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Case Report

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Guillain-Barre Syndrome Associated With Pulmonary Tuberculosis

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Abstract

Guillain-Barre syndrome is the commonest cause of neuromuscular paralysis. Antecedent infections, mainly bacterial and viral are common and are thought to play a role in triggering the accompanied immune response. We report the case of a 21-year-old man who presented with clinical features of Guillain-Barre syndrome and was found to have pulmonary tuberculosis. Review of the literature revealed only a few similar cases. We wish to alert clinicians to the possible association between Guillain-Barre syndrome and tuberculosis.

Keywords: Guillain-Barre syndrome; Pulmonary; Tuberculosis

Introduction

Guillain-Barre syndrome has become the most common cause of acute neuromuscular paralysis after the global eradication of poliomyelitis. The global incidence of Guillain-Barre syndrome ranges from 0.4 to 4.0 cases per 100,000 people annually, occurring slightly more often in adolescents and young adults than in children. The disease tends to affect males more than females with a ratio of 1.78 [1].

Antecedent infections are common with Guillain-Barre syndrome and are thought to trigger the immune response that leads to acute polyneuropathy [2]. The most frequent preceding symptoms are fever, cough, sore throat, nasal discharge, and diarrhea. The most frequently identified cause of infection is Campylobacter jejuni. Other well-defined infections related to Guillain-Barre syndrome are cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae, and Haemophilus influenza [3]. To note, less than 1 in 1000 patients with Campylobacter jejuni infection will develop Guillain-Barre syndrome. Host factors might influence susceptibility to Guillain-Barre syndrome or the extent of nerve damage and outcome.

We report the case of a patient who presented with the clinical features of Guillain-Barre syndrome and was found to have pulmonary tuberculosis and discuss the possible association between these two conditions.

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Case Report

A 21-year-old man presented with a 4-day history of lower limb weakness rendering him unable to walk. The weakness started suddenly, initially affecting the feet then it progressed to involve both legs. There was no upper limb weakness, neck stiffness, stool or urinary incontinence or sensory deficits. The patient reported cough that started a few days prior to the onset of the weakness. There was no associated fever, shortness of breath, headache, vomiting or diarrhea. There was no history of similar episodes in the past and no history of recent travel or contact with sick persons. The patient does not smoke or consume alcohol.

On physical examination, the patient appeared well in no acute distress. Blood pressure was 104/62 mmHg, heart rate was 92 beats per minute with a regular rhythm and temperature was normal. Examination of the chest, cardiovascular system, and abdomen was normal. He was oriented to time, place and person. Neurological examination revealed reduced power (grade 2/5) of both lower limbs. Reflexes of both lower limbs were absent while plantar reflexes were equivocal. Sensory examination was normal.

Laboratory investigations revealed the following: white blood cell count $10.1\times10^3/\mu L$, hemoglobin 11.7 g/dL, hematocrit 34.7 %, mean corpuscular volume 80.9 fL, platelet count $301 \times 10^3/\mu L$, and glucose 5.6 mmol/L, while sodium, potassium, HCO_3, creatinine and calcium were normal. Alanine transaminase and aspartate transaminase were elevated at 78 U/L and 70 U/L respectively (Normal less than 40 U/L). Total protein was 70 g/L, albumin was 32 g/L, alkaline

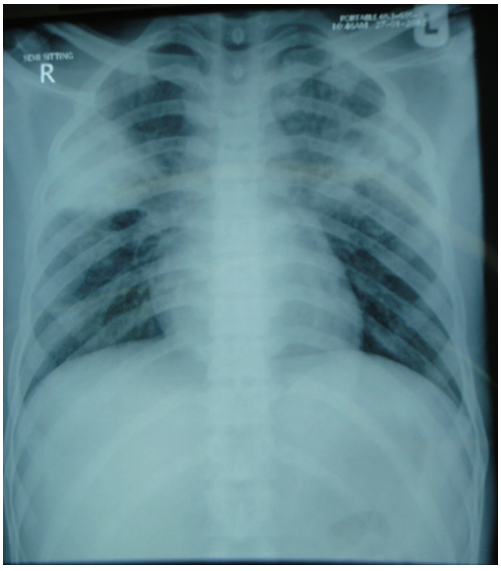

Figure 1. Antero-posterior view chest X-ray showing non-homogeneous opacities involving the right middle and left upper zones.

phosphatase was 171 U/L and bilirubin was normal. Cerebrospinal fluid analysis revealed a white cell count of less than 5/μL, no red blood cells, protein of 0.21 g/L, and glucose of 3.5 mmol/L. Gram stain and culture of the cerebrospinal fluid showed no organisms and no growth while viral serology and PCR testing for tuberculosis were negative. One day following admission, the patient developed fever with a temperature of 38.5°C. A chest X-ray revealed bilateral non-homogeneous opacities involving the right middle and left upper zones (Fig. 1). The purified protein derivative (PPD) test was positive at 14 mm after 48 hours. Examination of the sputum showed 25 acid-fast bacilli per 100 fields. Subsequent culture grew mycobacterium tuberculosis. The clinical impression was Guillain-Barre syndrome associated with pulmonary tuberculosis. The patient was started on intravenous Immunoglobulin G and anti-tuberculosis treatment along with physiotherapy. He improved and regained full power and mobility after 30 days.

Discussion

Guillain-Barre syndrome, first described in 1916, is characterized by acute areflexic muscle paralysis with albuminocytologic dissociation (high levels of protein in the cerebrospinal fluid with normal cell counts). Since poliomyelitis has nearly been eliminated, Guillain-Barre syndrome is currently the most frequent cause of acute flaccid paralysis and constitutes a neurological emergency.

Two-thirds of cases of Guillain-Barre syndrome are preceded by symptoms of an upper respiratory tract infection or diarrhea [3]. The most frequently identified infectious agent associated with the subsequent development of Guillain-Barre syndrome is Campylobacter jejuni followed by cytomegalovirus. The incidence of Guillain-Barre syndrome is estimated to be 0.25 to 0.65 per 1000 cases of Campylobacter jejuni infection and 0.6 to 2.2 per 1000 cases of primary cytomegalovirus infection. Other infectious agents with a well-defined association with Guillain-Barre syndrome are Epstein-Barr virus, Varicella-Zoster virus, mycoplasma pneumonia, and hemophilus influenza [3].

There is evidence from both human and animal studies that some cases of Guillain-Barre syndrome are caused by an infection-induced aberrant immune response that damages peripheral nerves [3]. Four key factors that control this response have been identified: antiganglioside antibodies, molecular mimicry and cross-reactivity, complement activation and host factors. In about half of patients with Guillain-Barre syndrome, serum antibodies to various gangliosides have been found in peripheral nerves such as LM1, GM1, GM1b, GM2 and GD1a [3]. These gangliosides have a specific tissue distribution in peripheral nerves and are organized in specialized functional microdomains called "lipid rafts", and play a part in the maintenance of the cell membrane structure. It is proposed that immune responses directed towards the infecting organisms are involved in the pathogenesis of Guillain-Barre syndrome by cross-reaction with neural tissues. The infecting organism induces humoral and cellular immune responses that, because of the sharing of homologous epitopes (molecular mimicry), cross-react with ganglioside surface components of peripheral nerves [2]. The phenomenon of molecular mimicry has been shown between the gangliosides of peripheral nerves of patients with Guillain–Barre syndrome and the lipooligosaccharide part of the outer membrane of C. jejuni [4].

We describe a patient with Guillain-Barre syndrome who was found to have pulmonary tuberculosis. We could find only 2 reports in the literature describing 3 patients with such an association [5, 6].

One report [5] described two patients: The first was an 18-year-old man who was admitted with acute onset of weakness involving upper and lower limbs for 10 days along with feeling unwell, weight loss and chronic productive cough. After treatment with anti-tuberculosis medications and steroids, the patient subsequently improved and had complete neurological recovery after 10 weeks. The second patient was a 56-year-old woman who was admitted to the hospital with a 3-week history of weakness of all four limbs, chronic productive cough, loss of appetite and fever. The patient was started on anti-tuberculosis therapy, but she was discharged against medical advice on the third day and died at home. No further details were provided in the report. The diagnosis of tuberculosis in both patients was made on the basis of radiological imaging, as they were too weak to produce a satisfactory specimen of sputum.

The second report [6] described a 35-year-old man with

a history of leprosy and tuberculous pleural effusion who was not adherent with therapy. He presented with sudden inability to walk and weakness of the upper limbs that was preceded by symptoms of a respiratory tract infection. Guillain-Barre syndrome was diagnosed. The sputum smear for Acid-fast bacilli was negative, but the chest X-ray revealed findings suggestive of pulmonary tuberculosis.

In a review of 1,100 cases of Guillain-Barre syndrome, about two-thirds of the cases were associated with medical conditions. The review reports tuberculosis as an associated illness in 8 cases. However, these cases were grouped under "tuberculosis of the lungs and brain"; the review does not provide further details and did not differentiate between pulmonary tuberculosis and other forms of tuberculosis [7].

To our knowledge, the patient we report is the first case of Guillain-Barre syndrome with microbiological confirmation of pulmonary tuberculosis. This association has not been confirmed and it remains a question whether tuberculosis is a contributory factor in the development of Guillain-Barre syndrome. We wish to draw the attention of clinicians to the consider tuberculosis as a possible associated condition when evaluating patients with Guillain-Barre syndrome.

Conflict of Interest

The authors certify no conflict of interest in the case report presented within. We report no financial or personal relationships with other people or organizations that could inappropriately influence our work.

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