CASE REPORT

Importance of Contrast Enhanced MRV in Diagnosis of Cortical Venous Sinus Thrombosis

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ABSTRACT

Introduction: Cortical venous sinus thrombosis is a rare condition and a diagnostic challenge due to its presentations. In this case report, we highlights the value of prompting diagnosis of cortical venous sinus thrombosis versus benign intracranial hypertension through CSF and neuroimaging, contrast material enhanced MRV and the importance of immediate anticoagulation as a part of patient management.

Case report: A 25-year-old male presented with complaints of headache, giddiness, and diminution of vision since 3 month, diagnosed to have cortical venous sinus thrombosis. He improved with anticoagulation and supportive therapy.

Conclusion: Cortical venous sinus thrombosis(CVST) is a challenging condition. A prompt diagnosis through neuroimaging contrast enhanced MRV is a good choice in case of CVST. Expedited standared management for the CVST should be employed to help ensure the best possible outcome for patient.

Keywords: Cortical venous sinus thrombosis (CVST), Magnetic resonance venography (MRV)

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INTRODUCTION

Cortical venous sinus thrombosis is a rare condition which occurs with an incidence of 3-4 cases/million/year. Cortical venous sinus thrombosis is a diagnostic challenge due to its presentations.Whenever, clinical suspected, promptthe investigation by Magnetic resonance (MR) imaging, un-enhanced computed tomography (CT), unenhanced time-of-flight MR venography, and contrast material-enhanced MR venography and CT venography will helpful in prompting diagnosis. Our report highlights the value of prompting diagnosis of cortical venous sinus thrombosis versus benign intracranial hypertension through CSF and neuroimaging, contrast material enhanced MRV and the importance of immediate anticoagulation as part of patient management.

Cortical venous sinus thrombosis (CVST) is an uncommon condition affecting 3-4 cases/million/year with a mean age of 37 to 38, though any age may be affected.[1,2] As compare to men, women are at an increased risk particularly between the age group of 20-35years which mainly due to the use of oral contraceptive pills and the postpartum state.[2] Upto 80% of patients, predisposing factors are identified.[3] In CVST, the clinical presentation is varied which include headache, vomiting and seizures. In this article we report a patient who developed a sigmoid sinus thrombosis with no discernable underlying cause.This rare case presentation help to increase early diagnosis and management of CVST.[1,3,4]

CASE REPORT

A 25-year old-male was admitted to the MGM hospital,aurangabad, with complaints of headache on & off, giddiness, and diminution of vision since 3 month, there was no history of trauma, no relevant past medical or drug history and no family history of note. On examination, patient was stable with a GCS of 15/15. His pupil, & speech appered normal &there was no evidence of any focal neurological deficiency, or meningitis. Hisfundoscopy shows chronic papilloedema. On imaging MRI brain withvenography was normal., CSF examination was done. CSF pressure was high (25mm/H_2O), 15mm/H_2O drained out. CSF examination (Rou-

tine&microscopy) - Normal with corresponding sugar level 98 mg %.6 hrs after Lumbar Puncture procedure, patient complain of headache which was low in intensity, increased on standing and sitting position. Headache was gradually increased over 2-3 days, associated with 1-2 episodes of vomiting. Patient also complaints of slight diminution of vision to right side. After 4 days, CT Brain (plain) was done which showed signs of cortical venous thrombosis. Repeat MRI with venography + contrast done on same day which showed Chronic Right Sigmoid sinus thrombosis. Patient was treated with low molecular weight heparin (LMW) for 5 days, After 5 days, patient shift on oral anticoagulants (Tab. warf). PT -INR was 1.28 on discharge. At discharge, patient was stable, pupils were normal and reacting to light, visual acuity improved. No complaints of headache. Patient was discharged on oral anticoagulant (T. warf) and to be follow up.

DISCUSSION

Venous drainage of the brain include blood flow through cerebral veins into the dural sinuses which drain into the internal jugular vein. Sigmoid sinus is the main cerebral venous sinus affected by thrombus which involved in 62% cases in literature.[4]

The pathogenesis of CVST involves two mechanisms. The first constitutes localized oedema and venous infarction due to cerebral vein occlusion. The second mechanism include the development of raised intracranial pressure due to occlusion of one of the cerebral venous sinuses.Our case showed both mechanisms. However, it showed that only raised intracranial pressure occurs without any signs of cortical vein thrombosis.[5]

Neuroimaging is usually plays a key role in the diagnosis of CVT, though angiography is still considered to be the gold standard.[6] The other useful techniques are magnetic resonance (MR) imaging, un-enhanced computed tomography (CT), and contrast material-enhanced MR venography and CT venography which are detecting cerebral venous and brain parenchymal changes related to thrombosis.[7]

Pathophysiology of brain parenchymal involvement in venous occlusion or venous infarction is the obstruction of venous drainage with increasingenous pressure in the affected region of brain. CVT progresses to cerebral venous infarction in approximately 50% of cases.[8] Parenchymal changes may be secondary to cytotoxic edema, vasogenic edema, or intracranial hemorrhage.[8]

Signs of CVT in radiologically basis are direct and indirect. The direct signs includes demonstration of the thrombus on imaging or indirect, as when there are ischemic or vascular changes related to the venous outflow disturbances.[9] The indirect signs involves brain swelling by parenchymal change, white matter oedema, cortical sulci effacement, loss of grey and white matter differentiation, hemorrhagic infarction as hemorrhagic spot in white matter oedema.[10] The classic finding seen on non contrast CT is the delta sign, which is seen as a dense triangle (from hyperdense thrombus) within the superior sagittal sinus. However, it is not specific, since high attenuation in the healthy non-thrombosed sinus can be occasionally observed and it is common in neonates because of an elevated hematocrit.[11] The reverse delta sign i.e. empty triangle sign can be observed on contrast-enhanced CT scan in the superior sagittal sinus. The presence of both the delta and reverse delta signs indicates the likehood of the diagnosis. Infarction in a non-arterial distribution in the white matter and/or cortical white matter junction, associated with hemorrhage will decide on CT brain scan which showed the possible diagnosis of venous thrombosis. Bilateral cerebral involvement can occurs, which includes the superior sagittal sinus thrombosis, or the basal ganglia and thalami from internal cerebral vein thrombosis. Indirect CT signs consists of focal cerebral cortical ischemia with gyral enhancement, small ventricles compressed by cerebral oedema, and intense tentorial enhancement.

MRI finding S/O diagnosis usually can be made without intravenous contrast, although contrast enhancement can aid in confirming the diagnosis.[12] A thrombus can be directly visualized within a vessel. Secondary venous infarctions and foci of hemorrhage can be seen with gradient-echo images. Susceptibility-induced signal loss from deoxyhemoglobin provides a basis for detection of even small foci of hemorrhage, which tend to occur in the subcortical white matter, thalami, and basal ganglia.Parenchymal regions of T2-hyperintense signal abnormality in the distribution of the draining sinus is often observed and may be reversible, even when large. This may occur independent of recanalization of the thrombosed vessels. Dilated venous collaterals, such as transcortical medullary veins, provide indirect evidence of venous thrombosis. The appearance of intravenous thrombus on conventional MRI depends on the age of the blood clot within the vessel.

Daily routine practical work for diagnosis this condition include CT, CTV, MRI and MRV contrast-enhanced MRV). CT scan include plain and dynamic sequences as CT venography are the simple, effective method to diagnosis of cerebral venous thrombosis and

to be the first screening method. Compare among those imaging modalities, Contrast-enhanced MRV is necessary in many situations without radiation exposure to confirm or exclude thrombosis such as clot identification, parenchyma changes to the most subtle condition as cortical cortical vein thrombosis.

CONCLUSION

CVST is a challenging condition due to its wide range of clinical presentations. Clinicians should have a high index of suspicion, even in the absence of predisposing conditions, in order to facilitate a prompt diagnosis through neuroimaging contrast enhanced MRV is a good choice in case of stenosis versus thrombosis. Expedited standard management for the CVST should be employed to help ensure the best possible outcome for patients.

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