Case Report 1:Guillain Barré Syndrome Post Primary Varicella Infection

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ABSTRACT

Guillain-Barré syndrome (GBS) is a critical condition that usually arises as a late complication of certain infections. Varicella zoster is (VZ) an extraneous antecedent infection that can cause GBS. We report a rare case of GBS following primary VZV infection in an adult.

INTRODUCTION

Guillain-Barré syndrome is an acute demyelinating polyneuropathy, which is autoimmune in nature. Most of the cases follow an antecedent respiratory or gastrointestinal illness one to three weeks after the onset of symptoms. Usual antecedent infections include *Camylobacter jejuni*, Cytomegalovirus and Ebstein-Barr virus. Other causes include HIV, *Mycoplasma* pneumonia, Lyme disease and lymphoma. Varicella zoster is one of the uncommon causes of GBS and when it does occur it is usually following reactivation of VZ. We are reporting a case of GBS following primary VZ infection.

CASE REPORT

A 28 years old Indian male patient presented to us with giddiness, progressive weakness of his extremities of 4 days duration. It began with difficulty in walking, getting up from the floor and then progressed to difficulty in lifting his hands within the next 2 days. Patient was diagnosed to have chicken pox ten days prior to the onset of these symptoms. On examination, he was afebrile, normotensive and had no respiratory distress. General physical examination revealed dry rash over his trunk and limbs suggestive of recent chicken pox infection. Neurological examination showed reduced muscle tone in all limbs with weakness of all muscle groups. Reflexes were absent and sensory system examination was unremarkable. He also had bilateral abducens paresis and bilateral facial palsy. There was no involvement of other cranial nerves. The cerebellar signs were absent with mute plantar response. At presentation there was no neck or respiratory muscle weakness. There were no fundoscopic changes.
Laboratory investigations showed the following values; a westerngreen erythrocyte sedimentation rate of 95 mm/hr, CRP 12 mg/dl, Hb 11.9 g/dl; Hct 36.5%, WBC 8.5 x 109, 30% segmented neutrophils, 62% lymphocytes, 12% band forms, 4.3% monocytes, platelet 225 x 109, AST 39 IU/L, ALT 32 IU/L, GGT 15 IU/L, LDH 286 IU/L. Blood sugar was normal. Anti-varicella virus IgM antibody was positive. A brain CT was normal and a cerebrospinal fluid tap showed 28 red cells, 4 white cells, protein 200 mg/l, glucose 65mg/dl, and chloride 119.5meq/l. No virus or bacteria were isolated. A nerve conduction velocity test showed increase latency and decreased amplitude.

Patient was started on intravenous steroid. On day four of admission patient developed respiratory distress with an episode of tachycardia and hypertension. He was intubated and connected to ventilator. Blood pressure was controlled by propranolol. Patient was given intravenous immunoglobulins daily for five days. He showed signs

of improvement. Three weeks later the patient was extubated and transferred to ward. One week later he had improved from his neurological condition and discharged from hospital. In the end he recovered completely with no neurological deficits.

DISSCUSSION

This patient had all clinical features of GBS such as weakness, paresthesias, diminished and absent deep tendon reflexes. The finding of nerve conduction study and CSF examination also supported the diagnosis of GBS **[1-3]**.

Primary VZ infection can cause neurological complications such as myelitis, aspectic meningitis, vasculitis, optic neuritis, the most common being encephalitis 1:1000 **[4]**. GBS is the least common 1:15000 **[5]**. GBS following varicella zoster typically has a latent period of 2 weeks to 2 months. Shorter latent periods, as in this case, are associated with more severe complication **[6,7]**.

It is thought that steroid can reduce the severity of GBS, but controlled clinical trials have demonstrated that this treatment not only is not effective but it can have detrimental effect on the disease. The immunoglobulin therapy in GBS can lessen the immune attack on the nervous system. Several hypotheses don't know why or how this works **[8]**.

The most critical part of the treatment for GBS consists of keeping the patient's body functioning during recovery of the nervous system. This can sometimes require placing the patient on mechanical ventilatory assistance,a heart monitor and blood pressure. That's why Guillain-Barré syndrome patients are usually treated in hospitals, often in an intensive care ward **[9,10]**.

CONCLUSION

GBS can present as neurological complication of primary HZV. It should be treated by intravenous immunoglobulin or plasmaphoresis together with supportive care which will lead to good recovery in the majority of cases.

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