

Case Report

Guillain–Barre Syndrome: A Rare Presentation of Borderline Tuberculoid Leprosy with Type 1 Lepra Reaction

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ABSTRACT

Guillain–Barre syndrome (GBS) is an autoimmune polyradiculoneuropathy usually preceded by respiratory tract or gastrointestinal infection. The pathogenesis in GBS is based on molecular mimicry mechanism. Hansen’s disease is common in India and is the most common infectious cause of neuropathy. We describe a 42-year-old man who was being treated for borderline tuberculoid leprosy and developed Type 1 lepra reaction followed by GBS and responded to plasmapheresis. Lepra reaction may lead to exposure of neural antigens, resulting in autoimmune mechanism and demyelination of peripheral nerves.

KEYWORDS: *Guillain–Barre syndrome, lepra reaction, leprosy, neuropathy*

INTRODUCTION

Guillain–Barre syndrome (GBS) is an autoimmune polyradiculoneuropathy which is characterized by acute-onset quadriparesis with cranial nerve involvement and areflexia on examination. It is usually preceded by respiratory tract or gastrointestinal infection. *Campylobacter jejuni*, *Mycoplasma pneumoniae*, *Cytomegalovirus*, HIV, Epstein–Barr virus, and vaccination are common agents implicated for GBS.^[1] We describe a 42-year-old male patient with a known case of borderline tuberculoid leprosy who developed GBS following the development of Type 1 lepra reaction.

CASE REPORT

A 42-year-old man who works in a rubber factory developed reddish-colored lesions over the inner aspect of the left forearm, left elbow and outer aspect of the left arm, which were raised above the surface of the skin and were nonitchy and painless and associated with diminution of sensation [Figure 1]. The patient was diagnosed leprosy and was on regular treatment for the past 3 months. Six days before admission, he developed paresthesia (pin and needle sensation) in the left hand followed by the right hand and subsequently in both the lower limbs within 1 day. On the 2nd day of illness, he needed support to stand from the sitting position and started dragging his left feet while walking. After

1 more day, he buckles his knee while walking and could walk only with support of one person and also developed weakness of the upper limb on the next day. He could not comb his hair and could not grip a glass of water. On presentation, he was unable to get up from lying to sitting position and unable to turn side to side in bed. There was no cranial nerve abnormality and no bladder or bowel complaints. There was no history of recent cough or loose stools before the illness. No new skin lesions have appeared, but there was development of inflammation in the existing skin lesion.

On examination, he had hypoesthetic patch on the above-mentioned sites with bilateral thickened and tender ulnar and common peroneal nerve. Cranial nerves were normal. Power in the upper limb was Medical Research Council (MRC) grade 3/5 both proximally and distally with a hand grip of 5%–10%. Power in the lower limb was MRC grade 2/5 proximally and 4/5 distally. Deep tendon reflexes were absent globally. Sensation was decreased (pain, touch, temperature) around 60% on the patch and vibration was impaired at toes bilaterally.

On investigations, he had hemoglobin of 14.23 g/dl, blood sugar (fasting) of 106 mg/dl, and serum creatinine

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Figure 1: Figure showing skin lesions in the left upper limb

of 0.7 mg/dl. Vasculitis markers were negative. Skin biopsy was suggestive of borderline tuberculoid type of leprosy. Nerve biopsy was done from the right sural nerve which was within normal histological limits and was negative for acid-fast bacilli. Nerve conduction study was suggestive of acute motor axonal neuropathy variety of GBS. Cerebrospinal fluid (CSF) was showing albuminocytological dissociation with nil cells and protein 92 mg/dl. He was given methylprednisolone 1 g daily for 5 days with no improvement, so he was treated with plasmapheresis five cycles after which there was a significant improvement in power. At the end of five cycles, he was able to sit up with some difficulty in bed, could turn side to side and lift both arms overhead, and could raise and sustain both legs in the air against gravity. His distal power in the upper limbs did not improve. The patient was thus discharged in a stable state and was able to walk 10–15 m with the support of one person. After 1-month follow-up, he was able to walk independently with persistent weakness in the distal upper limbs.

DISCUSSION

GBS is a disease characterized by acute-onset quadriparesis, cranial nerve palsy with areflexia, and albuminocytological dissociation in CSF. The pathogenesis in GBS is based on molecular mimicry mechanism in which immune mechanism damages the host nerve tissue because of resemblance of epitopes. Hansen disease is common in India and is the most common infectious cause of neuropathy. Nerve involvement in leprosy is in the form of mononeuropathy or mononeuritis multiplex. Small fibers are mainly affected, but large fibers can be affected later in the course. There is a possibility that GBS has occurred incidentally in the presence of leprosy. However, the presence of lepra reaction makes

it unlikely. GBS in a patient with lepromatous leprosy with Type 2 lepra reaction has been described.^[2] High level of tumor necrosis factor-alpha (TNF- α) has been found in patients of lepromatous leprosy developing Type 2 lepra reaction.^[3] TNF- α may act by immune mechanism and cause demyelination of peripheral nerves resulting in GBS.^[4] However, in our case, it was Type 1 lepra reaction that triggered autoimmune phenomenon resulting in GBS. Lepra reaction may lead to exposure of neural antigens resulting in autoimmune mechanism and demyelination of peripheral nerves.^[5,6] However, GBSs have been described rarely in patients of leprosy even without lepra reaction.^[7,8] It is very important to differentiate lepra reaction-induced neuropathy from GBS as early treatment is required in GBS and also the two conditions required different treatments. With leprosy being common in India, further studies are needed to establish the association between leprosy and GBS, and also neurologists should know this rare association to prevent delay in treatment.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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