Review Article

TUBERCULOMA OF BRAIN – A REVIEW

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Received 22 January 2014; accepted 14 March 2014

Abstract

Tuberculoma is usually caused by Mycobacterium tuberculosis that mainly affects the lungs and brain. Cerebral tuberculoma is the formation of non neoplastic, granuloma like lesions in the central nervous system. It can occur in any part of the brain. Various diagnostic techniques are, smear test for acid fast bacilli, presence of granuloma on histopathology, imaging techniques such as CT and MRI. Patient with tuberculoma should receive antitubercular drug treatment. Depending upon the clinical symptoms, conformation of diagnosis, early institution of specific antitubercular treatment and close clinical monitoring for ADR are the key to the successful management of tuberculoma. This review article is focused to review the pathogenesis and treatments of tuberculoma.

Key words: tuberculoma, granuloma, antitubercular drugs.

INTRODUCTION

Tuberculoma is an intracranial mass occurring secondary to dissemination of tuberculosis, elsewhere in the body. Tuberculoma may be solitary or multiple. The incidence rate of brain tuberculoma is 1.4%. It is a potentially curable disorder but delay of diagnosis can lead to increased mortality and morbidity rate. Tuberculoma affects 12-30% of all intracranial masses. It is more common in people with poor immune system, young children, HIV infected individuals and drug resistant strains of mycobacterium tuberculosis. Chance of occurrence of tuberculosis is 20 times more in HIV positive individuals when compared to HIV negative individuals. The main clinical symptoms are intracranial hypertension, papillary edema, hemiplegia, hemiparesis, epilepsy, pulmonary tuberculosis. Diagnosis is mainly based upon the clinical features like cerebrospinal fluid changes and imaging characteristics.

PATHOGENESIS

Mycobacterium tuberculosis is an aerobic, nonmotile non spore forming acid fast bacillus. Primary tuberculosis infection is associated with hematological spread, organism is found in different organs and lie dormant. Such dormant foci get distributed in the brain when organism reaches the brain and/or meninges from acute primary lesion via blood stream to establish focal lesion known as tuberculomas. Intracranial tuberculoma orginates as small tubercles and develop to form mature tuberculoma. Mature forms are composed of fibroblast, epithelial cells and lymphocytes. In early stage of tuberculoma formation, there is an inflammatory reaction and a capsule poor in collagenous tissue, but in the developmental stage capsule becomes rich in collagen, and surrounding inflammatory reaction will disappear. Tuberculomas may appear as small single or multiple nodules or as larger irregular lesions with a central area of caseous necrosis. Rupture of tuberculomas into the subarachnoid space results in the development of tuberculosis meningitis, which may present as acute or chronic meningitis.

RADIOLOGICAL FEATURES OF TUBERCULOMA

CT scan and MRI scan are the important imaging techniques for diagnosing tuberculoma. Tuberculoma appears as a round or lobulated mass with irregular walls and show homogenous ring enhancement after administration of radio contrast. In CT scan appearance of tuberculoma round or oval mass lesion that is isointense or hypotense on the plain of CT scan is observed. But the MRI findings of tuberculoma depend upon the stage of evolution. MRI has greater sensitivity and specificity than CT scan.

Figure 1. Axial T1

- Isointense to gray matter.
- May have central region of hyperintensity representing caseation.
Figure 2. Axial T2

- Isointense to gray matter.
- May have central region of hypointensity representing gliosis and abundant monocyte infiltration.
- Lesions are surrounded by vasogenic oedema.

**TREATMENT**

Based upon clinical, laboratory and imaging findings susceptible patients should be started with antitubercular medicine. Duration of treatment is mainly based upon the part that is affected. Patients with CNS tuberculosis should receive 12 – 30 months of antitubercular treatment\(^1\). Isoniazid, rifampin, pyrazinamide and ethambutol is the four-drug regimen that is most commonly recommended, also second line agents such as aminoglycoside and fluoroquinolones can also be added. Adverse drug reactions associated with antitubercular drug include, discolouration of urine associated with rifampicin, changes in serum uric acid levels in patients receiving pyrazinamide and alterations in visual activity in patients receiving ethambutol\(^5, 8, 9\) and \(^17\). Other ADRs associated with antitubercular drugs are gastrointestinal reactions, dermatological reactions and elevation of liver enzyme levels\(^10, 15\). New intracranial tuberculoma or expansions of older existing lesions require no change in antitubercular treatment. Adjacent corticosteroid therapy will help to reduce neurological symptoms. Today the use of dexamethasone as an adjunct therapy for 4-8 weeks in CNS tuberculosis is mostly accepted\(^1\). This is very effective. Dexamethasone reduces the deleterious effect of the immune response and reduces the incidence of hydrocephalus and brain infarction\(^14, 16\). If there is no response to the therapy within 8 weeks, a stereotactic biopsy of the tuberculoma should be performed\(^15\).

**CONCLUSION**

Early recognition and timely treatment of tuberculoma is critical to prevent the considerable mortality and morbidity associated with the condition. Mainly tuberculomas are treated by chemotherapy with antitubercular drug, a minimum of 10 months treatment is required. In severe cases surgical excision of the tuberculoma can be performed.

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Source of support: Nil; Conflict of interest: None declared