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CASE REPORT

GUILLAIN-BARRE SYNDROME ASSOCIATED WITH PULMONARY TUBERCULOSIS: A CASE REPORT

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ABSTRACT

Guillain-Barre Syndrome is one of the most common neuropathies causing neuromuscular paralysis. Majority have a preceding viral or bacterial infection. We report a case of 25-year-old man who presented with clinical features of Guillain-Barre Syndrome and was found to have pulmonary tuberculosis. Only a few similar cases were reported hitherto. It is important to bring the possible association between Guillain-Barre Syndrome and tuberculosis to the attention of clinicians.

***Key words**: Guillain-Barre syndrome, tuberculosis*

INTRODUCTION

Guillain–Barre Syndrome (GBS) is an acute onset immune mediated disorder of the peripheral nervous system. Its average incidence rates are 1 to 2 per 100,000 persons per year ranging from 0.4 to 4.0 (1,2 -4). The likelihood of any individual acquiring GBS is 1:1000 (5). No figure is available for Ethiopia.

A prodromal illness caused by viral, mycoplasmal, bacterial and chlamydial infections is seen in up to 70% of the patients with a latent period of few days to weeks (6). The most frequent preceding symptoms are fever, sore throat, nasal discharge, and diarrhoea (7). The most frequently identified cause of infection is *Campylobacter jejuni*. Other causes of infections related to Guillain-Barre syndrome include *Cytomegalovirus, Epstein-Barr virus, influenza virus, Mycoplasma pneumonia and Haemophilus influenza* (8-10).

In a review of the literature, Leneman (11) & Soysal y Cols (12) found 8 out of 1100 & 2 out of 104 cases respectively of GBS to be associated with TB. We report the case of a patient who presented with the clinical features of GBS and was found to have pulmonary tuberculosis (PTB).

CASE REPORT

A 25-year-old man from Ethiopia presented with one month history of fever and cough for which he took unspecified antibiotic. The cough subsided but developed lower limb weakness which started from the lower extremities since 5 days before presentation and ascended within one day to the upper extremities. Prior to the onset of weakness he had back pain and numbness of the lower extremities. He also had difficulty in swallowing and slurring of speech a day after the weakness started. At presentation, he had shortness of breath. Otherwise there was no associated headache, vomiting, diarrhea or bladder or sphincter incontinence. There was no history of similar episodes in the past and no history of recent vaccination.

On physical examination, the patient appeared in acute distress. Pulse rate 110 beats per minute with regular rhythm, blood pressure was 140/100mmHg, respiratory rate was 32 breaths per minute, temperature was of 38.3^{o}C and he maintained his saturation with atmospheric air. Examination of the chest, cardiovascular system, and abdomen were normal. He was oriented to time person and place. Neurological examination revealed single breath count of 8, reduced power (grade 0/5) of all extremities, depressed

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tone, absent deep tendon reflexes over all extremities, while plantar reflexes were equivocal. Sensory examination was normal.

Cerebrospinal fluid (CSF) analysis done on the 5th day after onset of symptoms showed protein of 33.5mg/dl, glucose of 75.2mg/dl and no cells. Nerve conduction study (done on the 3rd day) after onset of symptoms was suggestive of motor axonal polyneuropathy of lower and upper limbs with conduction block. Chest X ray showed right middle lobe collapse consolidation with right pleural effusion and bilateral miliary infiltration. Gene Xpert of bronchoalveolar lavage (BAL) detected *Mycobacterium tuberculosis* which was sensitive to rifampicin and BAL cytology was reported as suggestive of chronic suppurative tuberculoid process.

The patient was admitted to the ICU, put on mechanical ventilator and treated with anti-tuberculosis regimen (rifampicin, isoniazid, pyrazinamide, and etambutol with pyridoxine). He was also treated empirically for ventilator associated pneumonia. He was not given steroid or IVIg. After 33 days of ICU stay, he died of severe sepsis of chest focus.

DISCUSSION

The patient had acute onset of flaccid paralysis of all four limbs without sensory loss, that progressed to type 2 respiratory failure that necessitated ventilatory support. He had as well the typical CSF finding of albuminocytologic dissociation as described historically by Guillain, Barre and Strohl in 1916 for the first time and the neurophysiologic studies which conform to the description of Guillain-Barre syndrome and he was found to have pulmonary tuberculosis. We could find only five reports in the literature describing 6 patients with such an association (13-16).

One report (13) described two patients. The first was an 18-year-old man who presented with weakness of extremities and chronic productive cough. The patient had complete neurological recovery after 10 weeks of treatment with anti-tuberculosis medications and steroids. The second patient was a 56-year-old woman with weakness of all four limbs, chronic productive cough, anorexia and fever. She was started on anti-tuberculosis therapy, but she took her own discharge on the third day and died at home. 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 (14) described a 34 year old man who presented with weakness of extremities and symptoms of respiratory tract infection. Sputum smear for AFB was negative, but had radiologic evidence of pulmonary tuberculosis. GBS and pulmonary tuberculosis were then diagnosed.

The third report (15) described a 21 year old man with a 4 day history of weakness of the lower limbs associated with cough that started a few days before the weakness. He had radiologic and bacteriologic evidence of PTB. With the clinical impression of PTB and GBS, he was treated with intravenous immunoglobulin (IVIg) and anti-TB. He improved and regained full power and mobility after 30 days.

Another report (16) discussed the case of 50 yr old man with weakness of extremities with no respiratory complaints and subsequently developed respiratory failure. Tracheal aspirate was positive for AFB. Patient showed gradual improvement with anti-TB therapy, steroids and mechanical ventilation.

One last report (17) identified a 25 year old man with smear positive PTB and GBS who was treated with IVIg, steroids and anti-TB. Association with extra pulmonary TB has also been reported (11, 18).

The patient we report is the first case of Guillain-Barre syndrome with microbiologically confirmed PTB in Ethiopia. The exact pathogenesis for the association between TB and GBS is not certain. Postulated mechanisms include molecular mimicry leading to the immunological attack of peripheral nerves, tuberculous polyradiculitis, direct involvement of the peripheral nervous system as a result of meningeal involvement, direct nerve root involvement, and granuloma affecting peripheral nerves (15,18). We wish to draw the attention of clinicians to this association of GBS with TB.

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