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Case Report

Sagittal sinus thrombosis secondary to varicella infection-a case report

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ABSTRACT

Varicella zoster virus (VZV) is known to produce a benign self-limiting exanthematous illness in the pediatric population. The neurological complications which are seen in less than 1% cases include cerebellitis and encephalitis. Stroke due to arterial vasculitis is well known but cerebral venous sinus thrombosis following varicella infection is rarely reported. We present a case of sagittal sinus thrombosis subsequent to varicella infection. The mechanisms underlying cerebral vascular events after VZV infection could be vasculitis, thrombosis due to direct endothelial damage and acquired protein S deficiency.

Keywords: Sagittal sinus thrombosis, Varicella infection

INTRODUCTION

Chicken pox produces a benign self-limited disease in children. Varicella-related neurological complications are seen in less than 1% cases of chickenpox. The most frequently encountered are cerebellitis and encephalitis. Aseptic meningitis, Guillain-Barre syndrome and transverse myelitis are relatively less common. VZV cerebral vasculopathy can occur after a primary varicella infection or zoster. Most of the earlier literature on VZV vasculopathy describes it as a disease affecting predominantly large arteries causing strokes and transient ischemic attack. The occurrence of cerebral venous sinus thrombosis (CVST) following primary Varicella Zoster infection is very rare. We report a case of sagittal sinus thrombosis secondary to varicella infection.

CASE REPORT

Our patient was a 20-year-old previously healthy male who presented in the emergency room with history of generalized tonic clonic seizures and altered sensorium. It was preceded by fever and severe headache. 15 days back he had developed rash typical of chickenpox on the trunk and the limbs. Systemic examination revealed residual scars and crusting lesions. Fundus showed bilateral papilloedema. Motor system examination showed weakness of left upper and lower limb (grade 1-2) with left extensor planter response. Routine hemogram and serum biochemistry were normal.

MRI showed a large irregular hyperintense lesion in the right front parietal lobe superiorly. Haemorrhagic changes were observed in it. A similar lesion was identified in the left frontal lobe posterosuperiorly. There was loss of normal flow void in the superior sagital sinus. The picture was suggestive of haemorrhagic infarcts with diffuse cerebral swelling secondary to thrombosis of superior sagittal sinus (Figure 1).

Positive varicella antibodies were detected in the serum and CSF. The patient was given decongestive antiedema therapy, injectable acyclovir and antiepileptics. Low molecular weight heparin was initially withheld but started later. He improved gradually over the next two weeks and was discharged on oral anticoagulants. After

6 months the patient can walk with a limp though the upper limb is still not fully functional especially the hand.

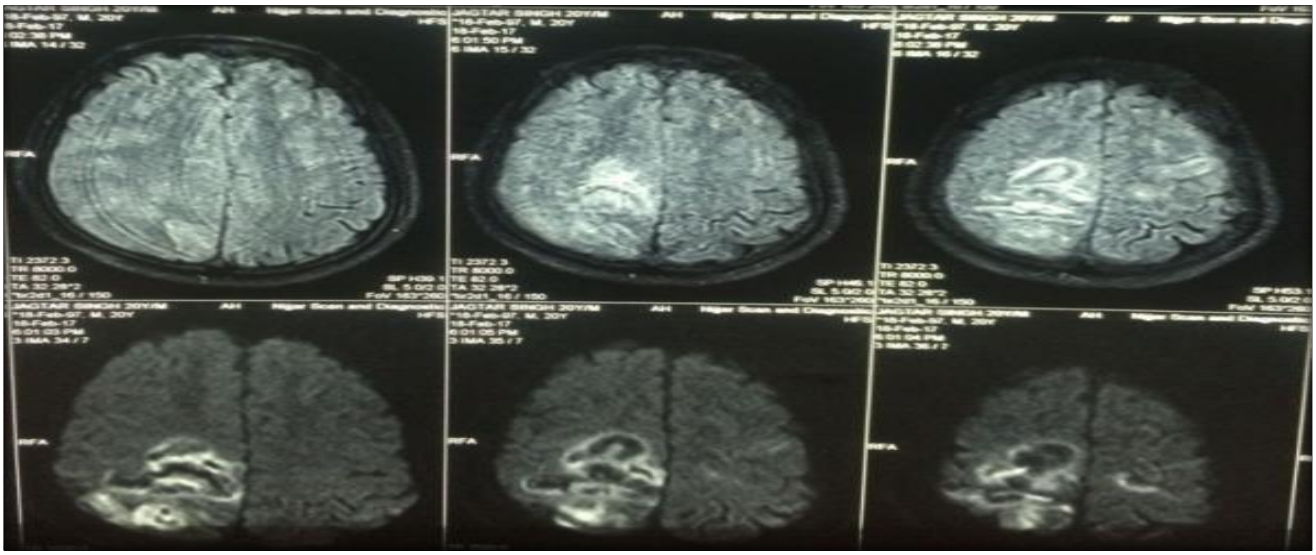

Figure 1: Haemorrhagic infarcts with diffuse cerebral swelling secondary to thrombosis of superior sagittal sinus.

DISCUSSION

After the primary infection period, the VZV virus may stay latent in the spinal and cranial ganglia neurons and can subsequently get reactivated to cause Herpes zoster in later life. As it is a highly neurotropic virus, neurological complications are expected following either primary VZV infection or reactivation. Neurological complications of varicella in children are common but rare in adults but associated with increased morbidity. It can affect both immunocompetent and immunosuppressed individuals. VZV is the only human virus that has been proven to replicate in cerebral arteries and cause cerebral arterial vasculopathy resulting in infarcts. It is believed that there is trans axonal transport of varicella zoster virus from cervical or trigeminal afferent fibers to the cerebral blood vessels to the adventitia followed by transmural migration to the arterial media and intima. The mechanisms underlying cerebral vascular events after VZV infection could be thrombosis due to direct endothelial damage, inflammation, vascular wall remodeling and disruption of internal elastic lamina.[1]

More recent studies have expanded the clinical spectrum of VZV vasculopathy to include aneurysms, dissection, ischemic cranial neuropathies, cerebral venous sinus thrombosis, and spinal cord infarction. The exact pathogenesis of varicella venous thrombosis is not known but similar to VZV arterial strokes. Activated varicella virus may migrate transaxonally to infect the meninges and venous sinuses of brain. Primary VZV infection can cause vascular thrombosis approximately 6weeks after primary infection. An acquired antibody-mediated hypercoagulable state resulting from decreased levels of natural anticoagulants like protein S in the viremic phase of the infection is also postulated to provoke thrombosis.[2-4] The latent period of 2-3weeks signifies the time for the direct venous endothelium damage or development of autoantibodies to natural anticoagulants leading to widespread thrombotic process.

Very few cases of venous sinus thrombosis have been reported in literature.[5-8] Our case developed CVST in the stage of primary infection itself. History of chickenpox, investigations and available literature helped confirm the diagnosis.

CONCLUSION

Cerebral venous sinus thrombosis (CVST) is a rare neurological complication associated with primary VZV infection. Early diagnosis and management can help prevent associated morbidity and mortality and lead to better outcome. The physician should have high index of suspicion for hypercoagulability in patients following varicella infection and institute appropriate therapy.

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