

CASE REPORTS

Imaging features of brain tuberculoma in Tanzania: case report and literature review

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SUMMARY

Brain tuberculomas are a rare manifestation of mycobacterium tuberculosis infection usually resulting from hematogenous spread of the bacteria from a primary focus elsewhere in the body. A 29-year-old female with no history of pulmonary tuberculosis or signs of pulmonary infection presented with signs and symptoms of raised intracranial pressure.

She underwent CT and MR imaging where multiple enhancing lesions were revealed in the brain parenchyma. The features of tuberculoma on CT and MR imaging may mimic the appearance of several other brain lesions. Histological diagnosis of tuberculoma was obtained. In areas where tuberculosis is endemic, the imaging features of brain tuberculoma have to be readily recognized by attending doctors.

Keywords: Brain tuberculoma; computed tomography; magnetic resonance

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Introduction

Brain tuberculomas are a manifestation of infection caused by mycobacterium tuberculosis. They usually result from hematogenous spread from a primary focus evident or dormant elsewhere in the body^{1,2,3}.

The imaging features of tuberculoma have been a subject of interest and many publications on this subject are available in literature¹⁻⁴. However, in most developing countries the diagnosis of this disease has been impeded by the lack of diagnostic facilities such as Computed tomography (CT) and Magnetic resonance (MR) imaging⁴.

Although tuberculosis is common in Tanzania, there is no report on brain tuberculoma available from Tanzania.

We present the imaging features of brain tuberculoma in a young non-HIV female. We also present a literature review with a perspective on imaging brain tuberculomas in a developing country.

Case Report

A 29-year-old female presented at our hospital with a 6-month history of headaches, generalized convulsions and weakness of right extremities. She also reported to have low-grade fever of almost the same duration.

On physical examination she was found to have papilloedema otherwise no other abnormality was found. Hematological examination revealed a raised ESR and lymphocytosis. She tested negative for the human immunodeficiency virus (HIV). Electroencephalography results were normal. No abnormalities were seen on her chest and skull x-rays.

Pre and post contrast CT examinations were performed. The pre contrast images revealed multiple Hypodense areas with poorly defined margins. Contrast enhanced CT (CECT) revealed multiple ring enhancing lesions with peripheral edema involving both cerebral hemispheres. No midline shift was noted (See figure 1).

The MR T1 weighted images showed multiple focal lesions which were slightly 'hypodense. On the T1 weighted post contrast images the lesions appeared brightly enhanced (See figure 2). The T2 weighted images showed multiple lesions with hypointense peripheries and central cores of varying degrees of brightness surrounded by high intensity edema.

Following the CT and MR imaging a diagnosis of space occupying lesion was made. The differential diagnosis included lymphoma, metastasis, cysticercosis, tuberculoma and pyogenic abscess.

CT guided Stereostatic biopsy was performed and histological diagnosis of tuberculoma was made. Histology showed giant cells, caseation, lymphocytes and epithelioid cells. Post operatively the patient was put on long term anti tuberculous chemotherapy.

Follow up CT examination 8 months after initiation of chemotherapy revealed complete resolution of the lesions.

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Discussion

Central nervous system tuberculosis is endemic in certain regions of the world and recently the prevalence of tuberculosis has been on the rise world wide as a result of the increased number of AIDS cases⁵.

Since prompt diagnosis of brain tuberculomas may result in earlier treatment and better prognosis, recognition of this disorder on radiological images may play a critical role in patients' management. When brain tuberculomas are associated with meningitis, the diagnosis is more apparent, and appropriate therapy can be readily instituted⁶. However, therapy may be delayed when the tuberculoma give rise to neurological symptoms without evidence of meningitis and when the spinal profile is normal. Previous series⁷ have shown that it is impossible to differentiate tuberculomas from other masses on the basis of neurological symptoms.

Tuberculomas may be solitary or multiple and may grow intraparenchymally, or have a combined meningeal and parenchymal course⁴.

With the increasing availability of imaging modalities such as CT scanners in Tanzania as well as in other developing countries where tuberculosis is endemic, there is a need for the imaging features of brain tuberculoma to be more readily recognized by the attending doctors.

The chest X-ray of our patient was normal with no evidence of tuberculosis. The foci may have been quite small and unidentifiable on routine radiographs. The absence of features of tuberculosis on chest X-rays should therefore not rule out the possible existence of brain tuberculomas^{1,2,4}. In a series of 70 patients with brain tuberculoma, only 30.8% revealed a positive chest radiograph⁴.

The skull x-ray was normal in our case. Evidence of calcification on skull x-rays may be present in only 6% of the cases of brain tuberculosis.

CT and MR are the main imaging modalities used in the localization and characterization of brain tuberculomas; prior to their advent diagnosis was usually confirmed at either surgery or autopsy^{1,4}.

The CT findings in our case, multiple ring enhancing lesions have been reported previously^{2,4}. It is important to recognize, however, that the CT findings of brain tuberculoma may be variable

since tuberculomas are basically evolving granulomas. During the acute stage non-contrast enhanced CT may show only a hypodense area caused by cerebritis or it may be normal. At the established inflammatory granulomatous stage, the lesion is either isodense or more commonly hypodense with a poorly defined outline on pre-contrast images and has marked enhancement following contrast. At the stage of central caseation, the tuberculoma is either hypodense, or less commonly, isodense or slightly hypredense on pre-contrast images, rarely small central calcifications may be seen. Post contrast images at this stage may show ring-like appearance^{1,2}. Occasionally a target sign is seen that consists of ring enhancing lesions. These features are however not pathognomonic of tuberculoma and can be seen in other conditions was evident from the differential diagnosis made for our patient after CT and MR examinations^{4,5}; furthermore, the differential diagnosis depends on the stage of development of the tuberculoma. These conditions include cystercosis, pyogenic abscess, gliomas, lymphoma and metastasis^{1,2,6}.

The diagnosis of brain tuberculoma basing on CT alone is presumptive and should be supported by findings such as history of fever, high ESR, positive tuberculin test and positive response to anti tuberculous treatment¹. CT is reported to have a sensitivity of 100% and specificity of 85.7 %, with the negative predictive value of 35% thus indicating a need for further analysis with MR and or histological diagnosis⁸.

The MR findings in our case were typical and have been previously reported¹. The lesions appeared isointense on pre-contrast T1-weighted images and with central hyperintense regions with hypointense rims on T2 weighted images. The hypointense regions on T2 weighted images are associated with increased fibrosis, gliosis and macrophage infiltration. On contrast enhanced T1 weighted images the lesions appeared enhanced and some presented as ring enhanced lesions.

The importance of use of contrast in CT and MR images when tuberculomas are suspected has been reported. Chang et al⁹ reported that without contrast enhancement on MR, the images were generally insensitive to detection of both meningeal inflammation and tuberculomas. Gupta et al¹ reported that the MR features of brain tuberculomas are more specific than those of CT. Other authors have concluded likewise¹⁰. With application of new MR techniques such as proton spectroscopy and diffusion weighted imaging the MR specificity can be significantly increased^{10,11}. A further advantage of MR is the multiplanar capability and the ability to show small skull base lesions can be overlooked on CT.

In our case histological diagnosis was made by

use of CT guided stereostatic biopsy (CTGSB). This procedure is widely used because of its less invasiveness compared to open biopsy. However, CTGSB is not always diagnostic and in those cases open surgery has to be done. In developing countries this facility may be less available and so open surgery may be used to provide biopsy for histological diagnosis

Some authors¹² have recommended a trail of anti-tuberculous drugs for patients with multiple enhancing intracranial space occupying lesions without mass effect in areas lacking advanced neurosurgical facilities.

CT and MR can adequately provide follow up studies for patients with brain tuberculomas. The tuberculomas in our patient were shown to have resolved on follow up CT performed 8 months after initiation of chemotherapy. Remarkable improvement can be evident on CT and MR 6 weeks after initiation of medical treatment and complete resolution in 12 weeks^{4,12}.

In conclusion, CT and MR provide essential information that aids in diagnosis of brain tuberculoma. It should be noted that diagnosis of brain tuberculoma is difficult because the imaging presentation is varied and can be non-specific; other parameters may be required to establish the definite diagnosis. MR is reportedly superior to CT for diagnosis of brain tuberculomas; however, MR is expensive and in developing countries this facility may not be readily available. Where available it should be the technique of choice.

References

1. Gupta RK, Jena A, Sharma A et al. MR imaging of Intracranial Tuberculomas. JCAT 1988; 12 :280-285
2. Draouat S, Abedenabi B, Ghanem M and Boujrat P. Computed Tomography of cerebral tuberculoma. JCAT 1987; 11:594-597
3. Bhargava S, Tandon PN. Intracranial tuberculomas. A CT study. Br J Radio! 1982; 53 : 935-945
4. Jinkins JR. Computed tomography of intracranial tuberculosis. Neuroradiology 1991; 33:126-135
5. Hopewell PC. Overview of clinical tuberculosis. In: Boon BR, editor. Tuberculosis: Pathogenesis, protection and control. Washington,

DC: American Society of Microbiology; 1994: 25-46.

6. Whelan M, stern J. Intracranial tuberculoma. Radiology 1981; 138: 75-81
7. Dastur HM, Desia AD. A comparative study of brain tuberculomas and gliomas based on 107 case records of each. Brain 1965; 88:375-396
8. Selvapardian 5, Rajeshkhar V, Chandy MI et a Predictive value of computed tomography based diagnosis of intracranial tuberculomas. Neurosurgery 1994; 35:845-850
9. Chang KH, Han M, Roh JK, Kim OH, Choi KS, Kim CS. Gd-DPTA enhanced MR imaging in intracranial tuberculosis. Neurology 1990; 32:19-25
10. Kaminogo M, Ishihasu H, Morikawa M, et al. Proton MR spectroscopy and diffusion weighted MR imaging for the diagnosis of intracranial tuberculomas. Report of two cases. Neurol Res 2002; 24:537-543
11. Gupta RK, Kathuria MK, Pradhan S. Magnetization transfer MR imaging in CNS tuberculosis. AJNR 1999; 20:867-875
12. Abdul-Ghaffar NU, El-Sonbarty MR, Rahman NA. Intracranial tuberculoma in Kuwait. mt J Tuberc Lung Dis 1998; 2:413-418
13. Ramamurthi B, Ramamurthi R, Vasudevan C, Sridhar K. The changing face of tuberculoma. Ann Acad Med Singapore 1993; 22:852-855

Figure Legends

Figure 1: Brain CT images of our patient. (a) and (b) show the pre-contrast and post contrast images. Note the ring-enhancing lesion seen on the left basal ganglia on post contrast image (a), this lesion cannot be clearly identified on the pre-contrast image (b). Post contrast images at another level showing multiple parenchymal lesions which have enhanced brightly and thus can be easily identified

Figure 2. MR Ti-weighted images of our patient. (a) Pre-contrast images and (b) post contrast images. Note the brightly enhanced lesions seen on (b) which can be seen with difficulty on (a) where they appear iso-slightly hyperintense. Slight edema is seen represented as hypointense areas around the lesion.

Figure 3 MR Ti-weighted images at a different level from Figure 2, (a) precontrast and (b) post contrast images showing multiple lesions. The lesion appear brightly enhanced on (b) and are easier to identify than on (a) where they appear iso to-hyperintense (c) shows a saggital section demonstrating the multiplanar capability of MR imaging.

Figure 1(a)

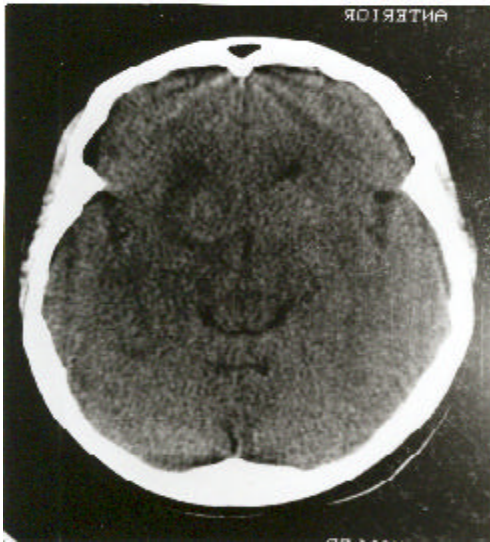


Figure 2(a)

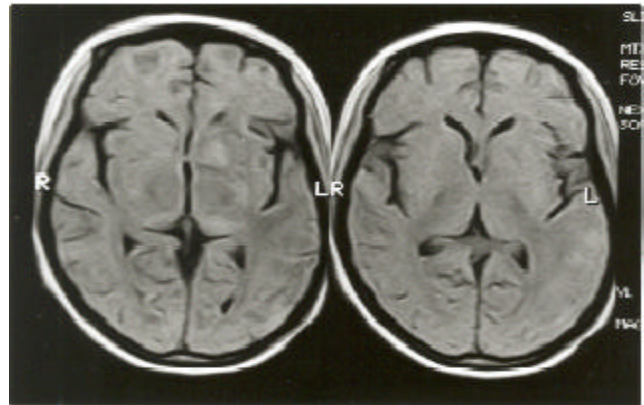


Figure 1(b)

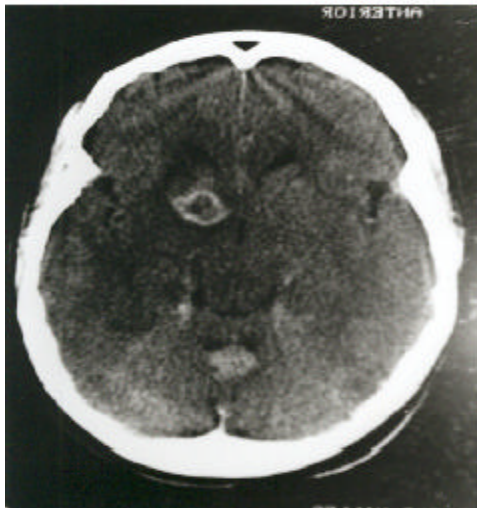


Figure 2(b)

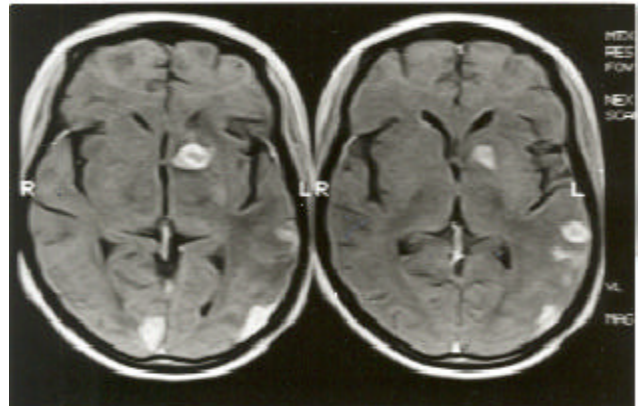


Figure 1(c)

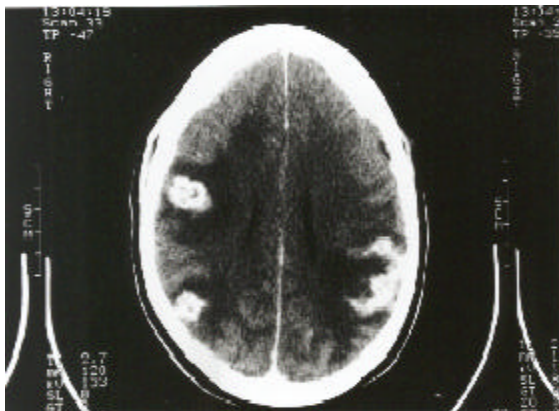


Figure 3(a)

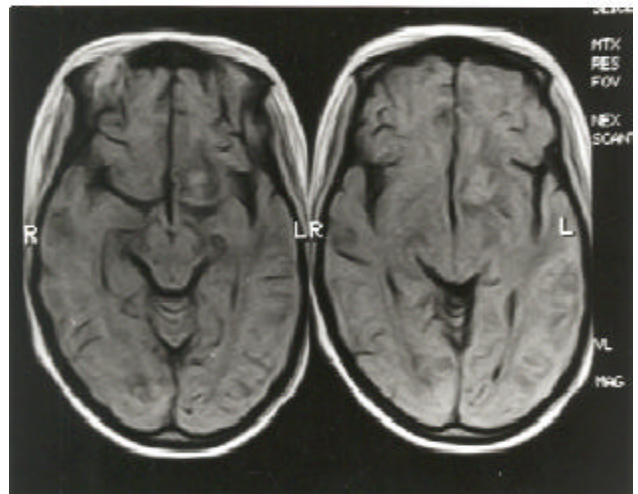


Figure 3(b)

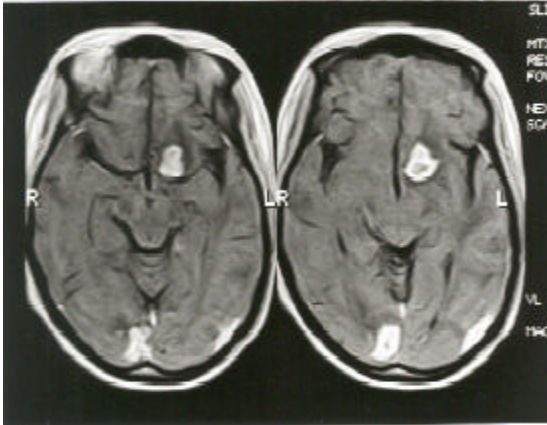


Figure 3(c)



Ninety Years ago

Syphilis and bubo

The patient was admitted on January 1st 1913.

The patient had local sores for 3 weeks. For one week has had a left bubo.

Present condition: *Has extensive local ulceration beneath the prepuce, and the penis is enlarged. The skin over the scrotum and left groin is unhealthy and shows Kiganda medicine. There is a fairly large bubo on the left side.*

Treatment:

- 1. To have a thorough wash*
 - 2. To paint on iodine and forment b.d.*
 - 3. Cleanse local sores with hydrogen peroxide*
- 8th January – Discharged cured.*

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