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Post-treatment Guillain-Barre Syndrome in a Patient with Brucellosis; A Case Report

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ABSTRACT

Introduction: Guillain-Barre Syndrome is an uncommon complication during acute brucellosis. **Case presentation:** In this study, we present a case of Guillain-Barre Syndrome in a 22-year old male patient with complaints of weakness in his lower limbs. He had a history of acute Brucella infection for four months and received antimicrobial medication. **Conclusion:** the patients can be affected by GBS after antimicrobial treatment

INTRODUCTION

Guillain-Barre Syndrome (GBS) is a neurologic disorder mostly appears with acute paralysis and peripheral neural deficit. The symptoms are symmetrical with progressive severity over the time. It is common among men and its prevalence increases in older populations (1). Post-infectious immune-mediated polyneuropathy is the most common form of the disease; however, several variants of GBS based on its clinical presentation and pathophysiological aspects also exists. Acute motor and sensory axonal neuropathy (AMSAN) are one of those variants present with severe loss of function in both motor and sensory fibers with marked axonal degeneration. Various etiologies and pathogens have been proposed as the underlying factors leading to GBS (2); however, reports of GBS incidence after brucellosis are very rare. Here, we present a case of GBS in a patient under treatment of brucellosis.

CASE PRESENTATION

A 22-year-old male patient was presented to the emergency ward of our hospital with a complaint of acute weakness in his legs. The weakness had started two days prior to the admission and had progressively worsened. Upon admission, the patient was not able to stand on his feet, but was able to use his arms completely without any respiratory difficulties. He had no history of respiratory infections or diarrhea before admission and mentioned no other accompanying symptoms other than a recent weight loss of over 8 kg. Family history for diseases was unremarkable, but in the medical history he mentioned that he had been hospitalized 4 months earlier due to fever, chills, nausea, vomiting, and weight loss. The medical records showed a diagnosis of acute brucellosis due to the consumption of unpasteurized dairy products. The patient was treated with antibiotics, like rifampin and doxycycline.

He was living in a rural area near Isfahan, a large province in central Iran. Upon examination, he was awake and responsive. His vital signs were normal with a blood pressure of 110 over 70 mmHg, pulse rate of 68 beats per minute, 95% oxygen saturation level, and 36.8˚C body temperature. Systemic examinations yielded no remarkable abnormalities except a significant proximal and distal muscle wasting in the lower limbs. Neurological examination of the cranial nerves showed to be intact. No dysarthria, dysphagia, or

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diplopia were reported by the patient. Sensory and motor functions of the upper limbs were normal. Touch and position sensation of the lower limbs were also normal, but motor function of both the lower limbs were limited, with a muscle strength ranging from one to two out of five. Muscle tone also decreased in both legs. Deep tendon reflexes were normal in the upper limbs but absent in both legs. Bilateral plantar reflexes were normal. There were no sensory levels or urinary/anal sphincter abnormalities.

Investigations included a normal chest X-ray and normal electrocardiogram along with sinus rhythm. Blood cell counts showed a mild decrease in white blood cells (3700 cells/mm3) with a higher percentage of lymphocytes (53%) and mild thrombocytopenia (136000 cells/mm3); hemoglobin levels were normal (14.9 g/dL). Liver (SGOT: 18 IU/L) and kidney (blood urea nitrogen: 13 mg/dL, creatinine: 0.9 mg/dL) function profiles were normal. Thyroid function tests were unremarkable (TSH: 1.08 IU/mL, normal range: 0.3-5.5; T4: 0.9 ng/dL, normal range: 0.6 – 1.6). Other biochemical tests including blood glucose (82 mg/dl), sodium (141 mEq/L), potassium (3.9 mEq/L), calcium (9.4 mEq/L), and phosphorous (3.5 mEq/L) were all within the normal range. The patient had an increased creatine phosphokinase level of 251 IU/L, and the level of lactate dehydrogenase was 271 IU/L. The level of C reactive protein was normal (0.6 mg/dL), and the serologic tests for brucellosis were negative.

Analysis of the cerebrospinal fluid (CSF) yielded the following results: glucose 30 mg/dL (blood glucose 75 mg/dL), protein 140 mg/dL, normal range: 15-45 (blood: 5.4 g/dL), LDH 26 IU/L (blood LDH 227 IU/L), and no red/white blood cells.

Magnetic resonance imaging of the brain and spine was normal, and we did not find any space-occupying lesions, disturbances in the grey-white matter interface, cerebral midline shift, signs of inflammation, or CSF blockage.
Further investigation by electromyography and neural conduction velocity studies reported a subacute severe peripheral axonal sensorimotor neuropathy with greater severity in the lower limbs. This result suggested an acute motor sensory axonal neuropathy (AMSAN) or GBS with secondary axonal features as the underlying cause for the patient's symptoms.

With the diagnosis of GBS, secondary to acute brucellosis, we conducted 8 rounds of 1 hr -long plasmapheresis sessions for the patient. His weakness subsided gradually and functional activities of his legs resumed. He did not develop any new symptoms during the course of treatment. He was discharged with an improvement in his general conditions and followed up regularly. He remained symptom-free with minor weakness in his legs and was referred to a rehabilitation clinic for further treatments.

DISCUSSION

The immune responses leading to GBS are often preceded by an episode of previous illness such as upper respiratory tract infections and gastroenteritis. The pathogens responsible for GBS include mycoplasma, cytomegalovirus, Ebstein-Barr virus, vaccinia, Campylobacter, varicella, measles, mumps, influenza, and type A and B hepatitis; however, there have been few reports of Brucella as a cause of GBS (3). Active brucellosis usually affects the gastrointestinal, hepatobiliary, and skeletal muscles during the course of the disease; nervous system involvement is not a common outcome of the disease (4).

While some studies claim the prevalence of neuro-brucellosis to be 3% to 25% among the patients with brucellosis (4), others report only 1.7% of the cases having symptoms of nervous system involvement. Brucellosis can affect both central and peripheral nervous system manifesting various symptoms including headaches, meningoencephalitis and encephalopathy, papilledema, and inflammatory demyelinating syndromes. GBS is reported to be an uncommon complication of this disease. In a study by Elzein et al., out of 1028 cases of active brucellosis, 58 patients had symptoms of nervous system involvement; only two of these patients were positively diagnosed with GBS (5).

The Brucella-induced GBS may present as simple peripheral demyelination of neurons or involve axons and cause polyneuropathy with axonal damage. In a report by Garcia et al., both types of GBS were observed in patients with neuro-brucellosis; however, axonal damage was accompanied by less favorable outcomes and worse prognosis after treatment (6). In contrast, Montalvo et al. reported a case of GBS during acute brucellosis with pure axonopathy that responded well to antimicrobial and plasmapheresis treatments (7).

Brucella canis and Brucella melitansis have been reported as the underlying agent for GBS previously(8, 9); however, in a study by Watanabe et al., a molecular resemblance was found between the lipopolysaccharide of Brucella melitansis and the GM-1 ganglioside. Anti-GM-1 antibodies showed high affinity for surface molecules in Brucella melitansis. Interestingly, when immunized with Brucella melitansis, the immunized mice showed a significant cross-reaction with the GBS-related Campylobacter jejuni. Moreover, the serum levels of anti-GM-1 antibodies were significantly higher in the samples infected with Brucella melitansis compared to those immunized with Campylobacter jejuni, indicating a stronger immune response to Brucella melitansis (10).

In contrast to all the cases reported GBS during the acute course of brucellosis, our report presented a case with a history of brucellosis and signs of nervous system involvement after proper anti-microbial treatments. He had a history of delayed GBS along with the nervous system involvement included axonal damage. In addition to the previous antimicrobial treatment, plasmapheresis usage resulted in a satisfactory outcome despite the presence of axonopathy.

CONCLUSION

The present case highlights Brucella as a possible cause of GBS in endemic areas, which agrees with previous reports.

Furthermore, we showed that the patients could be affected by GBS even after the acute illness is treated with antimicrobial agents and late onset of nervous system involvement is a probability that should be kept in mind.

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AUTHOR CONTRIBUTIONS

VS and NV contributed to the conception of the work, conducting the study, revising the draft, and approval of the final version of the manuscript.

SB and OM revised the draft and approved the final version of the manuscript.

CONFLICT OF INTERESTS

On behalf of all authors, the corresponding author declares there is no conflict of interest. This study was carried out without any financial support from any private or public institution.

ETHICAL STANDARDS

NONE

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