A retrospective study of CT findings of intracranial Tuberculosis in patients with AIDS in University Teaching Hospital, Lusaka, Zambia

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ABSTRACT

Objective: To study the computed tomography (CT) findings of intracranial tuberculosis (T.B) in patients with acquired immune deficiency syndrome (AIDS).

Design: CT findings of intracranial T.B in 40 patients with AIDS were retrospectively analysed.

Result: T.B meningitis were found in 26 cases (65%), Tuberculoma were found in 22 cases (55%), 8 cases had hydrocephalus (2%), 6 cases had cerebral infarts (15%), cerebritis were found in 4 cases (10%), combination of T.b meningitis and parenchymal lesions were found in 24 cases (60%).

Conclusion: T.B Meningitis, tuberculoma and combination of T.b meningitis and parenchymal lesion are common CT findings of intracranial T.B in patient with AIDS.

INTRODUCTION

T.B can occur in the early stages of human immunodeficiency Virus (HIV) disease and may be the first AIDS – defining illness. Central nervous system (CNS) involvement is seen in 10% of all patients with T.B[1]. T.B of the CNS is a granulomatous infection caused by mycobacterium tuberculosis. The disease predominantly involves the brain and meninges. Clinical diagnosis can be difficult; therefore, imaging has an important role in establishing the diagnosis. There are isolated reports in the literature of atypical mycobacteria involving the CNS in AIDS patients, but these reports have not concentrated on the radiographic features. To study the CT findings of intracranial T.B in patients with AIDS, 40 cases of intracranial T.B whom are all proved by clinically, and them were taken for my study from January, 2004 to March 2008 in University Teaching Hospital Lusaka, Zambia.

Key words: Computed Tomography, Intracranial Tuberculosis, Acquired Immune Deficiency Syndrome.

METHODS

40 cases (23 male, 17 female) of intracranial T. B from 2 – 65 years old (average 32 years old). 32 patients came from U. T. H., 8 cases came from the other hospital or clinic. The clinical features included fever, headache, seizures, weakness, neurologic deficit and increase intracranial pressure. All the patients were confirmed HIV – positive, in which 18 cases in highly active antiretrovirial therapy (HAART). The diagnosis of intracranial T. B was based on clinical manifestations, cerebrospinal fluid (CSF) criteria with / without polymerase chain reaction (PCR) assays, chest radiological result, posteropterative biopsy and following up after antituberculosis therapy (ATT). Brain CT scan with and without contrast were carried out in all the patients using Aura CT Scanner in U.T.H, Lusaka, Zambia. Axial sections were obtained at 5mm – 7mm parallet to the orbitomatal line.

RESULT

T.B meningitis were found in 26 cases (15%), in which 16 cases had isodesence to hyperdense exulate effaces CSF space, filled basal cisterns in non contrast enhancing CT (NECT) scan and intense basilar meningeal enhancement in contrast enhancing CT (CECT) (40%) and 10 cases had deffuse sulci or focal sulci enhancement surrounding by tuberculoma in CECT (25%). Tuberculoma were found in 22 patients (55%), in which 14 cases were with solitary lesions (35%) and 8 cases were with multiple lesions (20%). 16 cases of tuberculoma had ring – enhancing (40%) and 6 cases of tuberculoma had both ring and nodular enhancement (15%). There are 14 cases of tuberculoma exceed 10 mm in diameter (35%). In which had 8 cases involved both gray and gray-white matter junction associated with poorly peripheral ring enhancement and marled surrounding edema. 8 cases had hydrocephalus (20%), infarctions were

found in 6 cases in ganglia region (15), cerebritis were found in 4 cases which appeared as focal gyri-formed enhancement of hypodensities in cerebral hemisphere (10%). Combination of meningitis and parenchymal lesions were found in 24 cases (60%), in which 16 cases had T.b meningitis with tuburculoma (14%). 4 cases had T.B meningitis with infarctions (10%). 2 cases had tuberculoma with cerebritis (15%), 2 cases had T.B meningitis, tuberculoma, infarction and cerebritis together (5%). The compositive CT findings of intracranial T.B in 40 patients with AIDS are given in table 1

Table 1. CT findings of intracranial T.B in 40 patients with AIDS

CT findings	No of cases	% of Cases
T.B meningitis	26	65%
Tuberculoma	22	55%
Hydrocephalus	8	20%
Infarction	6	15%
Cerebritis	4	10%
Combination	24	60%

DISCUSSION

Intracranial T.B is a granulomatous infection caused by mycobacterium, the disease predominately involves the brain and meninges. Infection is hematogenously spread from a primary focus, usually in the lungs rarely gastroinstinstinal or genitourinary tract.[2] Intracvranial T.B infection starts in a subpial or subependymal cortical focus, resulting in a granuloma that erodes into the subarachnoid space, causing basal leptomenigitis. The meningitis usually causes communicating hydrocephalus, but it may also cause obstruction of the foramina of Luschka and Magendie, resulting in obstructive hydrocephalus. Vasculitis involving the lenticulostriate and thalamoperforatoring arteries may occur and cause small infarctions in the deep gray-matter nuclei and deep white matter. There are 26 cases (65%) of T.B meningitis in the study, in which 8 cases associated with hydrocephalus and 6 cases with infarcts in ganglia region. Hematogenous T.B spread to gray-white matter junction or extension of meningitis into parenchyma via cortial veins, resulting in focal parenchymal granulomas as tuberculoma, tuberculous cerebritis and pachymeningitis. There are 22 cases of tuberculoma (55%) and 4 cases of cerebritis (10%) present in the study. Autopsy study on intracranial T.B patient with AIDS have revealed small aggregates of cells containing the organism in a perivascular location. Granuloma formation is poorly formed or absent [3,4]. There are 8 cases (20%) had porrly peripheral enhancement tuberculoma and 4 cases (10%) of focal T.B cerebritis in the study, the result is compatible with that autopsy findings [fig 1,2,3]. Hansman reported intraparenchymal tuberculomas are seen as multiple lesion less than 1 cm that predominate at the grey-white matter interface and periventricular regions. They have little mass effect or oedema[5]. In this study there are only 6 cases (15%) had some manifestations as hansman above reported; therefore there are 14 cases (35%) demonstrated ring enhancement tuberculoma exceed 1 cm in size and associated with makered surrounding oedoma and mass effect.[Fig. 4.5.6] The reason why had this different findings in the study, it could be relative to immune reconstitution inflammatory syndrome (RIS) in patients who were receiving HAART [6,7]. The result need to confirm by more study in future.

The spectrum of intracranial disease in patient with AIDS is wide and can be broadly categorized into primary effects of HIV, opportunistic infections, neoplasms and vascular disease. T.B meningitis is more common in patients who are immuno suppressed, such as older persons, young children, patient with HIV or diabetes and patients taking steroids or cytotoxic drug.[8] T.B menigitis is seen as leptominingeal thecleening and enhacement predominatly involving the basal cirsterns, prepontine, ambient cisterns and suprasellar areas. [9] The brain parenchymal t.b as tuberculoma, tuberculoms cerebritis and tuberculous abcesses relatively are less common. The CT findings of intracranial T.B in patient with AIDS often minics other disease like Cryptococcus, coccididomycosis, toxoplasmosis, primary central nervous system lymphoma (PCNSL) or metastic tumours, cryptococcomas and other inflammatory or parasitic granulomas, neurosarcoidosis et. Distinguishing between them often poses a diagnostic challenge to the radiologist owing to their varied any similar appearances. The combinations of meningitis and patenchymal lesions, however, highlight suggests intracranaial T.B. The chest radiological findings and C.S. F analysis help for the diagnosis of intracranial T.B in patient with AIDS.

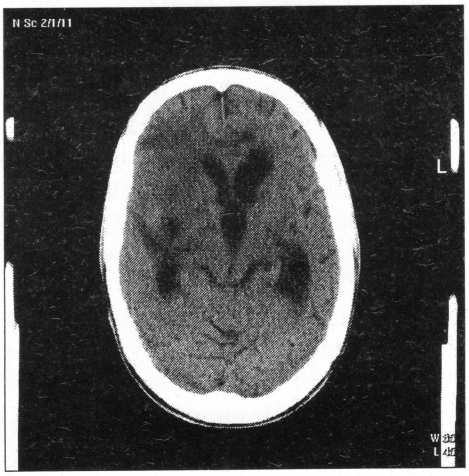

Fig. 1. NECT shows an ill-defined isodense / hypodense lesion with mass effect in right fronto temptal lebes and with hydrocephalus present

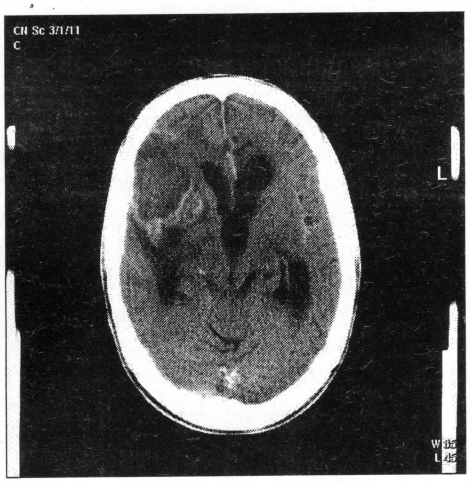

Fig 2. The same patient to fig 1. There is a peripheral poorly enhanced ring with the lesion, meningeal enhancement in left sylivian fissure present on CECT. It is suggestive of a poorly formed tuberculoma with T.B meningitis and hydrocephalus

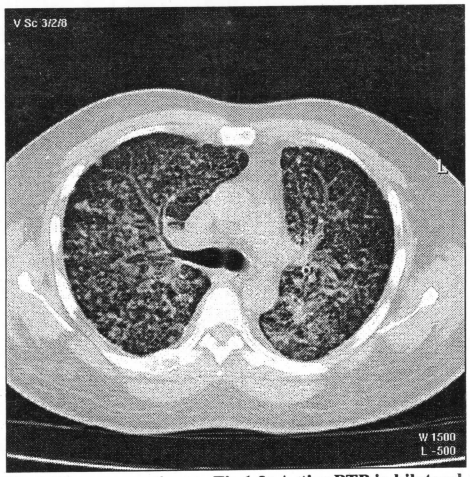

Fig 3. The same patient to Fig 1.2. Active PTB in bilateral lungs are demonstrated on chest CT

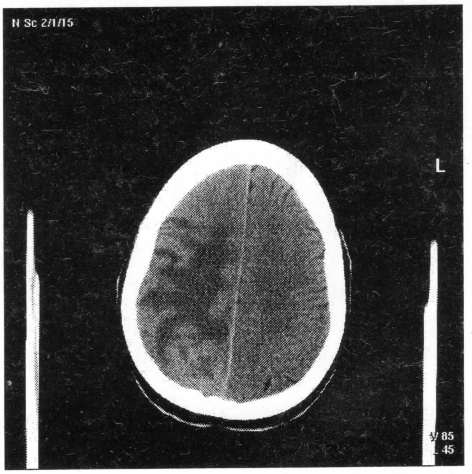

Fig 4. NECT show an isodense mass in right occipital lobe with marked surrounding edema

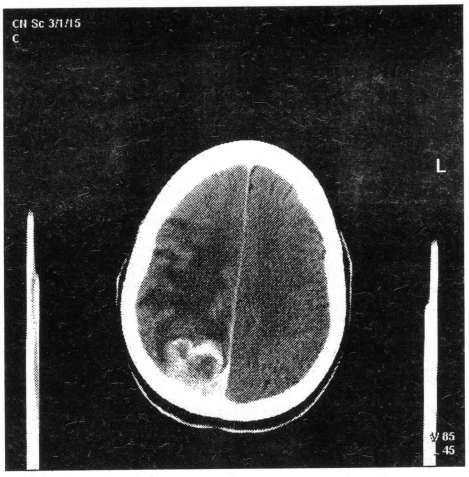

Fig. 5. The same patient to Fig 4. CECT show a 20mm x 25mm tuberculoma with rim enhacement and central necrosis in right occipital cortex

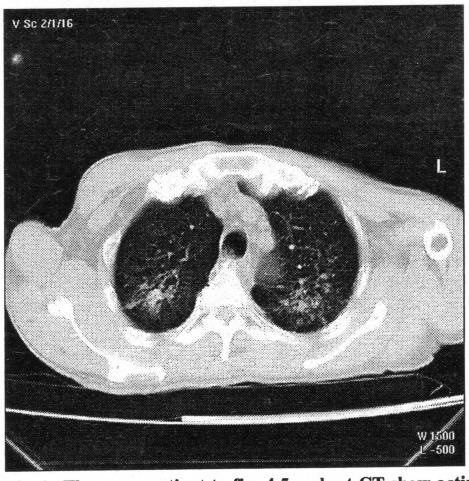

Fig 6. The same patient to fig. 4.5. chest CT show active PTB in bilateral upper lobes

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