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A RARE CASE OF GUILLAIN-BARRE SYNDROME PRECIPITATED BY VARICELLA ZOSTER VIRUS (VZV) INFECTION

General Medicine

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KEYWORDS

INTRODUCTION

Varicella zoster infection followed by polyradiculopathy is rare and happens in the reactivation of latent varicella roster virus (vzv).we report a case of polyradiculopathy triggered by varicella zoster infection.

Case Report

30 year old male patient came to causality with complaints of weakness of lower limb for 3 days which is sudden in onset and gradual in progression.Patient had a history of fever 12 days back and diagnosed varicella zoster infection for which patient was treated symptomatically, on day eight of fever patient had numbness and tingling sensation over lower limb following which patient had difficulty in wearing slippers , history of tripping over toes while walking.

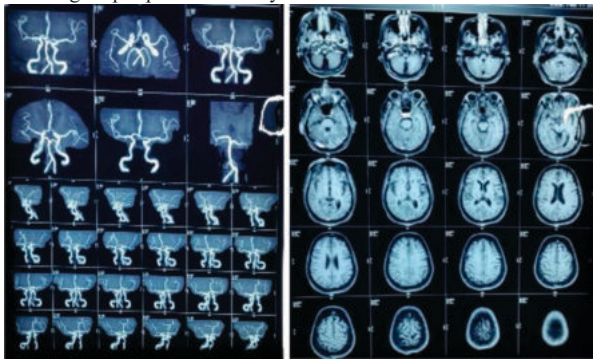
On examination pt conscious, oriented, afebrile, responding to oral commands.on systemic examination patient had weakness over distal lower limb of 2/5 (according to MRC scaling), there is no weakness over upper limb.finger to nose test, alternating hand movements were normal.The examination of gait and balance was impossible due to limb weakness.Normal bowel and bladder habits.lession over face and trunk revealed chicken pox scar session. The patient doesnot have previous history chicken pox and vaccinated witj and not vaccinated varicella. All baseline investigation done complete blood count , renal function test, liver function test came normal. C-reactive protein was elevated.Electroneuromyography done 4 days after the onset of symptoms and it showed reduced muscle action pottential,increased latency ,and reduced motor action velocity in lower limb.sensory nerve conduction study shows reduction in amplitude and sensory nerve action potential over lower limbs.MRI brain with MRA MRV contrast done it shows normal study.

CSF analysis done after 5days of onset of weakness csf glucose protein chloride ada are with in normal limits, no pus cells , malignant cells present.Antiganglioside antibodies was negative for both igM,igG.csf varicella zoster virus done it came positive for both IgG, IgM. Electroneuromyography done 4 days after the onset of symptoms.

Patient was started on intravenous acyclovir , IVIG for 5days followed by 500 mg of methyl prednisolone for 5 days and tapered and daily physiotherapy was given.patient had improved over lower limb weakness of 4/5 (MRC scaling) at the time of discharge and advised with oral corticosteroids and physiotherapy .after 1 month during follow up pt had complete recovery of his weakness.

DISCUSSION

GBS associated with primary VZV or chicken pox infection has characteristic a demyelinating pattern on nerve electrophysiology, sensory-motor neurologic involvement, and favorable clinical outcome. This is remarkable, considering patients with GBS have the axonal form of GBS. Although muscle weakness at nadir was severe, the outcome at 1 year was favorable for all seven patients even in the absence of specific treatment; in comparison, patients with C. jejuni associated GBS in this region have a poor outcome .The presence of anti VZV IgM antibodies and absence of anti-GM1 and C. jejuni related LOS antibodies in our seven patients and absence of anti-VZV IgM antibodies and presence of anti-GM1 and C. jejuni related LOS antibodies in patients with non-VZV-associated GBS provides strong evidence of an etiological role for VZV in GBS. The systematic review revealed most other reported cases of GBS after VZV infection also had the sensory-motor type at clinical presentation (> 50%) and neurophysiological evidence of demyelination.Varicella and shingles are the two clinical presentations of VZV infection. Among patients with GBS, both presentations of VZV were mostly associated with AIDP, the demyelinating form.In contrast to AMAN, the pathogenesis of AIDP is largely unknown. Most patients with AMAN have preceding C. Jejuni infections.AIDP may be mediated by cytotoxic T cells. The CD4+ T cell subpopulation also differs in patients with AIDP compared to the axonal subtype. The role of T cells in the pathogenesis of both AIDP and VZV infection is especially intriguing given the association between the demyelinating form of GBS and VZV. VZV is known to preferentially infect CD4+ memory T cells (with specific tissue-homing characteristics) in the lymphoid tissues of the upper respiratory tract, then T cells transport the virus to the tissues that are ultimately infected. Current evidence suggests VZV actively remodels T cells into activated specific tissue-homing infected T cells. During the remodeling process, VZV induces a spectrum of changes inthe surface and intracellular proteins within heterogeneous CD4 and CD8 T cell populations. In addition, VZV can directly infect the peripheral nerves, which may produce the neurophysiologic features of peripheral nerve demyelination. However, polyradiculoneuritis due to infection of nerve roots or nerves by VZV is unlikely, since weakness did not coincide with the skin rash in any case and none of the patients had an increased CSF cell count. Immune-mediated demyelination is also possible in other acute and mono or polyphasic neurological disorders preceded by VZV infection, including acute disseminated encephalomyelitis, acutetransvers myelitis (ATM) , or the relapse phase of multiple sclerosis, suggesting VZV has immune-mediated demyelinating potential.In terms of exploring the pathogenic role of VZV in GBS,our study has several limitations. First, we did not perform PCR for VZV in CSF or serum and hence cannot completely exclude direct infection of the nerves by VZV. Second, we didnot carry out complete screening of infectious etiologies thatmay trigger GBS (apart from VZV and C. jejuni and third, we did not perform lymphocyte subset assays to further explore the role of cell-mediated immune mechanisms in the pathogenesis of GBS. In conclusion, this study demonstrates VZV is associated with the demyelinating form of GBS, distinct to the C. Jejuni related axonal variants.Further studies are required to explore the mechanisms by which VZV infection leads to damage to peripheral nerve myelin.



MRI brain with a MRA MRV

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