

Unveiling the Connection: A Case Report on GBS Following Varicella Zoster Infection

Jeeva Elizabeth Thomas¹, Breezy Ani Sam², Dr. Hannah Joseph³, Dr. Anilkumar Sivan⁴

¹Pharm D Intern, Nazareth College of Pharmacy, Othara, Thiruvalla, India

²Pharm D Intern, Nazareth College of Pharmacy, Othara, Thiruvalla, India

³Clinical Pharmacist, Department of Neurology, Believers Church Medical College Hospital, Thiruvalla, Kerala, India

⁴Consultant, Department of Neurology, Believers Church Medical College Hospital, Thiruvalla, Kerala, India

Abstract: *GBS short for Guillain - Barre Syndrome, is a rare condition that affects the peripheral nervous system. It can sometimes occur after a varicella zoster infection, which is the virus responsible for chickenpox. GBS is a condition where the immune system mistakenly attacks the nerves, leading to muscle weakness and even paralysis. When it happens after a varicella zoster infection, it's known as GBS post varicella zoster. This case report presents the intriguing clinical scenario of a patient who developed Guillain - Barre syndrome (GBS) following to a varicella zoster virus (VZV) infection. The patient was presented with prodromal symptoms of difficulty in walking and loss of balance while walking followed by bilateral palm numbness, and a characteristic rash associated with VZV.*

Keywords: Guillain - Barre syndrome, varicella zoster virus

1. Introduction

Guillain - Barre syndrome (GBS) is a rare neurological disorder that affects the peripheral nervous system as the immune system attacks the nerves. Ascending muscular weakness, hypore - exia, cranial nerve dysfunction, and sensory abnormalities are its defining characteristics. GBS typically develops after an infectious disease. The development of antibodies against microorganisms that cross - react with specific gangliosides and glycolipids found throughout the myelin is most likely the cause. The neurotropic herpes virus known as varicella zoster virus (VZV) causes shingles, which is an expression of a latent disease reactivation, in addition to chickenpox ^[1]

When someone is infected with VZV, which causes chickenpox or shingles, the immune system responds by producing antibodies to fight the virus. In some cases, these antibodies mistakenly attack the body's own nerve cells, leading to inflammation and damage. This immune response can result in the development of GBS. This is how a person with chickenpox can develop Guillain - Barre syndrome.

It's important to note that not everyone who contracts VZV will develop GBS, and the exact mechanisms behind this association are still being studied. However, recognizing the potential link between VZV and GBS can aid in early diagnosis and appropriate management of the condition.

Diagnosis of GBS is done by a combination of tests, including a physical examination, nerve conduction studies, and a lumbar puncture to analyse cerebrospinal fluid. These tests help evaluate muscle weakness, reflexes, and nerve function. Additionally, blood tests and imaging studies may be conducted to rule out other possible causes.

It is necessary to recognize the neurological complications following viral infection associated with GBS as it is a

crucial step for proper diagnosis and treatment. Initiation of treatments, such as IVIG or plasmapheresis, can help mitigate the progression of GBS and improve the patient's overall outcome.

This paper will discuss case of a 44 year old male who was diagnosed with Guillain - Barre syndrome post varicella zoster virus infection.

2. Case Report

A 44 year old male patient with no known comorbidities was apparently normal. Then he developed rashes initially on face later spreads to all over body – diagnosed with chicken pox. Later he developed difficulty in walking and loss of balance while walking followed by bilateral palm numbness. Since 2 days, he found difficulty in closing left eyelid with right side facial deviation and pain over occipital region. His general examination was unremarkable. His neurological examination revealed incomplete closure of left eyelid, absence of forehead wrinkling left side, mild facial deviation to right side. This shows significance of facial nerve palsy. Reflexes were also absent. Bifacial weakness was positive. Power in upper limbs 5/5 and in lower limbs 3/5 showed muscle weakness in both lower limbs and bilateral facial muscles. Cerebrospinal fluid analysis revealed CSF protein was 143.60 mg/dL, CSF glucose was 67mg/dL, CSF ADA 4.70 U/L. Other biochemical and haematological investigations were within normal limits. Motor nerve conduction studies were normal from bilateral facial nerves. Blink reflex study from left orbicularis oculi showed absent ipsilateral R1, R2 and normal contralateral R2. Right side stimulation showed normal ipsilateral R1, R2 and contralateral R2. This electrophysiological study is suggestive of left LMN facial palsy. NCS of all 4 limbs suggestive of AIDP. CSF study showed albumino - cytological dissociation. A diagnosis of GBS was made after utilizing the criteria that strongly support the diagnosis of

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Guillain - Barre Syndrome. At the admission, acyclovir based therapy (500mg IV) was started Twenty - four hour after admission, treatment with intravenous immunoglobulin (IVIG: 2g/kg/d for 5 days) was started for the patient. After nine days, progressive neurological improvement occurred. He was then discharged with normal neurological examination.

3. Discussion

GBS is a significant acquired illness that is immune - mediated, inflammatory, and rapidly changing in the peripheral nervous system. As it advances, axonal atrophy and demyelination occur. Clinical features include symmetrical flaccid muscular paresis, areflexia in the presence of elevated protein level in the CSF fluid, and electrophysiological investigations showing progressive demyelination. ^[3]GBS is a complex degenerative neurological condition that can be manifested as either an acute or a chronic condition. It is an acquired condition that manifests as symmetrical, gradual tingling and weakening in the proximal and distal extremities. GBS was diagnosed due to suggestive clinical presentation and typical CSF albuminocytological dissociation. A hallmark of GBS is the presence of elevated protein and in our patient CSF protein was found to be 143.60 mg/dL. Usually the onset of GBS has a latency period of two weeks to one month after preceding infection, and shorten latency period are associated with more severe illness. For our patient after exposure to chickenpox within 10 days difficulty in walking was noted. Facial nerve palsy was noted in our patient which was similar to Sorour Inaloo et al. in which they have GBS with bilateral facial nerve palsy. Fewer cases of VZV - related GBS were reported. ^[2] Only a few case reports have described the development of GBS following chickenpox. Our case report is similar to P. Tatarelli et al. which shows improvement in patients after the treatment with intravenous immunoglobulins. ^[1]

4. Conclusion

In conclusion, Guillain - Barre syndrome (GBS) is typically an immune - mediated condition that develops after an infection. Numerous triggers have been identified, including varicella zoster virus (VZV) infection is known to have the potential to cause GBS. Less than 50 such cases have been documented in the literature, making this a rare occurrence. When it does happen, it almost often follows herpes zoster (also known as "shingles") and occurs in the setting of latent VZV reactivation illness. ^[4] While the exact mechanisms are still being studied, it is believed that an autoimmune response to VZV can lead to nerve damage and the development of GBS. It is extremely important to identify and urgently refer, potential severe cases in order to have the appropriate investigation and appropriate management. This report highlights the association between VZV and GBS, emphasizing the importance of recognizing and managing neurological complications following viral infections.

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Conflict of interest

The authors declare that the case report was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Abbreviations:

GBS: Guillain - Barre Syndrome

VZV: Varicella Zoster Virus

IVIG: Intravenous Immunoglobulin

CSF: Cerebrospinal Fluid

AIDP: Acute Inflammatory Demyelinating Polyradiculoneuropathy

LMN: Lower Motor Neuron

NCS: Nerve Conduction Study

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