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Intracranial Tuberculoma: comparison of MRI findings with histopathology

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Abstract

Background: Central nervous system (CNS) tuberculosis (TB) is a serious form of TB, due to hematogenous spread of Mycobacterium tuberculosis(MT), Manifesting as meningitis, cerebritis and tuberculous abscesses or tuberculomas(1). The main aim of this study is to compare the MR signal intensity patterns and enhancement pattern of intracranial tuberculoma with their histopathological features.

Method: This study includes 3 cases of intracranial tuberculoma which were examined histopathologically.

Result: On T1-weighted images, the granulomas showed a slightly hyperintense rim surrounded by a complete or partial rim of slight hypointensity and central isointensity . Histologically, the zone of central isointensity corresponded to caseation necrosis plus adjacent cellular infiltrates. The hyperintense and hypointense rims corresponded to the layers of collagenous fiber and the layers of the inflammatory cellular infiltrates, respectively. On T2-weighted images, the entire portion of the granuloma showed slightly heterogeneous isointensity or hypointensity .On postcontrast T1-weighted images, there were single or multiple conglomerate ring enhancements within a tuberculoma.

Conclusion: Combination of the described signal intensity patterns and conglomerate ringlike enhancing appearance of the lesion is characteristic of tuberculoma, and may play an important role in differentiating intracranial tuberculomas from other ring-enhancing brain lesions.

Keywords: Intracranial tuberculoma; Neurotuberculosis; Central nervous system; MRI

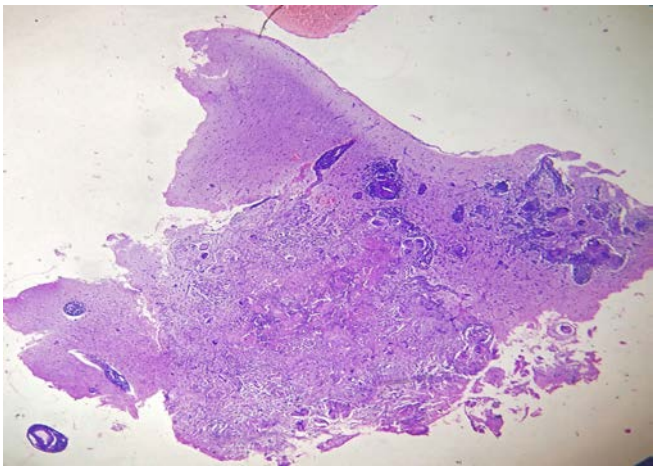
Introduction

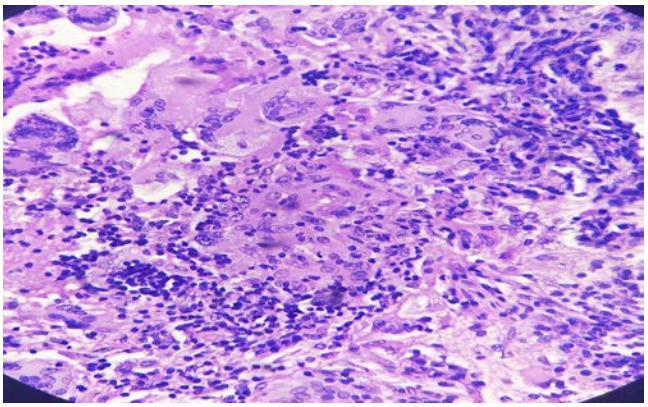
Tuberculosis (TB) is a disease that can involve all systems, and unless treated, can give rise to serious consequences by damaging the involved system. Involvement of the central nervous system (CNS) is the most dangerous form of TB, which continues to represent an endemic public health problem, particularly in developing countries [1-2].

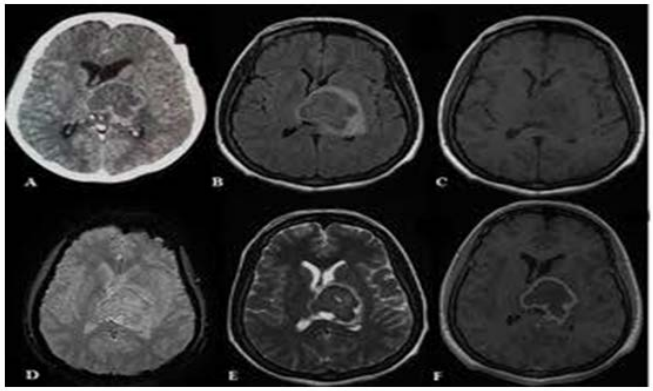
The disease occurs by spreading secondarily to the CNS years after the initial pulmonary infection with reactivation of the bacillus by the hematogenous route. CNS spread results in two interrelated pathological processes, in the form of TB meningitis or intracranial tuberculomas. The most commonly seen form is

meningitis, followed by tuberculomas and the much rarer TB abscesses [3].

Materials And Method : The present study was a prospective and retrospective study which included 3 cases, histopathologically proved as CNS tuberculoma.







Results

On the T1-weighted images, the lesions had a thick rim of slight hyperintensity compared with normal gray matter with surrounding complete or partial rim of slight hypointensity.The central portion of the lesion displayed isointensity . Histologically, the hyperintense rim corresponded to the layer of collagenous fibers, and the hypointense rim corresponded to the layer of outer inflammatory cellular infiltrates, which was composed of epithelioid cells and lymphocytes. The central isointense portions reflected the caseation necrosis with lining of inflammatory cellular infiltrates. On T2-weighted images, the entire portion of the lesions, including central caseation necrosis, showed slightly heterogeneous isointensity or hypointensity . Within the hypointense or isointense granulomas, multiple small markedly hypointense foci were found . After contract enhancement, there was ring like enhancement, either single ring or multiple conglomerate rings, that corresponded to the rims seen on nonenhanced T1-weighted images and correlated histologically with both the inner collagen and the outer cellular layers. The nonenhancing portions corresponded to central caseation necrosis.

Discussion

Tuberculosis of the central nervous system (CNS) accounts for approximately 1% of all of the diseases caused by Mycobacterium tuberculosis, but it comprises 10–15% of extrapulmonary tuberculosis. The tuberculoma may be single or less commonly multiple, and their sizes may vary from a few millimeters to a diameter of 3–4 cm[4-5].

Intracranial tuberculomas originate as a conglomerate of small tubercles that join to form a mature tuberculoma composed of a central caseation necrosis surrounded by a zone of fibroblasts, epithelioid cells, Langhans giant cells, and lymphocytes. In the early stage of tuberculoma formation, there is a predominate inflammatory reaction with an abundance of giant cells and a capsule poor in collagenous tissue. Later, the

capsule becomes richer in collagen and the surrounding inflammatory reaction may disappear. The central portion of the lesion is transformed into caseous material by a necrotic process [6]

Conclusion

Combination of the described signal intensity patterns and conglomerate ringlike enhancing appearance of the lesion is characteristic of tuberculoma, and may play an important role in differentiating intracranial tuberculomas from other ring-enhancing brain lesions.

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