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Medicine

CEREBRAL VENOUS SINUS THROMBOSIS – RARE COMPLICATION OF VARICELLA INFECTION

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ABSTRACT Primary varicella zoster infection also known as chicken pox, a self limiting illness seen in children, may infect adults causing exanthematous febrile illness. Rarely life threatening neurological complications can occur which include meningitis, encephalitis, arterial vasculopathy, and venous thrombosis etc., in <1% of the population. We present a 32 year old male patient with cerebral venous sinus thrombosis with primary varicella infection. Venous thrombosis has been suggested to be caused by endothelial damage, auto antibodies against protein C and protein S, Pre-existing hyper coagulable state. The patient was managed with IV acyclovir and anticoagulation therapy with complete recovery without neurological deficit. We conclude that physicians should be aware of such rare complication, early diagnosis and timely intervention can decrease mortality and improve quality of life.

KEYWORDS : Varicella, Cerebral venous sinus thrombosis

INTRODUCTION:

Varicella (Chicken pox), a benign febrile exanthematous illness caused by varicella zoster virus. predominantly seen in childhood. However, in adults, though self-limiting, at times is associated with life threatening neurological complications such as cerebellar ataxia, Guillain-Barré syndrome, meningo –encephalitis, transverse myelitis, aseptic meningitis, and peripheral motor neuropathy[1]. We report a patient with post varicella cerebral venous sinus thrombosis as a rare complication of Varicella zoster viral infection.

CASE REPORT:

A 32 year old male patient who had chicken pox of 5 days duration, presented with continuous holocranial throbbing headache, non-projectile vomiting of 2 days duration, weakness of right upper limb and lower limb, non smoker, non alcoholic, non diabetic and non hypertensive.

General physical examination revealed chicken pox lesions in crusting stage on trunk and face (Fig:1) and with normal vital parameters. Neurological examination showed bilateral papilledema, right hemiplegia with right side Babinski sign.

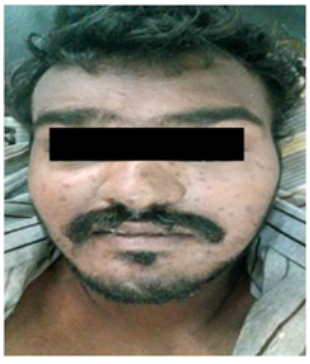

FIGURE: 1

Investigations - Hemogram and blood biochemistry were normal. Serology HIV, HBsAg, HCV were non-reactive. Thrombophilia profile, ANA was negative. Chest X-ray and 2D Echo were normal. Computed tomography (CT) brain plain showed diffuse cerebral edema with hemorrhagic infarcts in left parieto occipital region(Fig:2) and CT venogram was done which showed filling defects in left transverse and sigmoid sinus(Fig:3).

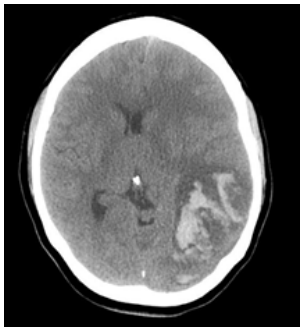

FIGURE 2

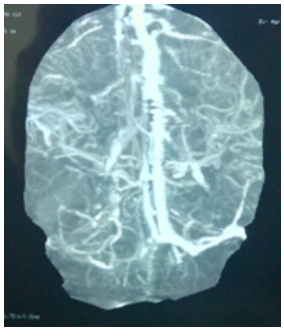

FIGURE 3

Patient was stared on IV mannitol and low molecular weight heparin subcutaneously. Patient improved gradually over 1 week. At the time of discharge, physiotherapy and oral anticoagulant was advised and continued to maintain INR between 2 – 3. Patient was on regular follow up for 6 months with no history of worsening of symptoms or bleeding manifestations.

DISCUSSION:

Primary varicella zoster viral infection, also known as chicken pox, usually seen in children as self limiting febrile illness with exanthematous rash. In adults, reactivation of varicella infection is more common compared to primary infection. Complications associated with varicella infection include encephalitis, cerebellar ataxia, transverse myelitis, ventriculitis, meningoencephalitis, and aseptic meningitis[2]. It can cause ischemic stroke, carotid dissection, aneurysm, and subarachnoid or cerebral hemorrhages as a

consequence of arterial vasculopathy. Rarely, cause purpura fulminans and venous thrombosis due to a hypercoagulable state[3-5]. We are presenting a case of cerebral venous sinus thrombosis, a rare complication of primary varicella infection.

The mechanism of venous thrombosis formation in post-primary varicella infection is hypothesized to occur due to either direct endothelial damage, inflammation of the vessel, induced autoantibodies against protein S, or a preexisting hypercoagulable state. In 2016, Paul, et al. stated that one out of three cases with post-varicella CVST had protein S deficiency, a preexisting hypercoagulable state [6]. In 2012, Siddiqi, et al. reported two cases of post-varicella CVST because of preexisting protein S and C deficiency [5]. According to a cross-sectional study done by Josephson, et al., 43 out of 95 children had antiphospholipid antibodies, while some had decreased protein S levels post-varicella infection. They called it varicella autoantibody syndrome [7].

Patients have varied clinical presentation like headache, vomiting, blurring of vision, cranial nerve palsies, focal neurological deficits, altered sensorium, convulsion etc. Vasculopathies after varicella infection are often diagnosed using a cerebrospinal fluid polymerase chain reaction for anti-varicella immunoglobulin G antibodies and Varicella deoxyribonucleic acid (DNA).

Varicella associated vascular complications are treated with IV acyclovir and anticoagulation with Low molecular heparin with warfarin as an oral anticoagulant. . With anticoagulation alone, the rate of partial or complete recanalization ranged between 47-100%. However, poor outcomes in 9-13% of the patients being treated with anticoagulation therapy for CVST have been reported. Alternate treatments for CVST include fibrinolytic therapy, direct catheter thrombolysis, mechanical thrombolysis and thrombectomy, and surgical management[8].

CONCLUSION:

Cerebral venous sinus thrombosis (CVST) is a rare neurological complication associated with primary varicella infection. The current case report highlights the importance of clinical examination and clinical suspicion of this rare entity for proper diagnosis and timely intervention which can help prevent associated morbidity and mortality and lead to better outcome.

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