Case Report

Stroke due to varicella zoster virus vasulopathy: A case report

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Abstract

Stroke is a common cause of mortality and morbidity worldwide. Apart from established risk factors, viral infections have also emerged as risk factors for stroke. VZV is the only virus that has been shown to directly invade cerebral arteries and produce vasculopathy in both immunocompetent and immunocompromised hosts. Contralateral hemiparesis usually results from infarction of the ipsilateral middle cerebral artery several weeks after the onset of herpes zoster ophthalmicus (HZO). Although only a few cases of stroke syndromes associated with HZO have been described thus far, it is very likely underestimated because of its diagnostic difficulty. The optimal treatment of CNS vasculitis caused by VZV is unknown and controversial results have been reported with many drugs. We are hereby reporting a case of 26-year-old immunocompromised male who developed transient ischemic attacks 8 weeks after an episode of HZO and responded well to anti- viral therapy.

Keywords: Anti-viral Therapy, Stroke, Varicella Zoster Virus, Vasculopathy**.**

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INTRODUCTION

Stroke is a common cause of mortality and morbidity worldwide[1]. While established risk factors, such as hypertension, diabetes, obesity and hypercholesterolemia have been identified and are targets for stroke prevention, viral infections have also emerged as risk factors for stroke. Varicella zoster virus (VZV), human immunodeficiency virus (HIV) and cytomegalovirus (CMV) have all been associated with stroke. However, VZV is the only virus that has been shown to directly invade cerebral arteries and produce vasculopathy.[2] The causative role of varicella-zoster virus (VZV) infection in vasculopathy in central nervous system (CNS) has been described in both immunocompetent and immunocompromised hosts; both varicella (primary infection) and herpes zoster (reactivation) have been associated with this disorder.[3,4] Contralateral hemiparesis usually results from infarction of the ipsilateral middle cerebral artery several weeks after the onset of herpes zoster ophthalmicus (HZO).[5] Stroke syndrome associated with herpes zoster can be further categorized into (i) that secondary to HZO, which develops in both immunocompetent and immunocompromised hosts, and (ii) that secondary to other forms of herpes zoster or even in the absence of herpes zoster lesion (zoster sine herpete), which develops exclusively in immunocompromised hosts. No effective treatment for the established stroke syndrome (contralateral hemiparesis) has been proved; there have been controversial results reported for treatment with prednisolone or anticoagulants while the role of anti-virals is still unproved. We are hereby reporting a case of young 26-year-old male who developed episodes of transient slurred speech and left hand weakness after an episode of HZO.

CASE REPORT

A 26 year old man with a history of End stage renal disease (ESRD) secondary to SLE on Haemodialysis with

recent VZV infection came to our emergency Department on 22/5/13 with transient slurred speech and left hand weakness. The patient had three such episodes in the last two weeks. The patient is young, with recent VZV meningitis and V1 zoster ophthalmicus (2 months back) with no other apparent stroke risk factors. MRI findings include abnormal signal and restricted diffusion in the right parietal centrum semi-ovale and internal capsule. Most of the signal abnormality seen on the trace images likely represents T2 shine through, more likely due to subacute insult in the right centrum semi-ovale region. No significant area of restricted diffusion (acute infarct) is present. No parietal signal abnormality infarct is identified. His initial MRA showed some narrowing in the right sided carotid siphon. MRA quality is limited due to motion. No high-grade stenosis or aneurysm is evident. No perfusion asymmetry is identified. MRA neck showed no abnormality. CT head showed hypodensity in the region of the right internal capsule likely corresponds to region of prior infarct noted on comparison MR. There is mild, generalized asymmetry of the prominence of the MCA distribution vessels, decreased on the right. The major arterial structures are otherwise normal in appearance. No definite stenosis or aneurysm. He was started empirically on 2 weeks of IV acyclovir. Serologic workup for other causes of vasculitis was negative including RPR, Hepatitis B and C, HIV. Cholesterol was negative and echo did not show evidence of a shunt. He refused line placement because of his ESRD and preferred to stay as an inpatient. He had one TIA while in the hospital on 27/5/13. As per his mother and him, he had slurred speech, left sided facial droop and left hand weakness. By the time the nurse got to the room, the facial symptoms had resolved and he only had left hand weakness. The left hand weakness was essentially stable. The patient refused further confirmatory work-up for VZV vasculopathy including angiogram or lumbar puncture and preferred to go home. He was discharged on tab. Valaciclovir (1gm) daily along with tab. Renagel (Sevelamer hydrochloride) 800mg thrice a day, Carvedilol (25mg) once a day, Hydroxychloroquine (200mg) once a day and artificial tears (2 drops in right eye). He was instructed to come back for regular follow up and also if the TIAs increased in frequency or persisted beyond 2-3 months. Patient came for regular follow up visits and has recovered well. No TIAs episodes were observed since discharge (6/6/13).

DISCUSSION

Reshef *at el.*[5] reviewed 51 cases of contralateral hemiparesis secondary to HZO. This is basically a syndrome of adulthood with mean age of 58 years. The early onset in our case (26 years) can be attributed to immunocompromised state. The mean time interval between HZO and contralateral hemiparesis was reported by Reshef *et al*.[5] to be 7.3 weeks. The time interval between the same in our patient was 8 weeks. Approximately one-half of the patients exhibited diffuse CNS symptoms/signs such as stupor, somnolence/drowsiness, general disorientation, confusion and memory deficits; documented seizure appears to be rare. Ichiyama *et al*.[6] estimated that the frequency of delayed hemiparesis secondary to varicella in the San-in district in Japan was roughly 1 in 6500 varicella patients. More recently a case-control study conducted in France demonstrated that 7 of 11 patients who had strokes (64%) had varicella within the 9-month period (median, 6 weeks; range, 9 days to 9 months) before the stroke, in contrast to 4 of 44 patients (9%) in the control group.[7] The optimal treatment of CNS vasculitis caused by VZV is unknown. There have been controversial reports regarding the use of steroids and anticoagulants. Review of the previously reported cases[8] showed no evidence to strongly support the use of these medications. Similarly the role of anti-herpes drugs such as acyclovir in this situation remains unproved; however, it should be recommended to treat VZV-associated stroke cases with large doses of acyclovir. Furthermore it could be reasonable to treat patients with HZO, a potentially vision-threatening disorder, with antivirals, because the treatment may limit pathologic process in the eyes and prevent viral spread to the cerebral blood vessels via trigeminal nerve branches. Although only a few cases of stroke syndromes associated with HZO or other types of herpes zoster have been described thus far, it is not unlikely that frequency of this disorder has been underestimated because of its diagnostic difficulty. CNS lesions caused by VZV are predominantly vasculopathies,[9] and VZV-associated CNS vasculitis occurs several months after the episode of primary varicella or HZO in otherwise healthy subjects. Although association of the virus with stroke attacks has been confirmed in only a few cases, the cumulative reports strongly suggest a critical role of VZV in stroke syndromes. Therefore this entity must be considered in apparently idiopathic cases of strokes.

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