

ICSOL: Tubercular Brain Abscess - A Rare Case Report

Ajima K S¹, Adithya S L¹, Shaiju S Dharan², Dr. Dhanya Dharman³

¹Pharm D Intern (Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala, India)
Corresponding Author Email: [dhanyadharman07\[at\]gmail.com](mailto:dhanyadharman07[at]gmail.com)

²Pharm D Intern (Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala, India)

³Principal/HOD (Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala, India)

⁴Associate Professor (Department of Pharmacy Practice, Ezhuthachan College of Pharmaceutical Sciences, Marayamuttom, Thiruvananthapuram, Kerala, India)

Abstract: Tuberculous Brain Abscess (TBA) is a rarely reported form of central nervous system tuberculosis. Brain TB abscess are devoid of granulomatous reaction associated with TB. Histologically and clinically these abscesses are similar to pyogenic brain abscess. ^[1] TBA is characterized by an encapsulated collection of pus, containing viable tubercular bacilli without evidence of tubercular granuloma. Patients may present with features of raised intracranial pressure and focal neurological deficit with the site of the abscess. ^[2] We report a rare case ICSOL Tubercular abscess in a patient with chief complaints of slurring of speech, drooling of saliva @ side of the lip. In developing countries, the prevalence of CNS tuberculosis is estimated about 4 - 7.5%. In Kerala its prevalence of tuberculous abscess is found only in 3% of total TB cases.

Keywords: ICSOL, Granuloma, TB Abscess, Stenosis, Cortical Sulci, Circle of Willis.

1. Introduction

TBA is a focal collection of pus containing abundant Acid-Fast Bacilli (AFB) surrounded by dense capsule consists of vascular granulation tissue. ^[3] The clinical presentation of brain abscess is influenced by a number of factors including the size and location of the abscess, the virulence of the infecting organism (s), and the presence of any underlying systemic conditions. In a typical case of pyogenic brain abscess, headache is clearly the most common presenting symptom. The nature of the headache has no particular distinguishing features, although it is often characterized by a dull aching that is poorly localized. ^[4] Rupture of the brain abscess into the ventricular space is often fatal and presents as sudden version of a pre existing headache accompanied by new onset of meningismus. ^[5] TB has been reported in 4% to 7.5% of patients with CNS tuberculosis without HIV infection compared to 20% in HIV positive patients.

2. Case Report

A 54 year old male patient presented with of chief complaints slurring of speech, drooling of saliva @ side of the lip. He had past history of Type II Diabetes Mellitus (20 years), CAD (15 years) and CLD (1.5 years). He had a known family history of cardiac disease (Father and brother had CAD). He was a known alcoholic for the past 17 years and had stopped 1.5 years ago. He had undergone an open heart surgery 15 years back.

His own medicines were T. URSODEOXYCHOLIC ACID 300mg 1 - 0 - 1, T. CARVEDILOL 6.25mg 1 - 0 - 0, T. RABEPRAZOLE 20mg 1 - 0 - 0, T. LASILACTONE (FUROSEMIDE+SPIRONOLACTONE 20/50mg 1 - 0 - 0).

Admission examination shows elevated blood pressure (170/90 mmHg). His pulse rate was slightly declined to 60 beats/min. On systemic examination CVS and lungs found to be normal. GIT found to be distended. His sugar level was not under control and it was confirmed by elevated HbA1c (11.3%). Haemoglobin was reduced to 10.6mg/dL. Lipid profile of the patient was within the normal limits. Liver function test shows elevation, since the patient is having CLD. Total Bilirubin and Indirect Bilirubin was 1.19mg/dL and 1.01mg/dL respectively.

Plain CT scan of brain (Fig: 1) reveals large well defined hypodense area @ Frontoparieto temporal lobe. There is evidence of hyperdensity seen along the cortical sulci of @ frontoparieto temporal lobe. Possibility of acute to subacute infarct likely. MRI of neck and cerebral arteries (Fig: 2) shows no occlusion and no evidence of vascular lesions. The circle of Willis and its branches shows normal morphology. MRI of brain (plain and contrast) (Fig: 3) confirms the presence of TB abscess on the brain. The findings were two intra - axial lobulated thick walled peripherally enhancing hyperdense lesion in @ frontal lobe opercular region. Significant internal diffusion restriction corresponding to T2W1 hyper intense area. Disproportional perilesional parenchymal edema extending into @ insular, ganglio - capsular region causing a mass. MRI features are more in favour of TB abscesses.



Figure 1: CT scan of brain (Plain): Possibility of acute to subacute infarct likely

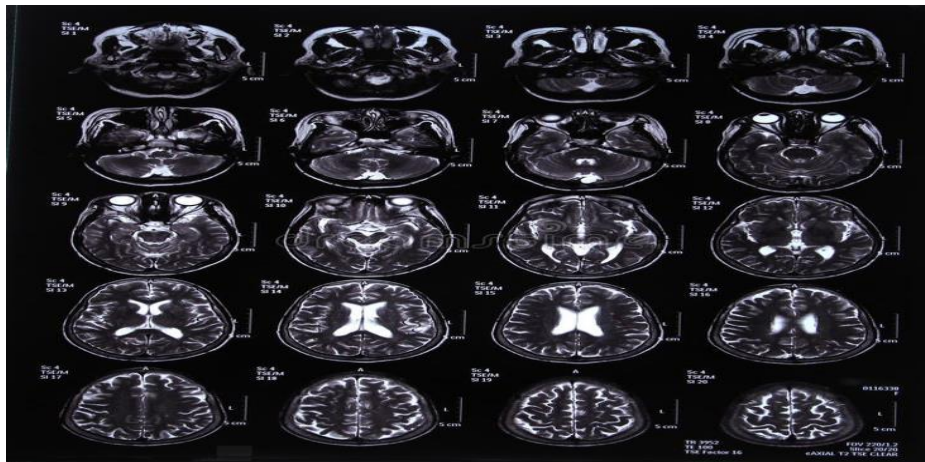


Figure 2: MRI of neck and cerebral arteries: Shows normal morphology

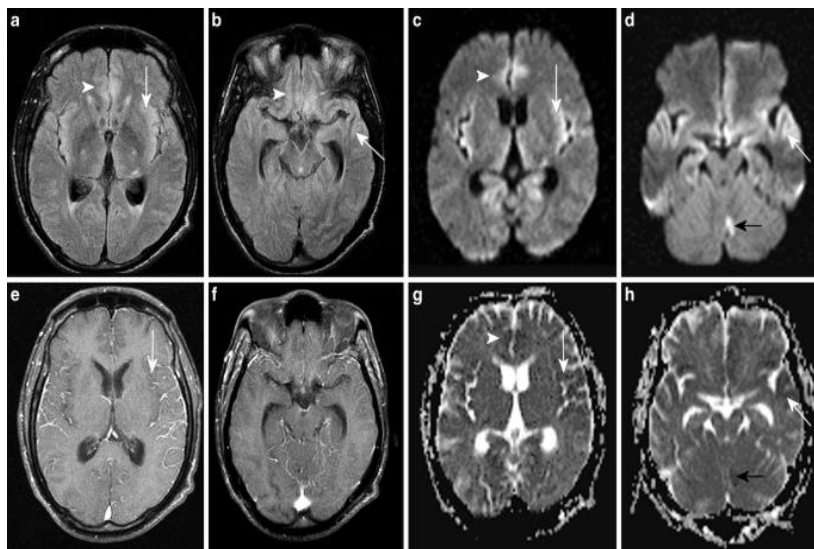


Figure 3: MRI of brain (Plain & contrast): More in favour of TB abscess

Patient was started with Anti tubercular drugs, Anti epileptics, Steroids, Antidiabetics, Antihypertensives, Hepatoprotectants, Diuretics, Proton Pump Inhibitors, Analgesics, Antiemetics and Antioxidants. ECG found to be normal. Patient’s Blood Pressure was not under control and given Antihypertensive (Calcium Channel Blockers) & the target was set to 140/90 mmHg. Anti TB drugs started on the 6th day of admission. And finally the patient was symptomatically improved and was discharged.

3. Discussion

Intracranial space occupying lesions (ICSOL) tubercular abscess refers to a collection of pus within the brain caused by tuberculosis infection. The etiology of tubercular abscess lies in the spread of tuberculosis (TB) infection to the brain. TB is typically caused by the bacterium *Mycobacterium tuberculosis*. In the case of a tubercular abscess, the bacteria reach the brain either through hematogenous spread from a primary site of infection or by direct extension from adjacent

structures such as the meninges. The immune system's response to the infection leads to the formation of pus within the brain, resulting in the abscess.

In the case report entitled "A rare case of tubercular cerebellar abscess" by K Wanjari, VP Baradkar, et al. presents a 55-year-old HIV seronegative non-diabetic female came with complaints of headache, neck pain, and unsteadiness of gait since two months. [7] She had been on treatment for pulmonary tuberculosis, diagnosed earlier. Diagnosis was made by CT Scan of brain and confirmed by bacteriological examination of drained pus obtained by suboccipital craniotomy. Next case report discussed with a title of "Tuberculous cerebellar abscess: a case report" by Abayomi G Oshinowo, B Wayne Blount et al. explains a 36-year-old previously healthy, immunocompetent woman with a diagnosis of extrapulmonary tuberculosis and no determined primary focus was found to have cerebellar abscess. Treatment included surgical excision of the abscess. Combined with 6 months of antituberculosis therapy. Diagnostic tools included the tuberculosis skin test, smears and culture of specimens, computed tomographic scans, and rapid assays based on nucleic acid amplification, ie, polymerase chain reaction. The polymerase chain reaction has great potential for rapid diagnosis of Mycobacterium tuberculosis, particularly when there might be few bacilli, as in pleural, peritoneal, or cerebrospinal fluid. Appropriate therapy for tuberculous cerebellar abscess includes standard antituberculosis medications for 6 to 9 months and surgical excision for the abscess.

Here in this case, the 54-year-old male patient was presented with complaints of slurring of speech, drooling of saliva. CT Brain report shows a large well-defined hypodense area noted in right frontoparietal temporal lobe region. Patient was managed with antitubercular medications include T. ISONIAZID 300mg OD, T. RIFAMPICIN 450mg OD, T. ETHAMBUTOL 800mg OD, INJ. STREPTOMYCIN 0.75mg IM OD. On admission time BP was elevated and managed with T. CILNIDIPINE 10mg BD. Further managed with T. CARVEDILOL 6.25mg OD, INJ. LEVETIRACETAM 500mg TDS, INJ. MANNITOL 100mg TDS, INJ. EDARAVONE 30mg BD, INJ. THIAMINE 200ml BD, T. RIFAXIMIN 400mg BD, INJ. 3% NACL 100ml TDS for correcting hyponatremia. Patient was managed with minimal hepatotoxic drugs. After 7 days patient condition improved and hence advised to continue anti-TB drugs for 6 months duration.

4. Conclusion

IC SOL Tubercular Brain Abscess is a rare manifestation of CNS tuberculosis. It is characterized by an encapsulated collection of pus, containing viable tubercular bacilli without evidence of tubercular granuloma. [7] Patient may present with features of raised intracranial pressure and focal neurologic deficit commensurate with the site of abscess. Here in this case patient presented with complaints of slurring of speech, drooling of saliva. He had past history of Type II Diabetes Mellitus (20 years), CAD (15 years) and CLD (1.5 years). He had a known family history of cardiac disease (Father and brother had CAD). He was a known alcoholic for the past 17 years and had stopped 1.5 years

ago. He had undergone an open heart surgery 15 years back. His own medicines were T. URSODEOXYCHOLIC ACID 300mg BD, T. CARVEDILOL 6.25mg BD, T. RABEPRAZOLE 20mg OD, T. LASILACTONE OD. Patient was started with Anti-tubercular drugs, Anti-epileptics, Steroids, Antidiabetics, Antihypertensives, Hepatoprotectants, Diuretics, Proton Pump Inhibitors, Analgesics, Antiemetics and Antioxidants. In conclusion, treatment for Tubercular abscess should be personalized, depending upon its severity.

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