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ABSTRACT

Introduction
Intracranial tuberculoma mostly occurs in supratentorial location in adult patients and there are isolated cases with Infratentorial tuberculoma reported in the literature in the last 10 years.

Case Report
A 49-year-old man presented with progressive staggering gait and headache for 3 months and was diagnosed with a giant cerebellar tuberculoma.

Conclusion
Although rare, our case demonstrates that tuberculoma should be considered in the differential diagnosis of infratentorial tumor in immunocompetent adults.

Keywords: Posterior fossa; Tuberculoma; Magnetic resonance imaging; Diagnosis.
INTRODUCTION
Neurological tuberculosis comprises of 10-15% cases of extra pulmonary tuberculosis. [1-2]
The focal forms of central nervous system tuberculosis are infrequently described and they are divided into tuberculoma and abscess. [3]
Central nervous system tuberculomas or tuberculous granuloma is uncommon, it is a granuloma formed by the inflammatory response to Mycobacterium tuberculosis infection. [4]
Cerebellar or brainstem tuberculomas are much less commonly found in the cases of adults. [5]
There are isolated adults cases with Infratentorial tuberculoma reported worldwide in the last 10 years. [6-7]
Because of its rarity in immunocompetent patients and the difficulty in preoperative diagnosis, we have illustrated this case. We describe the clinical manifestation, imaging, pathologic findings, management, and outcome associated with such lesion.

CASE REPORT
History and Presentation
A 49-year-old right-handed male patient without any other medical history was admitted complaining of progressive staggering gait and headache for 3 months.

Physical Examination Findings
General physical examination was normal.
A neurologic examination showed cerebellar signs, including cerebellar ataxia, dysmetria, and dysdiadochokinesia. The fundus oculi was normal.
Routine blood tests were normal and the chest X-ray was normal. The patient was HIV-negative.
Radiologic Findings
A cerebral computed tomography (CT) scan showed a midline vermian lesion extending to the left cerebellar hemisphere. The lesion was spontaneously hypodense with mass effect on the fourth ventricle (Figures 1).

Cranial magnetic resonance imaging (MRI) showed irregular contours of mass lesions. The processes were heterogeneous on T1- and T2-weighted images with discrete surrounding edema. After gadolinium was administered, the lesion was 3×4cm in size and shows multiple nodules of intense enhancement (Figures 2).

Surgical Findings
A suboccipital craniotomy was performed while our patient was in the prone position. After opening of the dura mater and corticectomy, the lesion was white, infiltrative, with a firm consistency, and not hemorrhagic, had no planes of cleavage. A complete tumor resection was performed. The postoperative course was satisfactory.

Histopathological Findings
Histopathological examination confirmed the diagnosis of tuberculoma (Figure 3). Daily antituberculous chemotherapy with isoniazid, rifampicin, pyrazinamid and ethambutol was started and the patient was referred to the department of Infectious Diseases for follow-up.

Follow up
Two months later, the patient was discharged to complete 8 months of antituberculous treatment. 14 months after surgery, our patient was improved clinically and he has very mild residual cerebellar ataxia. Postoperative control MRI study was performed 1 year after surgery and there were no signs of recidive (Figure 4).

DISCUSSION
Intracranial tuberculoma mostly occurs in supratentorial location in adult patients and in children it frequently occurs in infratentorial region and they are commonly found
in the frontal and the parietal regions. [5] There are isolated adults cases with cerebellar tuberculomas reported in the literature. [7]

Despite being preventable and curable, diagnosis of infratentorial tuberculoma remains difficult. Parenchymal tuberculomas show a typical granulomatous reaction consisting of epithelioid cells and giant cells mixed with predominantly lymphocytes around a central area of caseating necrosis. Any liquefaction of the central area of necrosis contains clear or straw-colored fluid, as opposed to pus. [8]

Posterior fossa tuberculoma poses a more risk to the patient life than supratentorial one. They more commonly arise as solitary lesions without evidence of systemic tuberculosis, mimicking tumors rather than the infectious process. [6]

Clinical manifestations included various combinations of focal signs and symptoms, similar to those produced by other lesions in the brain stem and cerebellum. Our review of the literature describing infratentorial tuberculoma, intracranial hypertension and localising signs (a cerebellar syndrome and a syndrome of the ponto-cerebellar angle) may all occur. In our patient, preoperative symptoms included a cerebellar syndrome.

There is no imaging technique that can differentiate tuberculoma reliably from other intracranial mass lesions. [9] On CT Scans, tuberculomas may be isodense, hyperdense round or lobulated masses with irregular walls showing homogenous enhancement after contrast administration. They typically appear as ring-enhancing lesions with surrounding vasogenic oedema. [10] MR imaging plays a crucial role in diagnosis because of its inherent sensitivity and specificity in detecting such lesions earlier than CT. Non-caseating tuberculoma: it is usually iso-/hypo-intense on T1 and hyper-intense on T2-weighted images. Homogeneous enhancement is seen with gadolinium. Caseating solid tuberculoma: it is usually hypo-intense on T1 and strikingly hypo-intense on T2-weighted images. This relative hypo-intensity is attributed to the granulation tissue and compressed glial tissue in the central core resulting in greater cellular density than the brain parenchyma.

Tuberculoma with central liquefaction: it appears centrally hypointense on T1, and hyperintense on T2-weighted images with a peripheral hypo-intense rim on T2W images. The low signal intensity of the capsule may be related to a layer of collagenous fibres with high protein concentration and low water content and a layer
of outer inflammatory cells. Gd-DTPA-enhanced T1W images show rim enhancement
in caseating granulomas. [11-12] On MR spectroscopy, tuberculomas are
characterized by elevated fatty-acid spectra. This technique is being increasingly
used now a day to differentiate tuberculomas from other intracranial mass lesions.

Unfortunately, for our case, spectroscopy hadn’t been proposed in the the
preoperative MRI study. The degree of perilesional edema has been described as
being inversely proportional to duration of the lesion. [14]

The differential diagnoses of infratentorial solitary tuberculoma are metastatic
tumours, lymphomas, toxoplasmosis, abscesses, meningioma, neurinoma, gliomas,
cysticercosis… [15].

Mainly central nervous system tuberculomas are treated with antitubercular drug, a
minimum of 10 months treatment is required. [16] Isoniazid, rifampin, pyrazinamide
and ethambutol are the four-drug regimen that is most commonly recommended.

Adjuvant corticosteroid therapy is indicated when there is peri-lesional oedema or
paradoxical progression during treatment. [17]

The indications of surgery today are very small because parenchymal tuberculomas
completely disappear with conservative treatment in the majority of cases. [18] The
two main indications remain the same where there is diagnostic confusion from
infratentorial tumors and where there are features of acute rise in intracranial
tension in spite of medical treatment. [18] Tuberculomas may resolve, decrease or
increase in number and size, or remain unchanged after completion of therapy. [19]

Total resolution of the tuberculoma is observed when scans demonstrate no
enhancing lesions or only an area of calcification or only an area of calcification. [17]

Patients usually do not need the follow-up after completion of therapy if signs or
symptoms do not recur. [20]

CONCLUSION

Cerebellar tuberculomas are relatively rare but diagnosis should be kept in mind
when confronted with infratentorial masses.
They are generally difficult to diagnose and the delay of recognition and treatment of this rare location can lead to increased mortality and morbidity rate.

CONFLICT OF INTEREST

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AUTHOR’S CONTRIBUTIONS

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REFERENCES


FIGURE LEGENDS

Figure 1: Unenhanced Cranial CT scan CT revealed low-attenuation vermian mass extending to the left cerebellar hemisphere and compressing the fourth ventricle.

Figure 2: Magnetic resonance imaging of the brain showing a large (4*3 cm), well-defined, vermian lesion extending to the left cerebellar hemisphere with irregular margin. It was hypointense on T1-weighted images (a) and giving heterogeneous high signal on T2-weighted (b) with perilesional edema. A contrast-enhanced T1-weighted image shows multiple nodules of intense enhancement (c and d). On diffusion-weighted imaging (DWI) sequences, the lesion appeared to be slightly lower signal (fig: e true diffusion \((b=1000 \text{ s/mm}^2)\) and displayed significantly higher averaged apparent diffusion coefficient (ADC) value (f).

Figure 3: Photomicrograph of histologic examination demonstrated granulomatous inflammation composed of aggregation of epithelioid histiocytes associated with giant cells and lymphocyte cuffing foci of caseating necrosis under low and high magnification (a, H&E × 200; b, H&E × 400). Staining for vimentin (c), epithelial membrane antigen (EMA) (d), cutokeratine (e) and CD10 (f) was negative.

Figure 4: The postoperative brain magnetic resonance imaging made 14 months after surgery, axial (a) T1-weighted, axial (b) T2-weighted, axial (c) and sagittal (d) T1-weighted after contrast, showing the disappearance of the lesion.
FIGURES

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