Cerebral Venous Thrombosis Following Varicella Infection

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8-year-old male was brought to the hospital with severe headache, vomiting and seizures. The illness started with profuse rash predominantly on the trunk, limbs and also on the face two weeks back. The lesions were centripetal and were diagnosed to be chicken pox. The lesions were in crusting stage when the patient developed neurological complaints. His symptoms started with sudden onset of headache which was associated with vomiting followed by seizures. Past history was not significant. When the patient came to the emergency room, he was conscious and appeared confused. Skin lesions were in the form of pigmentation and scarring following varicella infection (Figure 1).

Neurological examination revealed, pupils were bilateral equal reacting to light. Fundus showed bilateral papilloedema.. Motor examination showed no weakness of limbs, bilateral extensor plantars. His routine investigations including full blood count, blood sugar, renal, liver function tests, electrolytes and blood culture were normal. CT brain (plain) showed hyperdensity in superior sagittal sinus with hyperdense cortical veins (Figure 2). A possibility of venous sinus thrombosis was considered and magnetic resonance venography (MRV) was done. MRV (sagittal and coronal planes) showed thrombosis of superior sagittal sinus, left transverse sinus and sigmoid sinus (Figures 3 and 4). Cerebrospinal fluid examination showed pleocytosis with 40 cells/mm³, mildly raised protein 60 mg%, and normal glucose. Diagnosis of cerebral venous thrombosis following varicella infection was made. Patient was started on antiedema measures, antiepileptics and low molecular weight heparin. Headache improved and seizures controlled. Patient was discharged on oral anticoagulants with monitoring of coagulation parameters. His thrombophilic and vasculitic workup was negative. He is asymptomatic on follow up after one year.

Neurological complications of varicella in children is common and rare in adults. Neurological complications frequently encountered are cerebellar ataxia and encephalitis. Less frequent complications are Guillain-Barré syndrome, meningoencephalitis, transverse myelitis, aseptic meningitis, ventriculitis, optic neuritis, post-hepatic neuralgia, herpes zoster ophthalmicus and peripheral motor neuropathy.¹ Our patient had post-varicella infective cerebral venous thrombosis. The vasculitic and thrombophilic work was normal which ruled out other etiological possibilities. The CSF result suggested

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an infective etiology. Cerebral venous thrombosis after varicella infection is rare. The exact pathogenesis of varicella venous thrombosis is not known but similar to Varicella Zoster virus (VZV) arteriopathy, activated varicella may migrate transaxonally to infect meninges and venous sinuses of brain. The mechanisms underlying cerebral vascular events after VZV infection could be vasculitis, thrombosis due to direct endothelial damage, and acquired protein S deficiency. Our patient developed thrombosis during primary varicella infection and not as a delayed complication. He had no other risk factors for cerebral venous thrombosis. In conclusion, our case demonstrates that a rapid diagnosis of cerebral venous thrombosis following varicella infection which was essential for the proper management of the patient.

References