diagnostic suspicion was that of an embolic stroke with hemorrhagic transformation secondary to AF\[^{11}\]. However, the amount of the hemorrhage was disproportionately larger than the size of the CT-detected infarction. Other causes of intracerebral hemorrhage (ICH) were considered and were meticulously excluded\[^{12}\].
Further evaluation with MRI revealed ICH with absence of the transverse sinus flow void (Fig. 2). Furthermore, Gadolinium enhanced T1-weighted MRI revealed high signal intensity in the left transverse sinus (Fig. 3), which may be otherwise interpreted as normal blood flow within the sinus. MRV documented the absence of flow signals in the left transverse sinus (Fig. 4) that indicates venous sinus occlusion. The high signal intensity seen in the left transverse sinus was due to enhanced thrombus rather than normal flow of gadolinium within the sinus. This finding was confirmed by MRV that showed the absence of flow signal within the left transverse sinus.

Magnetic Resonance Imaging (MRI) and Magnetic Resonance Venography (MRV) are considered sensitive and specific non-invasive tool for diagnosing CVSS[6,7,13,14]. Classically, MRI with gadolinium may show the filling defect within the occluded sinus or may show peripheral enhancement in acute or sub-acute stages. A more uniform enhancement will be seen in chronic clots due to recanalization or vascularization[6]. MRV usually confirm the diagnosis and shows the actual occlusion within the sinus[15].

In the presented case, the thrombus was enhanced in spite of the acuteness of the occlusion. This gave a false impression of the normal flow within the sinus. The clinical presentation of this case was potentially misleading since it was suggestive of an embolic stroke with hemorrhagic transformation secondary to AF. As indicated above that few clinical data did not support this diagnosis with the presence of relatively large volume of hemorrhage compared to the small size of the infarct.

Cerebral Venous Sinus Thrombosis (CVSS) should be always considered in cases of unusual intracerebral hemorrhage where detailed MRI/MRV examinations should be acquired.

Physicians should not be confounded by the presence of AF and acclaim all strokes to thromboembolism. This case teaches us that the etiology of stroke should be carefully evaluated, particularly, when there is conflicting clinical or radiological data.

REFERENCES