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CASE STUDY

TUBERCULOSIS AND ANTERIOR HORN CELL DISEASE; A COINCIDENCE OR ASSOCIATION

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ABSTRACT

Tuberculosis is one of the commonest maladies which have plagued developing countries like India with its various clinical manifestations. Here we describe a case of disseminated tuberculosis with involvement of brain and lymphoreticular system in form of generalized lymphadenopathy, splenomegaly and testicular mass. After biopsy confirmation he was started on Antitubercular treatment to which he responded adequately. However the same patient suffered from slowly progressive weakness and muscle wasting of bilateral upper and lower limbs symmetrically. On further investigations he was found to have Anterior Horn Cell disease a common type of motor neuron disease. Simultaneous occurrence of both these diseases in same individual has been seldom reported in scientific literature.

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INTRODUCTION

India has a huge burden of patients with tuberculosis with a myriad of clinical manifestations. The prevalence is ever increasing which can be gauged from the fact that the World Health Organization (WHO) TB statistics for India for 2016 gives an estimated incidence figure of 2.79 million cases of TB. It is estimated that about 40% of the Indian population is infected with Mycobacterium tuberculosis bacteria, the vast majority of whom have latent TB rather than active disease. On the other hand motor neuron disease and its commonest prototype Amyotrophic Lateral Sclerosis (ALS) is a rare and often underreported entity. With gradually increasing life expectancy particularly among developing nations and enhanced healthcare facilities the prevalence has substantially increased. A study by Karissa C Arthur et al projected the number of ALS cases across the globe will increase from 222,801 in 2015 to 376,674 in 2040, representing an increase of 69% (1). Here we present a case with both diseases in same patient. Coincidence of motor neurone disease and tuberculosis is unique and there is only one case reported in scientific literature till date (2).

Case presentation

A 42 years old gentleman presented with sudden onset weakness of left side of body since 3 months and dysarthria. It was slowly progressive in nature. He also had low grade intermittent fever with evening rise of temperature without any night sweats or chill and rigor. He had no history of any convulsion, diplopia, ptosis, dimness of vision, nasal intonation, dysphagia, headache, vomiting, any sensory abnormalities, fasciculations, tremor or neck stiffness. There was no history of shortness of breath, chronic cough, haemoptysis, abdominal pain, jaundice, chest pain, bleeding from any site. The patient had no history of contact, or any high risk behavior. He was a teetotaler and there was no exposure to environmental fumes or toxins. On physical examination the patient was conscious, alert, haemodynamically stable, moderate pallor was present with firm, mobile, non-tender axillary and epitrochlear lymphadenopathy. On neurological examination speech was dysarthric with upper motor neuron type VII cranial nerve palsy. There was spastic weakness of left side of body along with muscle wasting of all 4 limbs symmetrically. Sensory system examination and cerebellar functions was within normal limit. On examination of the gastrointestinal system there was firm, non tender hepatosplenomegaly. There was a 3 cm X 3 cm soft left testicular mass with loss of testicular sensation. Examination of other system was within normal limit. Laboratory results showed normocytic normochromic anaemia Haemoglobin 10.9 gm%, raised ESR 48 mm in 1st hour, raised Lactate dehydrogenase levels 593 U/L. CSF analysis showed 10 cell with 50% lymphocytes, low glucose, raised protein 181 mg/dl, ADA was 7, AFB stain and CSF CBNAAT was negative. Other investigations including HIV serology, serum IgM and sputum for acid fast bacilli were negative.

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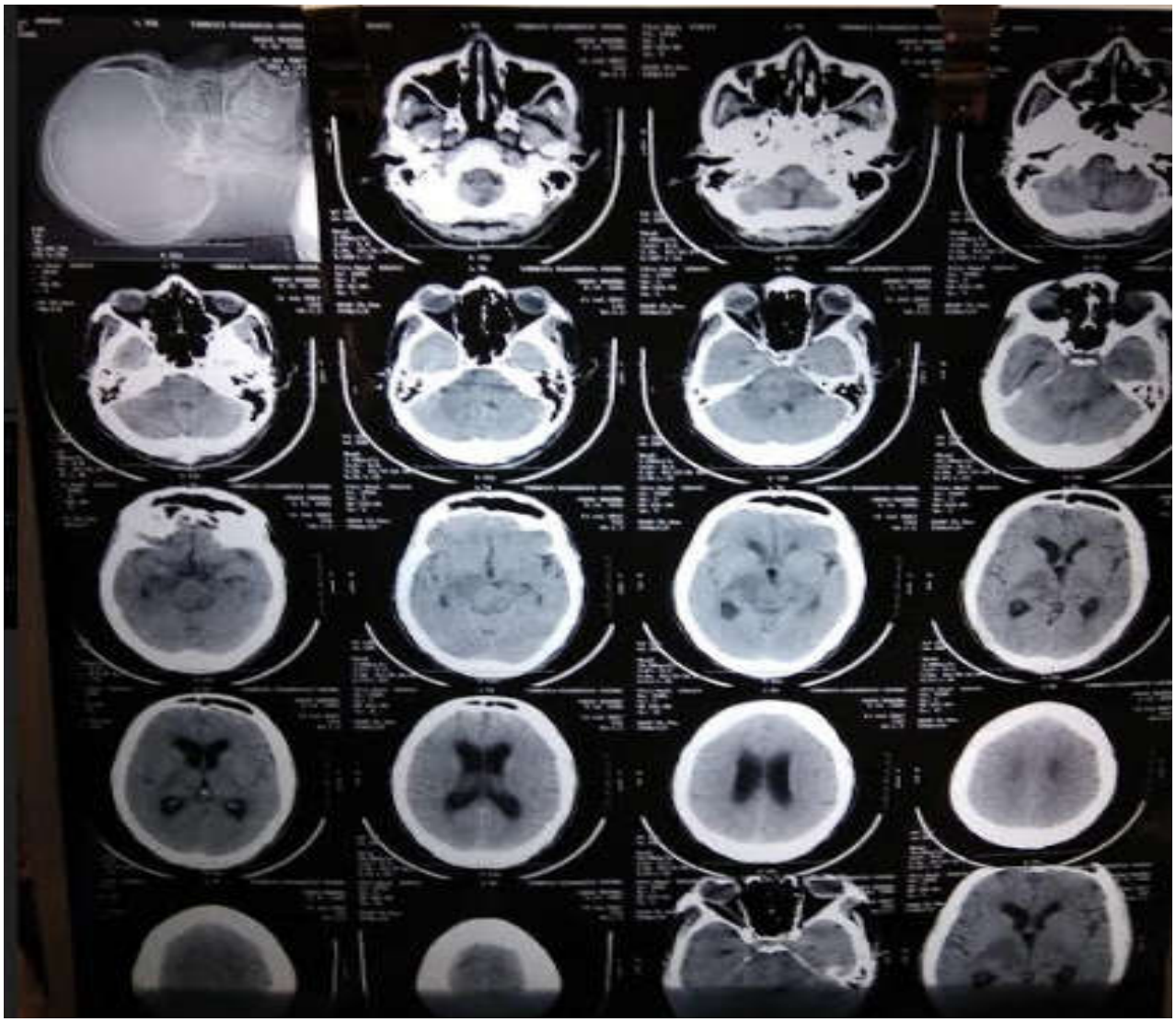

Figure 1. CT scan of brain a heterodense area in brainstem with mass effect over 4th ventricle with dilatation of supratentorial system. Right thalamic region shows hypodense area? Infarction

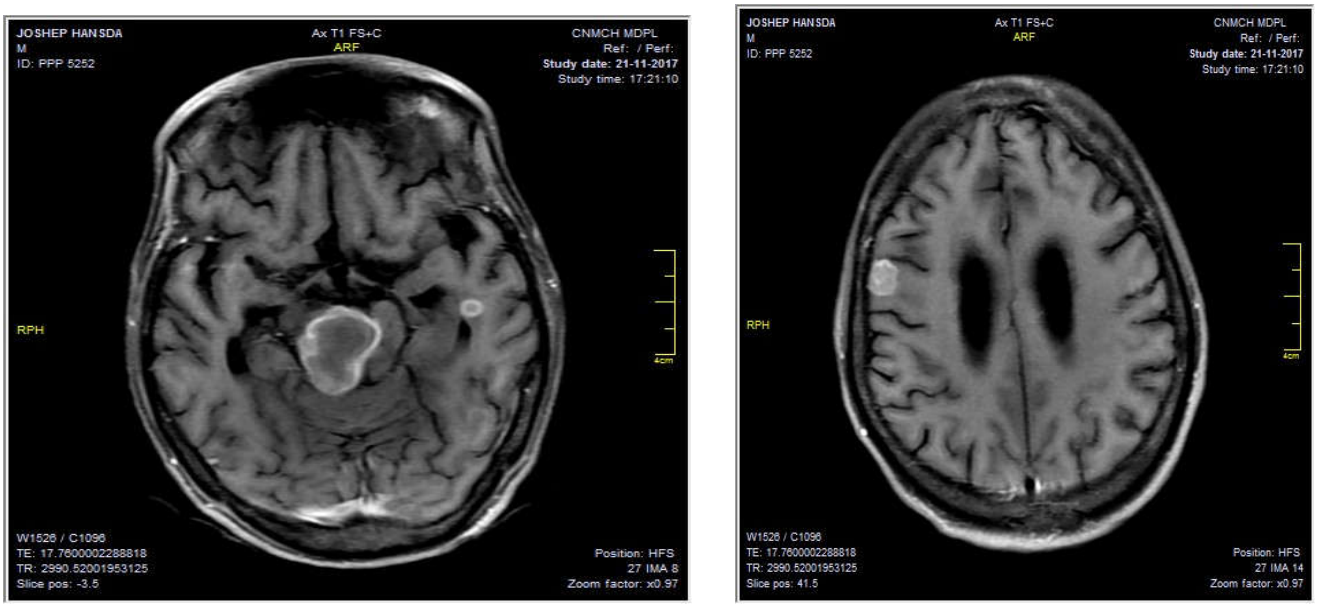

Figure 2. MRI Brain: features suggestive of tuberculoma/metastasis? In both cerebral hemispheres and a larger one in brainstem. Diffuse cerebral and cerebellar atrophy

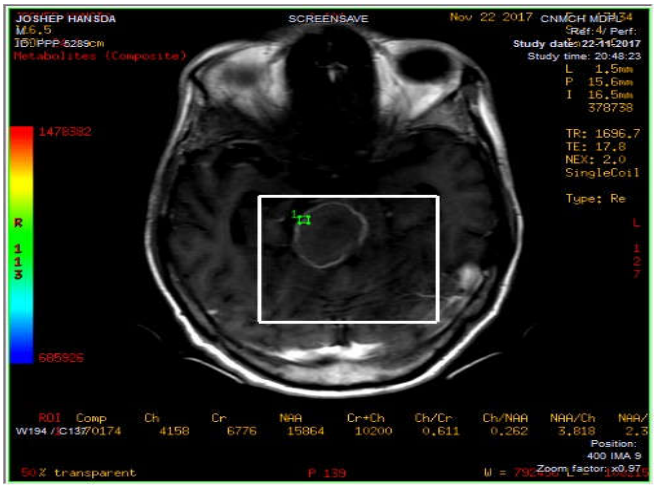

Figure 3. MR Spectroscopy suggestive of tuberculoma

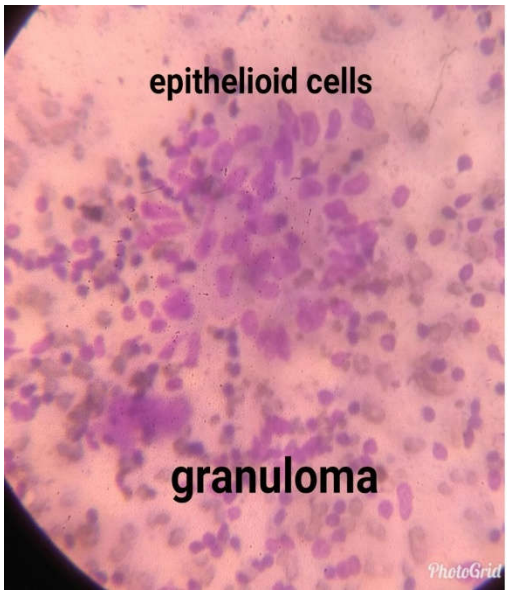

Figure 4. Biopsy from epitrochlear lymph node suggestive of tubercular lymphadenitis

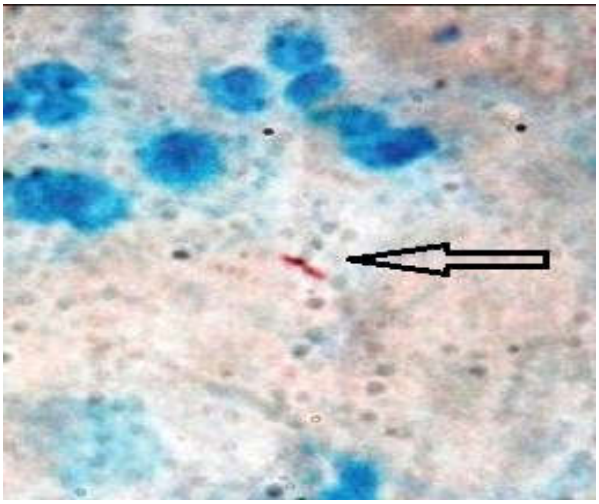

Figure 5. FNAC from testicular swelling shows mycobacterial lesion. The arrow marks shows acid fast bacilli in Zeihl-Neelsen stain

Among radiological investigations Chest X Ray was normal, Ultrasonography of whole abdomen showed splenomegaly and hypoechoic space occupying lesion of 3 cm X 2.4 cm in left scrotal sac. A non-contrast CT scan of brain done during onset of the disease showed hypodense area in right thalamic region (infarction?) and another heterodense area in the brainstem region with mass effect over 4th ventricle with dilation of supratentorial ventricular system. (Figure 1) After admission to our institution a contrast enhanced MRI brain was done which showed multiple ring enhancing lesions in both cerebral hemispheres on post contrast T1W1. There was a 3.8x3.4x3 cm discrete lesion on brain stem more on right side. Lesions were hypo to intermediate on T1, T2 and T2 FLAIR. DWI & ADC mapping did not show any restriction of diffusion. The brain stem lesion compressed the 4th ventricle with dilatation of the 3rd and lateral ventricles. The impression of radiologist was metastasis/ tuberculoma? in both cerebral hemisphere & brainstem. (Figure 2) So for distinction between the two single voxel [STEAM with short TE (35 ms), PRESS with longer TE (144ms) and multivoxel proton MR Spectroscopy was done. The larger ring enhancing lesion in brainstem revealed markedly elevated lipid peak. So MR Spectroscopy was in favour of tuberculoma. (Figure 3) For histopathological confirmation a biopsy from right epitrochlear lymph node was done which showed necrotic material distorting the lymph node architecture with Epithelioid and Langhans type of giant cells in the periphery suggestive of tubercular lymphadenitis. (Figure 4) A FNAC from the testicular swelling also showed abundant caseous necrotic material along with degenerated lymphoid cells and histiocytes. Zeihl- Neelsen staining showed acid fast bacilli (Figure 5). So the cytological features were suggestive of mycobacterial lesion. So the diagnosis was disseminated tuberculosis.

He was started on category I Antitubercular drugs, Isoniazid, Rifampicin, Pyrazinamide, Streptomycin according to weight band and in addition dexamethasone and pyridoxine. After 2 months when the intensive phase was completed he was evaluated and it was found that his fever had subsided, weakness had improved, the lymph nodes had disappeared, spleen was no longer palpable and the testicular mass had decreased in size. However despite adequate nutrition and appetite the muscle wasting of upper & lower limbs persisted. So nerve conduction study was done which showed absent CMAP with bilateral S1 radiculopathy. A EMG was done which showed spontaneous activity in form of fasciculations at rest, high amplitude, low duration polyphasic MUAP with decreased recruitment suggestive of neurogenic pattern classical of anterior horn disease. So the final diagnosis was disseminated tuberculosis with anterior horn cell disease.

DISCUSSION

The patient was diagnosed with histologically confirmed disseminated Kochs with concomitant motor neuron disease. But the association of tuberculosis with Anterior Horn Cell disease is very rare. Anterior Horn Cell Disease is a degenerative disease of the nervous system without clear cause. Its pathogenesis is still unknown. Anterior Horn Cell disease the prototype of Motor Neurone Disease is a disease of multifactorial inheritance and the potential cause is the disturbance to the RNA metabolism (3). Besides genetic factors, other causes include excitatory toxicity of amino acids, dysfunction of glial cells, abnormal protein deposition, oxidative stress, mitochondria dysfunction, and abnormal autoimmunity. Some substances may be specifically toxic to neurons.

It may be hypotheticated that the long-term chronic inflammation of tuberculosis resulted in some substances with neuronal toxicity so this may be an avenue for further research.

Conclusion

This may be a case of two distinct disorders occurring simultaneously. But it is unclear whether the relationship between tuberculosis and Anterior Horn Cell disease is occasional suppression due to the chronic inflammation or causality.

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