

Isolated Tuberculous (And Klebsiella) Brain Abscesses In An Immunocompetent Nigerian Adult With Good Outcome

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Abstract

Tuberculous brain abscesses are uncommon and often found in immunocompromised patients. The first tuberculous brain abscess in 500 consecutive CT brain scans in our unit was in an immunocompetent, hypertensive Nigerian adult being managed initially as a case of hypertensive cerebrovascular disease, but later diagnosed and managed as mixed tuberculous and klebsiella brain abscesses with good outcome is discussed. The difficulties often encountered in making an accurate diagnosis, on which the management and outcome are dependent are highlighted with a review of literature.

INTRODUCTION

Tuberculosis still remains an important public health problem especially in developing countries ¹, it is said to be responsible for the death of 2 million people each year especially in low income countries. ² The most common manifestation of tuberculosis is pulmonary disease ² but central nervous involvement may occur commonly as tuberculous meningitis and rarely as tuberculomas or tuberculous abscess ^{3,4,5}. The diagnosis of cerebral tuberculosis is more difficult in the absence of concomitant extra cranial disease ^{[[5.]]} We discuss a case of tuberculous brain abscess in an immunocompetent known hypertensive woman that was clinically diagnosed as left hemispheric hypertensive cerebrovascular disease, the difficulties at arriving at a correct diagnosis, which determines the prognosis, are discussed.

CASE PRESENTATION

A 50 year old female teacher was hospitalized because of 4 months history of throbbing, frontal headache and weakness of right upper and lower limbs of two weeks duration. She was a known hypertensive patient with poor drug compliance, but not diabetic. No previous history of pulmonary tuberculosis or exposure to tuberculous patient.

The central nervous system revealed a conscious patient not oriented in time, place and person with impaired attention and short term memory; low mood and retarded speech.

There was bilateral 6th and unilateral right VII, X and XI

cranial nerve palsy; her gait was hemiplegic.

The muscle bulk was normal, no fasciculation but reduced power on the right (Grade 2 for both upper and lower limbs) and increased tendon reflex (on the right). The plantar response was equivocal.

The pulse rate was 74b/m with admitting blood pressure of 160/120mm Hg, the Jugular venous pressure was not raised and the precordial activity was normal. The 1st and 2nd heart sounds were heard, no murmur. The respiratory and abdominal systems were within normal limits.

A diagnosis of left hemispheric hypertensive cvd was made. The full blood count, random blood sugar, urinalysis, electrolytes and urea with creatinine and electrocardiography were normal.

The ESR was raised. – 50mm / HR (westergreen). However, due to cost implication, Computerized Tomography Scan was not done until 4 weeks after admission.

Computerized Tomography Scan showed multiple rounded thick-walled hypodense lesions in the left frontal and parietal lobes. These lesions showed ring enhancement while few showed homogenous enhancement after intravenous contrast. None showed calcification target signs. There was severe perilesional edema and mass effect. No hydrocephalus seen (Fig 1 and 2).

Figure 1



Figure 2



Differential diagnoses of Tuberculous brain Abscess, Tuberculomas, glioblastoma Multiformes and metastatic deposits were considered.

Left frontal craniotomy was done and multiple cystic and solid masses containing thick yellowish pus were found in the left frontal lobe. Few of these abut on the corpus callosum. The solid masses had tough craggy parts. Each of

these masses was sent in different specimen bottles for pathologic evaluation.

The histopathology report showed multiple pieces of grayish-white soft tissue masses, covered in some areas by fibrinopurulent exudates. The largest measured 5 x 3 x 2.5cm and the smallest 2 x 1x 0.5cm. The cut surfaces were similar showing cystic tissues whose rough nodular walls were composed of grayish white tissue and yellow areas. The content of one of the cyst was thick purulent material with greenish tinge.

Microscopy section showed the walls of the cysts were densely infiltrated by numerous dead and dying polymorphonuclear leucocytes, lymphocytes and some plasma cells. The stroma was edematous and composed of proliferating fibroblasts with numerous thick walled vascular channels. There were areas of hemorrhage and necrosis but no definite granulomatous reactions seen.

Microscopy, culture and sensitivity showed turbid yellow pus with 5-6 white blood cells, 8-9 red blood cells/HPF and cultured klebsiella (sensitive to fortwin and cefuroxime) and mycobacterium tuberculosis.

Some of the specimens showed abundant Acid-alcohol fast bacilli on Ziehl Nielsen staining.

DISCUSSION

Brain abscess is a focal suppurative process of brain parenchyma that can arise by extension from contiguous infection ⁶, penetrating head injury ⁷, neurosurgical procedures or hematogenous spread from extra cranial sources ⁹. Most frequently isolated microorganisms are viridians streptococci, staphylococcus aureus, gram-negative bacilli and anaerobes ⁹.

Tuberculous brain abscess is rather rare ^{3,4,6} and few reports are available among the patients without AIDS ⁹. Prior to HIV epidemic, only 17 cases were reviewed by Whitener from 1896 to 1978; ⁴ and three cases found in 30,000 autopsies in an historical study ¹.

Although the HIV/AIDS pandemic, emergence of multiresistant tuberculosis and intensification of travel/migration are expected to contribute significantly to the spread and prevalence of tuberculous (and tuberculosis brain abscess) in both developing and industrialized countries, ⁵ only two cases were found in brain neuropathology study of 174 autopsied AIDS patients by

Mathiessen et al¹⁰ and one case in 52 autopsies of HIV – infected patients by Moskowitz et al¹¹. Unexpectedly, this case presentation is the first seen in 500 computerized Tomography brain Scan in an environment with high prevalence of pulmonary tuberculosis.

Cerebral tuberculosis manifests predominantly as tuberculous meningitis followed by tuberculoma;^{2,3,4,6} other forms of manifestation include cerebral abscess, cerebral miliary tuberculosis, tuberculous encephalopathy, tuberculous encephalitis and arteritis¹². It is commoner in immunocompromised patients than immunocompetent individuals^{1,5,12}. The histopathology features of focal lesions due to cerebral tuberculosis are of two different varieties tuberculoma and abscess. Abscesses, unlike tuberculomas are usually single, larger and grow worse rapidly and do not demonstrate either giant cells or granulomatous reaction of epithelioid cells but are characterized by vascular granulation tissue with acute and chronic inflammatory cells resembling pyogenic brain abscess^{4,5}. The cerebral lesions in the case presented were multiple with presence of vascular granulation tissue and inflammatory cells and no giant cells nor granulomatous reaction so seen.

The clinical diagnosis of cerebral tuberculosis may be difficult because of the non-specific clinical presentation of patients and its early clinical course which is compatible with most other bacterial, fungal, or parasitic infections of the central nervous system as well as non infectious disease of the CNS¹². The case being discussed was initially diagnosed as hypertensive CVD.

The clinical features of cerebral tuberculosis may include malaise, fever, anorexia, fatigue, myalgia and headache in the initial stage. Others are photophobia, fever, headache, nausea, vomiting, neck stiffness and a variable degree of mental status abnormality¹². These symptoms are not modified by HIV infection¹³ and the duration may vary from one month to 9 months¹⁴. History of contact with TB can be more helpful (70-90%) than the clinical features of the patients (50%)¹⁵.

Clinical signs may also include Hydrocephalus, especially in children, clinical nerve palsy, hemiparesis papilloedema and seizure disorders.¹² Behavioral changes consisting of apathy, confusion or bizarre behaviors may progress to lethargy, stupor and coma¹². However, tuberculous abscess may be distinguished from tuberculoma by its acuteness and

accelerated clinical course. Abscess (tuberculous) usually present with fever, headaches and focal neurological deficits and common in the supratentorial region⁴ but tuberculomas are usually devoid of fever and signs of systemic infection. The patient being discussed had no history of fever but there was history of headache, behavioral changes, and focal neurological lesion.

The absence of fever may not be unconnected with ease of purchasing antibiotics and analgesics from drug stores often without prescription in this environment, which may explain why the patients must have 'coped' with the headache for several months and may contribute to late presentation. Also, not all patients with tuberculosis brain abscess present with fever.¹ The diagnosis of cerebral TB cannot be made on excluded on clinical grounds alone but in conjunction with neuroimaging, cerebrospinal fluid findings, evidence of extra neural TB and appropriate microbiological, and or histopathological finding.¹²

On neuromaging, a true tuberculous abscess may be indistinguishable from pyogenic abscess on CT and MRI It usually appears as a hypodense lesions with ring enhancement on intravenous contrast. It can be large, multiloculated with marked surrounding edema. There features may be indistinguishable from neoplasms and granulomas like sarcoidosis, cysticercosis, toxoplasmosis, pyogenic and fungal lesions^{1,12} while tuberculomas appear on CT as low or high density rounded masses with ring or homogenous enhancement after intravenous contrast and mild perilesional edema, often in frontal and parietal lobes and may show target sign⁶.

The cranial CT in this reported case showed multiple rounded masses with sizes ranging from 1.9cm – 4cm in the left frontal and parietal lobes, with extensive perilesional edema; which are more in keeping with tuberculous abscess but unlike tuberculous abscess, some of the lesions showed homogenous enhancement.

This difficulty in accurately differentiating brain tuberculoma from tuberculous abscess makes biopsy imperative¹⁷. Magnetic Resonance imaging is more sensitive than CT and provides a more precise evaluation of TB lesion in the brain, CSF analysis also aid in arriving at the diagnosis, for CNS tuberculosis generally, a demonstration of AFB in the CSF by microscopy smear and by culture is the gold standard and isolation rate is higher in ventricular and cisterna than lumbar CSF; cytology and biochemistry may

also be suggestive. However, in cases of tuberculomas and abscesses, the csf analyses may be unremarkable and histology remains the gold standard as approximately 60% of tissue specimens will show AFB in smear and culture ¹².

The sophisticated molecular diagnostic approaches which are less time consuming and more sensitive than the microscopy and culture techniques ¹² are not available in most developing countries and have inherent problems such as use of various antigenic sources, antibody sources, cross reactivity and heterogeneous host immune responses ¹².

The treatment of tuberculous brain abscess includes simple puncture, continuous drainage, fractional drainage, repeated aspiration through burrholes, stereotactic aspiration and total excision. ¹⁸ These surgical options are combined with antitubercular therapy ^{1,12,18}.

In cerebral tuberculosis, the prognosis is good if promptly diagnosed and treated early. ^{1,12} However, 20-25% of which includes survivors may manifest a variety of neurological sequelae which includes mental retardation, psychiatric disorders, seizures, blindness, deafness, ophthalmoplegia, hemiparesis and endocrinopathies. Our patient is doing very well and none of these complications developed after two years of follow up.

CONCLUSION

Tuberculous brain abscess is rare and may be simulated by other infectious, parasitic and neoplastic space occupying lesions.

It has good prognosis if accurate diagnosis is made and prompt treatment initiated. A high index of suspicion and a multidisciplinary approach system i.e. clinical features, neuro-imaging and pathological evaluation are required to make accurate diagnosis.

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