Paradoxical Reaction in a case of Brain Tuberculoma

Sushma Dugad¹, Saurabh Ambadekar² and Anand Singh^{2*}

¹Professor & HOD, Department of TB Chest, Dr. Vasantrao Pawar Medical College Hospital and Research Centre, Nashik, India; sushmadugad@gmail.com ²Resident, Department of TB Chest, Dr. Vasantrao Pawar Medical College Hospital and Research Centre, Nashik, Inida; sambadekar@yahoo.com, anand69_ace@hotmail.com

Abstract

A 15 year old male was diagnosed to have central nervous system (cns) tuberculosis with ataxia. After starting him on conventional anti tuberculous treatment he deteriorated both clinically and radiologically. On investigation the diagnosis of drug resistant tuberculosis was ruled out and corticosteroid treatment was added. Eventually patient improved symptomatically and had good weight gain.

Keywords: Brain, Paradoxical reaction, Tuberculoma

1. Introduction

Paradoxical Reactions (PRs) usually occur at the start of treatment in patients having tuberculosis. PR basically deals with the clinical symptomatology, signs and imaging modalities consistent with tuberculosis, Treatment failure must be ruled out¹. Time to onset of paradoxical reactions is period between start of treatment to worsening of symptoms. In majority of cases the period is under 3 months². PR is frequently seen to involve lymphatic, nervous, respiratory system³. Among patients with tuberculosis 1% may develop PR and it occurs more frequently in meinigeal tb⁴. PR in brain occurs in patients with meningeal, pulmonary (especially miliary) tb. This case is being published in order to sensitise practioners about the prompt accurate diagnosis of PR and its best available treatment regimens.

2. Case Report

A 15 year old male patient came with history of cough with white expectoration, decreased appetite, diplopia,

imbalance during walking, giddiness, puffiness of face, difficulty in swallowing since 6 months.

Patient's brain Magnetic Resonance Imaging [MRI] dated 24/12/2012 showed multiple ring enhancing lesions randomly scattered in bilateral cerebral and cerebellar hemispheres. Neurologist opinion was done and he gave the diagnosis of CNS Tuberculoma with Cerebellar ataxia.

Patient was started on conventional anti Kochs treatment with Rifampicin, Isoniazid, Pyrazinamide, Ethambutol and steroids. Patient developed Rifampicin toxicity and hence it was stopped.

Repeat MRI dated13/3/13 showed increased size and number of ring enhancing lesions.After 3 months of treatment patient developed swelling in right cervical region.On examination patients Right Cervical lymph node was palpable. On Fine Needle Aspiration Cytology [FNAC] of the lymph node pus was aspirated and sent for culture and sensitivity. Acid Fast Bacilli [AFB] was detected however no resistance documented on gene expert study.

Patient was deteriorating but we continued the same treatment along with steroids in tapering dose. Patient

^{*}Author for correspondence

again came with enlarged lymph node in August 2013. FNAC showed AFB however no resistance was documented on gene expert study to Rifampicin, Isoniazid. Rifampicin was reintroduced in August 2013.

Patient tolerated Rifampicin very well and improved symptomatically. His ataxia, nystagmus and other CNS symptoms improved. His weight gain was 14 kg.

3. Discussion

The likely explanation for PR is an interaction between the host's immune response and the direct effects of mycobacterial products PR appear within range of 20–109 days⁵.

PR during ATT most commonly involves lungs and pleura followed by spine and paraspinal tissue, intracranium, peritoneum, bone and joint, lymph node⁶.

Teoh R et al. reported 10 patients with PR IT of which five patients made a full recovery, 3were left with mild neurological deficit and in two it was too early to assess the outcome⁷.

PR involving the CNS may be life threatening Therapy with steroids appears to diminish neurological symptoms and may improve outcome. Infliximab (tumor necrosis factor antibody) profoundly inhibits cellular immune responses to Mycobacterium tuberculosis⁸ Neurosurgical referral and intervention is recommended for patients with raised intracranial pressure, convulsions and spinal cord compression symptoms.

Speedy diagnosis of CNS tuberculosis is need of the hour since prognosis is directly proportional to promptness of treatment initiation. Stereotactic biopsy is the gold standard but is resorted to only when indeterminate results are obtained by non interventional techniques⁹.

Multiple tuberculomas and infratentorial locations are being recognized with importance. An isointense or hypointense core with a hyperintense rim on T2-weighted and FLAIR images is the commonest radiological apprearance. Core hypointensity of lesions on these images correlates with necrosis and the large number of cells¹⁰.

Ependymomas and tuberculomas are common paediatric posterior fossa lesions in developing countries and can be mistaken for one other¹¹.

Opthalmic problems, cranial nerve paralysis and the occurence of basal exudates are vital predictors of hydrocephalus. In early meningeal tb patients ther may be full recovery. Hydrocephalus causes enhanced mortality and poor outcome¹².

The MRI is the gold standard in the assessment of brain tb. The tuberculous lesion displays isointense to hyperintense depending on the stage of MRI and type of T weighed image. caseation makes the center becomes bright and produces "target sign"¹⁰.

Central nervous system tuberculosis includes meningitis, tuberculoma. PR are well known phenomenon¹³.

4. References

- 1. Burman WJ, Jones BE. Treatment of HIV-related tuberculosis in the era of effective antiretroviral therapy. Am J Respir Crit Care Med. 2001; 164:7–12.
- 2. Jniene A, Soualhi M, Bouassel M, Nayme I, Zahraoui R, Iraqui G. Epidemiological, therapeutic and evolutionary profiles in patients with lymph node tuberculosis. Tuberk Toraks. 2010; 58:366–74.
- 3. Smith H. Paradoxical responses during the chemotherapy of tuberculosis. J Infect. 1987; 15:1–3.
- 4. Malone JL, Paparello S, Rickman LS, Wagner KF, Monahan B, Oldfield EC. Intracranial tuberculoma developing during therapy for TBM. West J Med. 1990; 152:188–90.
- Afghani B, Lieberman JM. Paradoxical enlargement or development of intracranial tuberculomas during therapy: case report and review. Clin Infect Dis. 1994; 19(6):1092–9.
- Cheng VC, Yam WC, Woo PC, Lau SK, Hung IF, Wong SP et al. Risk factors for development of paradoxical response during antituberculosis therapy in HIV-negative patients. Eur J Clin Microbiol Infect Dis. 2003; 22(10):597–602.
- 7. Teoh R, Humphries MJ, O'Mahony G. Symptomatic intracranial tuberculoma developing during treatment of tuberculosis: a report of10 patients and review of the literature. Q J Med. 1987; 63(241):449–60.
- 8. Blackmore TK, Manning L, Taylor WJ, Wallis RS. Therapeutic use of infliximab in tuberculosis to control severe paradoxical reaction of the brain and lymph nodes. Clin Infect Dis. 2008; 47(10):e83–5.
- 9. Delance AR, Safaee M, Oh MC, Clark AJ, Kaur G, Sun MZ et al. Tuberculoma of the central nervous system. J Clin Neurosci. 2013; 20(10):1333–41.
- Wasay M, Kheleani BA, Moolani MK, Zaheer J, Pui M, Hasan S, et al. Brain CT and MRI findings in 100 consecutive patients with intracranial tuberculoma. J Neuroimaging. 2003; 13(3):240–7.
- 11. Futane S, Salunke P, Sahoo S, Mulinani N, Vyas S . Ependymoma misdiagnosed as tuberculoma, misguided by overemphasis on MR spectroscopy in endemic area. Acta Neurochir (Wien). 2013; 155(10):1969–71.

- Raut T, Garg RK, Jain A, Verma R, Singh MK, Malhotra HS, Kohli N, Parihar A . Hydrocephalus in TBM: Incidence, its predictive factors and impact on the prognosis. J Infect. 2013; 66(4):330–7.
- Das A, Das SK, Mandal A, Halder AK. Cerebral tuberculoma as a manifestation of paradoxical reaction in patients with pulmonary and extrapulmonary tuberculosis. J Neurosci Rural Pract. 2012; 3(3):350–354.