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Case report *Medical research*

Herpes zoster and multiple sclerosis: case study and short review

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ABSTRACT

A 37 year old man developed typical varicella zoster virus (VZV) infection affecting the left clavicular area (C4 dermatome). Subsequently, he experienced numbness over the left scapula, arm, and palm. This improved gradually over one and half months. Two months later, he had numbness affecting the right thigh. MRI showed ovoid lesions in the corpus callosum and hyperintense signal in the cervical cord area. Based on the clinical features of presence of CNS lesions that were disseminated in time and space with no better explanation for the disease process, multiple sclerosis (MS) was diagnosed. The patient was treated with oral azathioprine, gabapentin and amitryptyline and patient improved symptomatically . A brief literature review was conducted on the etiological link between VZV infection and MS, early diagnosis, and early treatment of MS. The ultrastructural finding of viral particles identical to VZV, together with the simultaneous presence of large quantities of DNA from VZV in the CSF, almost restricted to the periods of exacerbation, as well as its steady diminution and eventual disappearance from clinical relapse to clinical remission constitute the strongest evidence to date to support the participation of VZV in the pathogenesis of MS. Suppression of relapsing phases of the disease with immuno-modulatory treatment is the present day management consensus.

Keywords: Multiple Sclerosis, Varicella-zoster virus (VZV), Human herpesvirus (HHV), Numbness, Paraesthesia, MRI, Corpus callosum, Cervical cord, Dawson fingers.

CASE REPORT

A 37 year old male who is an AutoCAD designer had a VZV eruption in the left clavicular area (C4 dermatome). The vesicles healed with symptomatic treatment. Ten days later he experienced numbness over left scapula, arm, and

palm (C8 dermatome), which improved gradually over a period of one and half months. Two and half months later, which was four months since the beginning of the VZV eruption, he returned complaining of numbness over the right thigh (T2 dermatome). General and neurological examinations in each of the episodes were unremarkable. The initial eruption was clinically diagnosed to be due to VZV. MS was considered to explain the two episodes of numbness. Efforts were made to rule out other conditions which can present with numbness namely, systemic lupus erythematosus, structural lesion of the brain and spinal cord; and B12 deficiency.

Complete blood count, renal function tests, liver function tests, blood sugars, thyroid function tests, electrolytes , vitamin B12, were normal. X-rays of the cervical spine and lumbosacral spine were reported as normal. Complement levels – C3, C4, Anti Nuclear Antibody (ANA), Anti Neutrophilic Cytoplasmic Antibody (ANCA), VDRL, herpes simplex virus & enterovirus Polymerase Chain Reaction (PCR) were negative. X – Ray cervical and lumbosacral spine, Electro Physiological Study (EPS), Electromyography (EMG) and Nerve conduction studies were within normal limits.

Magnetic Resonance Imaging (MRI) of the brain and cervical spine (pre and post contrast) was done and the sagittal T2 – weighted FLAIR (fluid attenuated inversion recovery) image of the brain and corpus callosum showed Dawson fingers. These are ovoid lesions perpendicular to the ventricles. They are indicated by white arrow in Figure 1. Sagittal T2 - weighted MRI image showed hyper intense signal over cervical cord area. This is indicated by white arrow in Figure 2. CSF - studies were within normal limits. A final diagnosis of VZV eruption of left C4 dermatome with multiple sclerosis with plaques in corpus callosum and cervical cord was made.

The patient was treated empirically with oral azathioprine to reduce the inflammatory response. Gabapentin and amitryptyline were also given for symptomatic treatment. Interferon therapy was offered but he was not keen. Currently, the patient is being followed-up for monitoring of progress and further episodes of numbness.

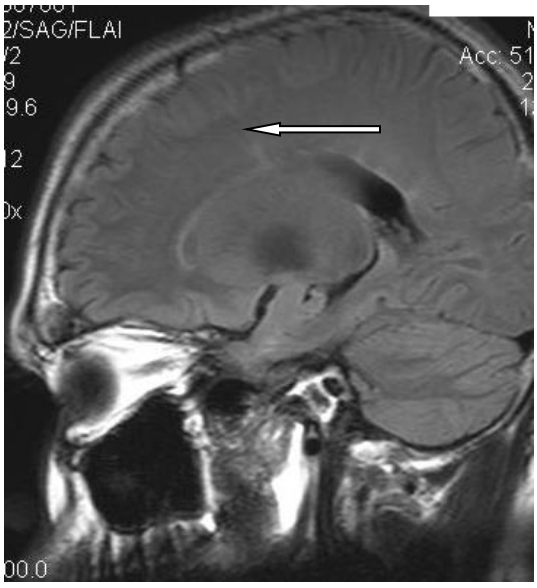

Figure 1: MRI image showing Dawson's fingers

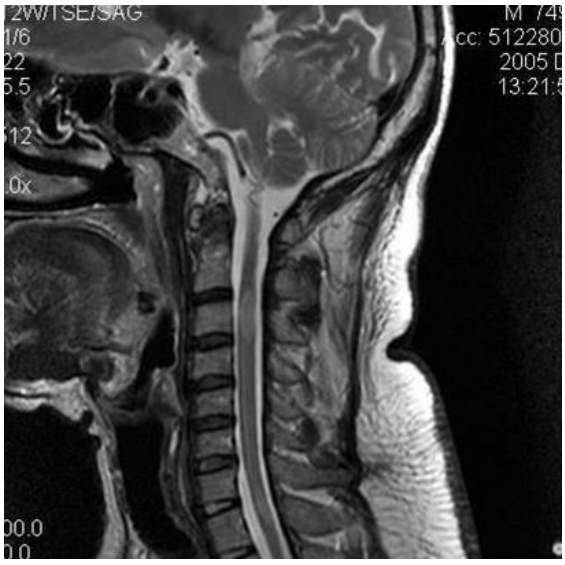

Fig 2: MRI of Cervical spine showing hyperintense signal

DISCUSSION

Multiple sclerosis is a progressive, disabling neurological disease with a variable clinical course [1]. Inflammatory and neurodegenerative processes appear to occur in parallel, with the former driving clinical manifestation of disease in the early, relapsing phase and the latter driving the accumulation of irreversibility seen later in the disease [1-3]. A viral etiology has been considered since the 1960s [4-5]. A study based on history of viral infections in 50 consecutive cases of multiple sclerosis with 50 matched controls for sex, race, and age within five years found herpes zoster to be the only statistically significant in patients with MS compared to controls [5]. In 633 patients there were 106 MS patients with a history of herpes infection before they had MS compared to 42/616 in the control group of other neurological disease. [6]. In a series of 85 cases of MS reported by Perez-Cesari et al, there were six cases of VZV preceding the MS. [7].

The human herpesvirus (HHV) are likely candidates as the viral links to MS because they are highly prevalent worldwide, they all cause latent infections and are all capable of reactivation. Epstein-Barr virus (EBV), HHV-6A, and varicella zoster (VZV) have been consistenly linked with MS, particularly with respect to epidemiology, antibody responses in serum and cerebrospinal fluid antibodies. MS relapses are associated with EBV and HHV-6A and VZV titres viral reactivation [8].

The latest implications of VZV as the causal virus in the virus-MS link come from the studies of Sotelo et al [9]. They found on electronic microscopy, abundant viral particles identical to VZV in CSF from 15 MS patients within the first few days of an acute relapse. In contrast, viral particles were not seen in CSF samples from the 19 MS patients in remission or from the 28 neurological control subjects. Also, DNA from VZV was present in CSF and peripheral blood mononuclear cells (PBMC) during relapse, disappearing in most patients during remission. The mean viral load was 542 times greater in CSF at relapse than in CSF at remission and 328 times greater in CSF at relapse than in PBMCs at relapse. These findings constitute the strongest evidence to support the participation of VZV in the pathogenesis of MS.

About 85% of MS cases originate with relapsing-remitting disease (Relapsing-remitting MS, or RRMS), which moves through periodic exacerbations with subsequent full or partial recovery before entering the progressive phase, in which any recovery of function is rare (secondary progressive MS, or SPMS). In about 15% of MS patients, the disease is progressive from onset and they rapidly disabled (primary progressive MS, or PPMS).

Consensus treatment guidelines now recommend initiating treatment as soon as a diagnosis has been made. If the patient presents with a typical of MS and alternative diagnoses have been excluded, the CSF is tested, and a MRI evaluation is done to identify possible subclinical disease activity and to help confirm the diagnosis. If these procedures indicate a high probability of the patient developing MS, immuno-modulatory treatment is considered. [1].

First line therapy is recommended either with a beta-interferon preparation or with glatiramer acetate. Second line treatment where the first two medications cannot be used would be azathioprine or intravenous immunoglobulins. [1].

In the early stages of treatment (the first year), the patient should be monitored every three months to evaluate any changes in disease activity, emergence of possible side effects and treatment adherence. Monitoring patients regularly with a standardized clinical assessment tool such as the Expanded Disability Summary Scale (EDSS) is warranted. Natalizumab or mitoxanthrone is recommended for patients with severe disease chacterised by two or more severe relapses per year. The recommended treatment of acute relapse is a corticosteroid pulse therapy with intravenous methylprednisolone or, in severe cases which fail to respond to corticosteroid therapy, plasma exchange therapy [1, 18, 26].

CONCLUSION

We report a patient who presented with VZV eruption in the left clavicular region followed two episodes of numbness of the body. The preceding episode of VZV followed by numbness highlights the VZV and MS link in this patient. A short review was conducted on the etiological link between VZV infection and multiple sclerosis (MS), diagnosis and management of early MS.

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