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Tuberculosis causing gullaine barre syndrome: A rare presentation

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Abstract

A 23 year old male, a known case of Psoriasis presented with significant loss of weight and loss of appetite over a period of 1 month. After examination and investigations, patient was found to have left sided pleural effusion, mediastinal lymphadenopathy and hepato-splenomegaly, which was diagnosed to be of tubercular origin. Patient was started on first line Anti-tubercular treatment. Three days later, patient started having complaints of weakness involving all four limbs without any bladder or bowel involvement. Nerve conduction studies was suggestive of motor polyneuropathy and a diagnosis of Gullaine Barre Syndrome was made. Patient was given IVIG following which there was no further progression of weakness and he started to improve so that he was able to stand and walk on his own at 6 weeks follow-up. Tuberculosis and GBS are commonly seen in our clinical practice. Despite knowing various infectious etiologies causing GBS, there have been only a few cases of Tuberculosis associated with GBS. This association needs to be reviewed further and strength of the same should be seen for in coming future.

Keywords

tuberculosis; GBS; anti-tubercular treatment (ATT); IVIG

Introduction

Guillain–Barré syndrome is currently the most frequent cause of acute flaccid paralysis worldwide [1] and constitutes one of the serious emergencies in neurology. Immunopathogenesis of the Guillain–Barré syndrome suggest that the disease actually encompasses a group of peripheral-nerve disorders. Subtypes are described based on electrophysiological patterns, the most common being Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and rarer ones being Acute Motor Axonal Neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN). Many antecedent infections have been identified as a cause of GBS including – Campylobacter jejuni, Cytomegalo virus Mycoplasma pneumonae, Epstein Barr virus and Influenza virus. Immunization and parturition have also been associated. But the association of

tuberculosis with GBS has rarely been reported.

The incidence of tuberculosis varies from 9 cases per 100,000 population per year in the US to 110–165 cases per 100,000 population in the developing countries of Asia and Africa [2]. It remains a major global problem and a public health issue of considerable magnitude. It has been estimated that approximately 10% of all patients with tuberculosis have CNS involvement [3]. Various CNS manifestations of tuberculosis include tubercular meningitis (TBM), tubercular encephalopathy, tubercular vasculopathy, space-occupying lesions: Tuberculoma, tubercular abscess, Pott's spine and Pott's paraplegia, tubercular arachnoiditis, spinal tuberculoma, spinal meningitis etc.

Here we report a case of a young male having disseminated tuberculosis with sudden onset quadriparesis which later on was found to be Guillaine Barre Syndrome (GBS).

Case Report

A 23 year old male, diagnosed case of plaque psoriasis, presented with complaints of loss of weight of 8kgs over one month and loss of appetite. This was not associated with fever, cough, joint pain, pain abdomen, or loose stools. There was no history of breathlessness or chest pain. There was no history of contact with tuberculosis or family history of tuberculosis. On examination, patient was conscious, oriented, having a normal BMI of 23.8kg/m2. His blood pressure was 120/80mm of Hg, pulse rate – 82/minute, respiratory rate 16/minute. On chest examination, there was dullness on percussion in left infrascapular area and breath sounds were diminished in the same area. So possibility of left side pleural effusion was kept and patient was investigated for the same. His routine investigations showed a hemoglobin of 11.3gm/dl, TLC- 4300/cumm and platelet count – 1.7 lac/cumm, ESR was 79mm. Liver and kidney functions were normal. Protein levels showed Albumin- Globulin reversal with a total protein of 8.1 gm/dl and albumin and globulin levels of 3.8gm/dl and 4.3gm/dl respectively. Electrolytes including serum sodium, potassium, calcium and phosphate were normal. Urine and Stool examination were normal. Chest X-ray revealed left sided pleural effusion with mediastinal widening. Ultrasound of abdomen revealed a liver span of 16.8cm and spleen of 14.8 cm both with normal echotexture. Montoux test was done which was strongly positive (15 x 18mm). Pleural fluid study showed an exudative picture with 350 cells(100% lymphocyte), a total protein of 5.6 gm/dl and ADA of 94.06U/L. Pleural fluid LDH was also raised with a level of 1146U/L against a serum LDH value of 228U/L. Pleural fluid study for gram stain and culture were negative and also there were no AFBs seen in pleural fluid. CBNAAT was done for pleural fluid which was also negative. There were no malignant cells in the pleural fluid. So, patient was started on anti-tubercular treatment. After three days of hospital admission, patient noticed that he was unable to open water bottle and not able to hold objects properly. The weakness progressed and he was unable to wear sleeper, climb stairs, further he was not able to go to washroom and stand to bear his weight. There was no history of altered sensorium, seizures, urinary retention, constipation, fecal incontinence. No history of any recent diarrhea, any recent vaccinations for influenza, meningococcus or any upper respiratory symptoms were there.

On neurological examination he was conscious and oriented to time, place and person, MMSE was 30/30, Tone was decreased across all joints in bilateral upper and lower limbs with a power of 2/5 in lower

limbs and 3/5 in upper limbs(according to the Medical Research Council (MRC) Scale for Muscle Strength). Deep tendon reflexes were absent, plantars were mute, single breath count was 26, superficial reflexes were intact, there was no cranial nerve & cerebellar involvement, sensory examination was also normal. So we kept differential diagnosis of Gullaine Barre Syndrome, Tuberculous arachnoiditis, Tuberculous vasculitis and Hypokalaemic paralysis. Patient was further investigated. His CEMRI Brain and Spine was normal, Nerve Conduction Study (NCS) showed no recordable response from bilateral peroneal and tibial nerves and low amplitude response attained from bilateral median and ulnar nerves with temporal dispersion. Sensory nerve conduction velocity was normal in bilateral upper and lower limbs and also no F-wave response was recordable from all the nerves. All these findings were suggestive of motor neuropathy of demyelinating type but also some axonal loss.

CSF analysis done after 7 days showed – Pauci-cellular picture with raised protein level of 112.6 mg suggestive of raised protein but no cells, CSF gram stain and culture was negative, AFB or malignant cells were not seen, India ink and Cryptococcal antigen was negative, CSF for HSV and CMV – PCR was also negative. So a diagnosis of LGBS was made, patient was started on intravenous immunoglobulin at a dose of 2gm/kg over 5 days. Now, we started to look for any triggering etiology of LGBS as tuberculosis had been rarely seen causing LGBS. All viral studies including Dengue IgM, Chikungunya IgM, HIV, HBsAg, IgM anti HCV, IgM anti HAV, IgM anti HEV, CMV and EBV serology were done and were negative. Typhidot IgM, stool routine, stool culture and blood culture were also negative. Thyroid profile was normal. Lastly Tuberculosis versus lymphomas was also ruled out. CECT chest & Abdomen showed Peri-bronchiovascular nodular thickening of lateral segment of Rt. Upper lobe with multiple necrotic & conglomerated mediastinal nodes suggestive of infective etiology (?) Koch's. Pleural biopsy showed fibro-collagenous tissue with few epithelioid cell granulomas with Langerhans giant cell and moderately dense lympho-histiocytic inflammatory infiltrate. ZN stain for AFB was non-contributory and a diagnosis of granulomatous pathology was kept. Final diagnosis of Disseminated Tuberculosis with LGBS was made. Patient was continued on ATT & Intavascular Immunoglobulin was given in a dose of 2gm/kg for 5 days. Physiotherapy was done. Patient improved symptomatically. Power also improved to 5/5 in both UL & 4+/5 in both LL.

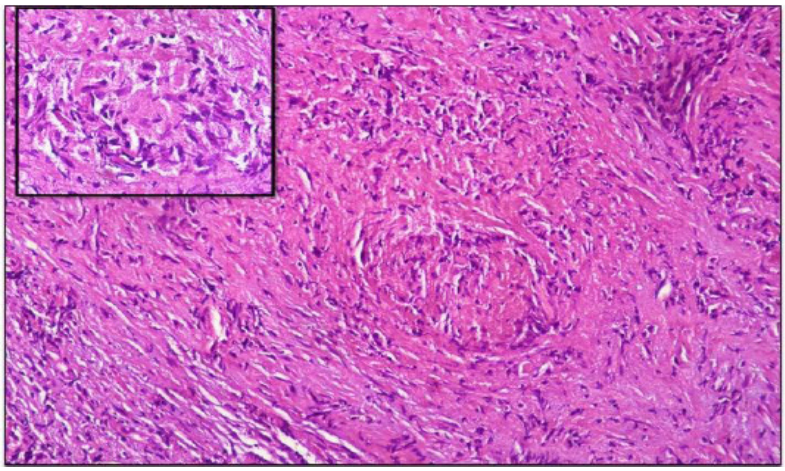
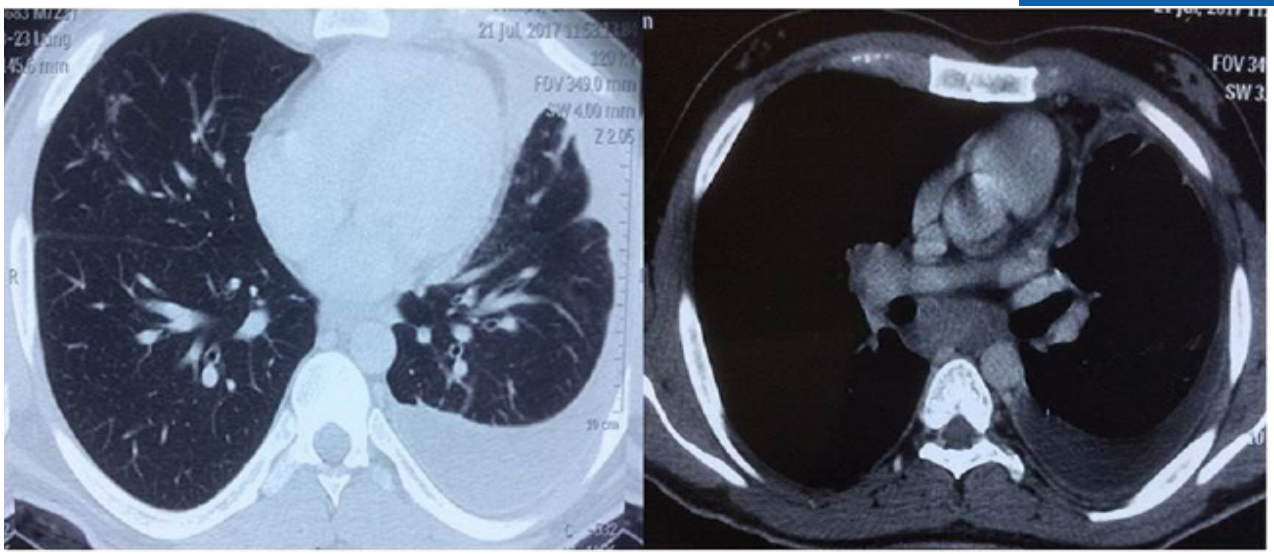
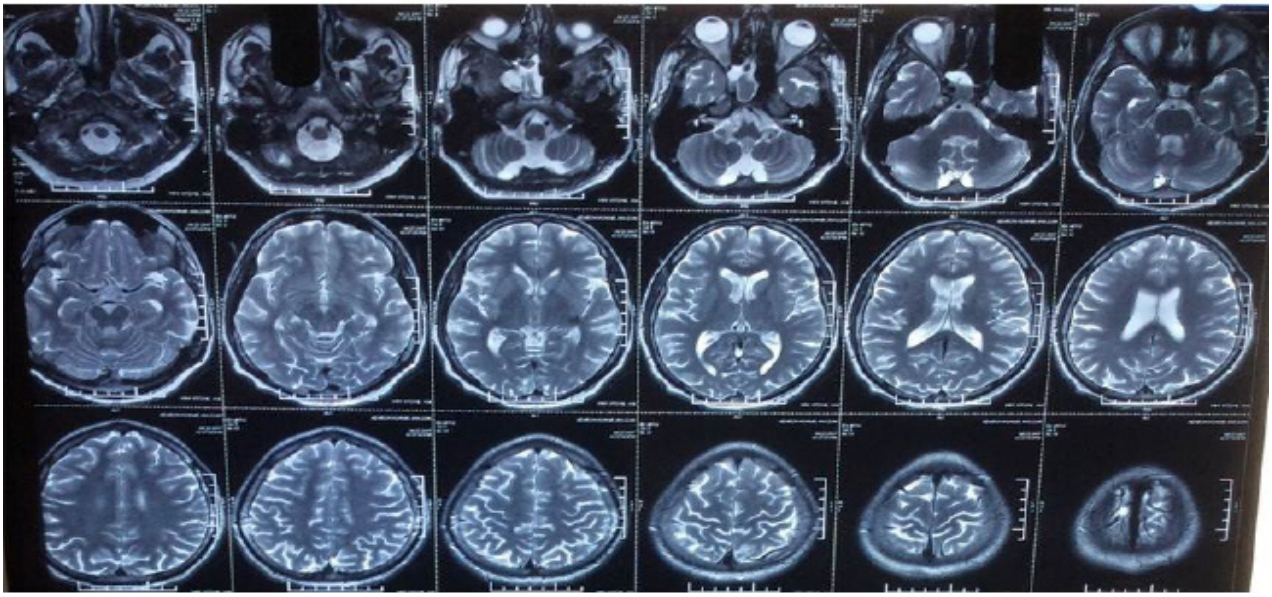


Figure 1: Section from the pleura shows inflammatory granulation tissue with the presence of ill- defined histiocytic collections and necrosis (H&E, 100×). Inset shows an epithelioid cell granuloma (H&E, 400×)



CECT Thorax: Peri- bronchiovascular nodular thickening of lateral segment of Rt. Upper lobe, Right pleural effusion with multiple necrotic & conglomerated mediastinal nodes suggestive of infective etiology (?)



CEMRI BRAIN: There are no abnormal focal areas of altered signal intensive in cerebral, hemisphere, brainstem and cerebellum. Brain parenchyma and ventricular system or normal. No evidence of intracranial space occupying lesion. There is no shift of midline structures.

Discussion

GBS is a post-infectious, immune-mediated disease. Both cellular and humoral immune mechanisms play a role in its pathogenesis. Many of the identified infectious agents are thought to trigger antibody production against ganglioside and glycolipids of myelin through molecular mimicry and cross-reactivity [4].

The co-occurrence of GBS and tuberculosis is rarely described in literature. In our case, patient was diagnosed to have left sided exudation pleural effusion, of granulomatous etiology and highly raised ADA levels. It was further confirmed with pleural biopsy which showed granulomatous inflammation. Pt was given ATT & 2 days later he started complaining of weakness of both lower limbs which on NCS was found to be because of motor polyneuropathy (AIDP variant of GBS). Isoniazid was withheld in view of neurotoxic effect of drug. We tried to rule out all other cause of GBS and all neurological manifestation of tuberculosis including Tubercular meningitis (TBM), tubercular encephalopathy, tubercular vasculopathy, space-occu-

pying lesions: tuberculoma, tubercular abscess, Pott's spine and Pott's paraplegia, tuberculous arachnoiditis, spinal tuberculoma, spinal meningitis etc.

The differential diagnosis of isoniazid-neuropathy was considered very early on in this patient. However, we consider it highly unlikely as the isonaizid induced neuropathy usually does not present with just 2 days of intake and also no significant improvement occurred in this patient despite early stoppage of isoniazid. In literature, there had been only 2 cases of early onset isoniazid induced motor dominant neuropathy in which also isoniazid was taken for atleast 2 weeks [5].

Tubercular etiology was confirmed with subsequently patient being afebrile, gain of weight and improvement in weakness over follow-up visits. In a review of 1100 cases of GBS, Leneman reported tuberculosis as an associated illness in only eight cases [6]. Vyravanathan and Senanayake reported two cases of tuberculosis with GBS in SriLanka, and they proposed that a cell-mediated hypersensitivity reaction, or invasion of the nerve root by tubercular bacilli, would seem to be the likely explanation of the neuropathy [7]. Only few other cases have been reported so far of GBS with tuberculosis and most of them were associated with pulmonary tuberculosis. These include case reports by Canham et al. [8], de la Torre et al. [9], Soehardy et al. [10], Taha et al. [11], Mohta et al [12].

Conclusion

All previously reported cases had features of tuberculosis prior to onset of weakness. The temporal association between GBS and Tuberculosis suggest that this is unlikely to be due to chance. Hence, the ability of Mycobacterium Tuberculosis to trigger GBS either due to immunological response or molecular mimicry further need to be investigated.

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