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***Case Report***

# GUILLAIN BARRE SYNDROME DUE TO PULMONARY TUBERCULOSIS

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## ABSTRACT

We report a case of G.B. syndrome in a 50 yr old male patient in whom there was quadriparesis with respiratory failure who was treated with artificial respiration. When tracheal aspirates were sent for analysis, baetrioragical AFB was found. It was pulmonary TB. The pathogenesis of GB syndrome in infective disease in by no means clear. The majority view that GB syndrome represents a cell mediated, delayed hypersensitivity reaction which is a recognized feature of tuberculosis. However the possibility of tuberculous polyradiculitis being the cause cannot be ruled out.

***Keywords:*** *G.B. Syndrome, Poly Radiculitis, Pulmonary Koch's*

## INTRODUCTION

Guillain Barre Syndrome (GBS) is known to follow a variety of viral, myoplasmal, bacterial and chlamydial infections (Arnason 1975). In a review of the literature, Leneman (1966) found 8 out of 1100 cases of this syndrome to be associated with tuberculosis. We report a case of GBS due to pulmonary tuberculosis, from Mandya Institute of Medical Sciences, Mandya, Karnataka.

## CASES

A 50 year old male patient was admitted to hospital with

1) H/o fever – 20 days
2) Weakness of both upper and lower limbs – 15 days.

Fever was intermittent in nature, low grade, not associated with chills, rigors, decreased on taking medications.

Weakness of upper and lower limbs- insidious in onset, gradually progressive involved the lower limbs first, later involved the upper limbs. Later patient developed difficulty in breathing. Pt developed respiratory distress for which pt was given ventilator support.

There was no h/o cough, expectoration, there was no H/o giddiness, syncope, chest pain, palpitations, No H/o pain abdomen, vomiting, loose stools, No h/o facial weakness/features of other cranial nerve involvement, No h/o headache, convulsions, loss of consciousness. There was no H/O tingling/ numbness.

On examination, Patient was moderately built and nourished. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy, pedal oedema.

Pulse -80/min, Bp-110/70 mm hg temp-normal respiratory rate -20/min.

***Central Nerve System***

Patient was conscious and oriented; swallowing difficulty was present, pharyngeal wall movements decreased. There was no wasting of limbs. Hypotonia was present in upper and lower limbs, Power in all the four limbs - 3/5. DTRS-absent, planters mute B/L Sensations – both posterior column sensations and lateral column sensations were intact. There was no neck rigidity/abnormal movements.

RS- NVBS heard, B/L Scattered crepitations+, B/L rhonchi +,

CVS - Normal

PA – Normal

***Investigations:*** complete haemogram, Urine routine }Normal.

ECG – Normal

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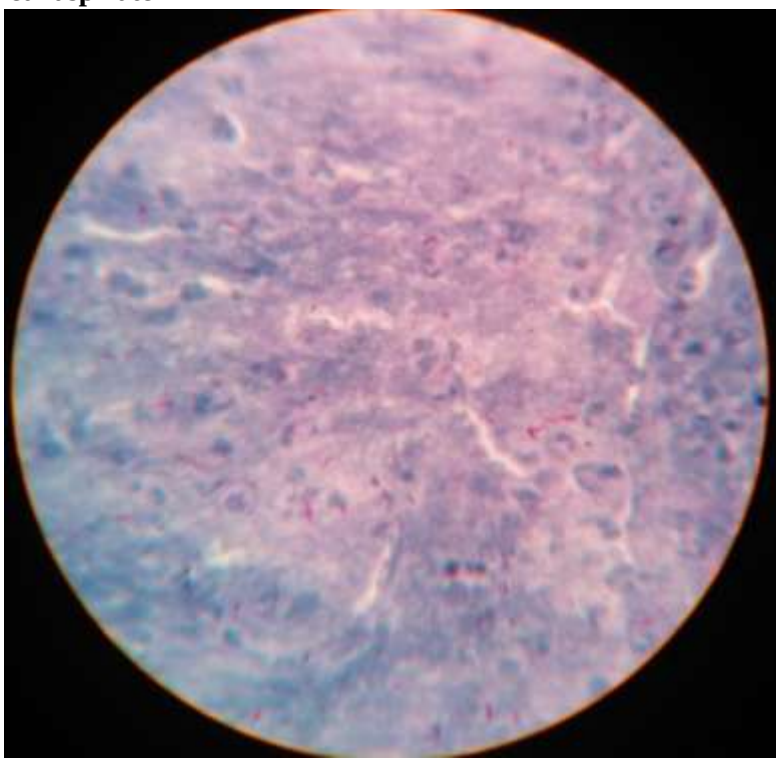
***Case Report***

**Z-N staining of tracheal aspirate**



***CXR-PA View:*** Patchy pneumonitis. Of left lower zone.

***CT Head:*** No evidence of intracranial lesion.

***CSF Analysis:*** Protein – 210 mg % sugar – 70 mg%, no cells were seen.

Patient did not show significant improvement patient after treating for pneumonia. Patient was referred to NIMHANS Bangalore for tracheobronchial aspirate culture sensitivity no growth. AFB- Positive. Pt was referred to NIMHANS for Neurologist's opinion considering GB Syndrome. At NIMHANS, plasmapharesis was done and referred back to our hospital. Patient was then out of ventilator and was able to breath independently. By then the tracheo bronchial aspirate report from our hospital showed AFB.

Patient was treated with ATT (Monto 4 Kit), Steroids (Dexona 8mg $12^{th}$ hrly), Antibiotics, proton pump inhibitors. Physiotherapy was given to the limbs. Patient started improving gradually and motor weakness improved with physiotherapy.

## DISCUSSION

Guillain – barre syndrome is a diffuse, predominantly motor polyneuropathy, due to possibly an autoimmune mechanism resulting in inflammation and edema of spinal nerves A prodromal illness is observed in 2/3 of the patients with a latent period of hours to 18 days. Respiratory failure is seen ¼ of patients with GB syndrome.

In our patient, clinical features suggestive of motor weakness, decreased reflexes, respiratory paralysis along with CSF analysis showing albumin cytological dissociation were characteristic of G.B. Syndrome.

The Patchy pneumonitis of the lung which was not responding to antibiotics later showed AFB in the tracheobronchial aspirate analysis.

The incidence of Guillain- Barre syndrome in western countries is 1-2 cases per 1,00,000 per year. In India the incidence is <5 percent.

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The pathogenesis of GBS in infective disease in by no means clear. The majority view is that GBS represents a cell mediated, delayed hypersensitivity reaction (Thomas, 1978) which is a recognized feature of tuberculosis. However the possibility of tuberculosis polyradicutits being the cause cannot be ruled out.

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