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Guillain-Barre' syndrome following Primary Infection with Varicella Zoster Virus in an Adult

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ABSTRACT

Introduction: *Guillain Barre' syndrome (GBS) is an acute demyelinating polyneuropathy. The exact pathogenesis is not known, but it is suggestive of an autoimmune mechanism. Varicella neurological complications are rare, encephalitis being the most common and GBS the least common. We report here a case of GBS following primary VZV infection ('chickenpox').*

Case report: *A 19-year old male patient was admitted who presented with progressive ascending weakness of his limbs since 9 days. It began with weakness of both lower limb, difficulty in walking, getting up from the bed, which progressed to difficulty in lifting his both upper limbs and later difficulty in breathing over the period of 5-6 days. Patient was diagnosed to have chicken pox approximately 2 weeks prior to the onset of these symptoms.*

Findings: *MRI Spine and CT Brain (P) done outside prior to admission, both where within normal limit. Nerve Conduction Velocity (NCV) study was done which was suggestive of Sensory + Motor Mixed Polyneuropathy (Axonal + Demyelinating), which confirmed our diagnosis of GBS.*

Conclusion: *Seronegative patients exposed to the virus may avoid symptomatic illness by taking prophylactic antiviral therapy of acyclovir after exposure to VZV, allows a viremia and seroconversion, but may prevent systemic and neurological disease, thus avoiding the heightened morbidity associated with VZV infection in adulthood.*

Key words: Guillain-Barre' syndrome (GBS); Primary varicella zoster virus, Nerve Conduction Velocity (NCV), IV immunoglobulin (IV IgG), acyclovir.

INTRODUCTION

Guillain Barre' syndrome (GBS) is an acute demyelinating polyneuropathy. The exact pathogenesis is not known, but it is suggestive of an autoimmune mechanism.[1,2] Preceding infection with various infectious agents may trigger the process. Viruses such as cytomegalovirus, campylobacter jejuni, ebstein barr virus and human immune deficiency virus have been convincingly associated with GBS. Other viruses such as varicella-zoster have been rarely reported.[3] Varicella neurological complications are rare, encephalitis being the most common (1:1000),[4] and GBS the least common (1:15000).[5-8] Fewer than 50 such cases have been reported in the literature, and, when it does occur, it is almost always in the context of reactivation disease from latent VZV, following herpes zoster ('shingles').[9] We report here a case of GBS following primary VZV infection ('chickenpox').

CASE REPORT

A 19-year old male patient presented with progressive ascending weakness of his limbs since 9 days. It began with weakness of both lower limb, difficulty in walking, getting up from the bed, which progressed to difficulty in lifting his both upper limbs and later difficulty in breathing over the period of 5-6 days. Patient was diagnosed to have chicken pox approximately 2 weeks prior to the onset of these symptoms. On examination, patient was conscious, co-operative and well oriented to time, place and person, afebrile,

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normotensive but patient had mild respiratory distress (Pulse- 102/min, BP- 100/ 70 mm of Hg, SpO2-92% on room air with RR-34/min). Patient had neck muscle weakness and single breath count (SBC) was 8. General physician examination revealed healing lesions over his trunk suggestive of recent chicken pox infection. Muscle tone was reduced in all four limbs with grade zero power in both upper and lower limbs. Reflexes were absent and sensory system examination was unremarkable. Mute plantar response was noticeable. Provisional diagnoses of Guillain-Barre' syndrome (GBS) was made and patient was admitted in our ICU care. Patient had got MRI Spine and CT Brain (P) done outside prior to admission, both where within normal limit. Patients urgent bed sided Nerve Conduction Velocity (NCV) study was done which was suggestive of Sensory + Motor Mixed Polyneuropathy (Axonal + Demyelinating), which confirmed our diagnosis of GBS. Patient was intubated and was taken on ventilator support in view of respiratory muscle weakness. Patient was started on INJ. IV Immunoglobulin (IV IgG) at dose of 0.4 gm/kg/day. Patients all routine lab investigations were done, which were as follows; Hb-17.3 gm/dl; TLC-10200/mm3; HCT-47.4 %; Platelets- 423,000/mm3, Urea- 43 mg/dl, Creatinine-0.7 mg/dl, Na^+-133meq/L, K^+-4.9 meq/L, CK total- 190 IU/L. Patient was started on IV antibiotics consisting of combination of piperacillin and tazobactum; and levofloxacin. Later patient developed right lung collapse for which bronchoscopy was done, thick purulent secretion and mucous plug was removed. Patient was advised daily chest and limb physiotherapy. Patient completed 5 days of IV IgG therapy, patient had improvement in muscle power of both upper limb and lower limb. Patient's right lung collapse resolved and after 8 days patient was extubated. By this time patient had grade 3 power in both upper limb and grade 2 power in lower limb. Patient was shifted in ward and daily physiotherapy continued. Patient's anti-VZV antibody IgM was send after 20 days which was negative. Patient had good neurological recovery and patient was discharged after 28 days with grade 5 power in all four limbs.

DISCUSSION

This patient has all the cardinal clinical features of GBS; weakness, paresthesias, and diminished or absent deep tendon reflexes.[10] VZV, however, is a rare antecedent for GBS. In a study examining antecedent infections in GBS, Jacobs et al. found only one case out of 154 that could be attributed to VZV.[3] Of the fewer than 50 reported cases in the literature, only six involved primary infection and of these none involved adults.[5-6,11-12] In 302 cases of GBS studied by Samantray et al. only one (0.3%) was related to chickenpox.[13] Depending on the pattern of involvement (axonal, sensory or autonomic), patients generally improve over several days. It is thought that the well-described association of GBS with Campylobacter jejuni infection arises because the lipopolysaccharides of these organisms contain epitopes that are similar to peripheral nerve gangliosides. They act as 'molecular mimics', driving production of antibodies with anti-ganglioside specificity, which result in injury to peripheral nerve fibers.[14] Whether a similar 'molecular mimicry' occurs with post-VZV GBS remains speculative. Such antibody generation may occur at higher titers and more frequently in the secondary response associated with VZV reactivation (shingles), but this case suggests that primary VZV infection per se may sometimes be a sufficient stimulus to drive antibody generation and precipitate severe clinical symptomatology.

CONCLUSION:

Treatment is with IV immunoglobulin or plasmapheresis along with ventilator support when needed.[15-16] Seronegative patients exposed to the virus may avoid symptomatic illness by taking prophylactic antiviral therapy. A one-week course of acyclovir at treatment doses, starting one week after exposure to VZV, allows a viremia and seroconversion, but may prevent systemic and neurological disease, thus avoiding the heightened morbidity associated with VZV infection in adulthood.[17-18] Understanding the activating factors as in this case post-varicella infection, may be helpful for understanding the pathogenesis, the prevention of neurological involvement and is important for planning follow up and understanding the prognosis.

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