Surgical Neurology International

OPEN ACCESS

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Editor: **James I. Ausman,** MD, PhD University of California, Los Angeles, CA, USA

Case Report

Paradoxical evolution of a cerebellar tuberculosis abscess after surgical drainage and antibiotic therapy

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Received: 01 May 14 Accepted: 14 July 14 Published: 30 September 14

This article may be cited as:
Joshi V, Germano I, Meenakshi R, Doshi A. Paradoxical evolution of a cerebellar tuberculosis abscess after surgical drainage and antibiotic therapy. Surg Neurol Int 2014;5:143.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2014/5/1/143/142033

Abstract

Background: Involvement of the central nervous system (CNS) by a tuberculosis abscess is a rare form of extra-pulmonary tuberculosis. With proper treatment, the abscess most commonly follows a pattern of continued reduction in size.

Case Description: A 71-year-old male with a past medical history of kidney transplant on immunosuppressive therapy, presented to the hospital with a 1-day history of headache. On physical examination, the patient had no focal neurological symptoms. Initial laboratory reports were unremarkable. Contrast enhanced magnetic resonance imaging (MRI) was performed, which showed a ring enhancing mass and perilesional edema in the left cerebellar hemisphere. The patient underwent a left posterior fossa biopsy and drainage. The lesion was encapsulated with a purulent center. Cultures revealed pan-sensitive mycobacterium tuberculosis and the patient was started on rifampicin, isoniazid, pyrazinamide, ethambutol, and B6. The patient was monitored carefully and brain MRIs were obtained at 1, 4, 9, 11, and 14 months. It was noted that the tuberculosis abscess had grown in size from month 4 to month 9 of treatment. Since the patient's neurologic examination and symptoms were stable at that time, the drug regimen was not changed. The 14-month follow up MRI showed that the abscess had nearly resolved.

Conclusion: Rarely, the pattern of CNS tuberculosis abscess evolution may include growth, even with proper treatment. This pattern does not necessarily signify treatment failure, as our abscess resolved without change in treatment. Given the possibility of asymptomatic abscess enlargement, close clinical and imaging follow up are crucial in management of these cases.

Key words: Central nervous system, cerebellum, tuberculosis abscess

Access this article online

Website: www.surgicalneurologyint.com

DOI: 10.4103/2152-7806.142033

Quick Response Code:

INTRODUCTION

Central nervous system (CNS) tuberculosis abscess is a rare form of extra pulmonary tuberculosis with high morbidity and mortality rates internationally.[6] A tuberculosis abscess is a focal collection of pus surrounded by a dense capsule containing acid fast bacilli compatible with mycobacterium tuberculosis.[6] With proper treatment, the most common pattern of abscess evolution is gradual reduction in size.[5] We present a rare case of a pan-sensitive CNS tuberculosis abscess affecting the cerebellum of an adult, with paradoxical enlargement

upon treatment and follow-up imaging, and eventual resolution without change in treatment.

CASE REPORT

A 71-year-old male with a past medical history of kidney transplant on immunosuppressive therapy, diabetes, and hypertension presented to the hospital with a 1-day history of headache, nausea, and vomiting. On physical examination, the patient had no focal neurological symptoms, and normal blood pressure. Initial laboratory reports showed an unremarkable cell blood count. Basic metabolic panel, liver function tests, and coagulation panel parameters were all within normal limits. Blood cultures and urine analysis were both negative. Prior purified protein derivative (PPD) tuberculin test and human immunodeficiency virus (HIV) results were negative.

Chest X-ray demonstrated a stable right upper lobe cavitary lesion that had been biopsied twice previously with nondiagnostic results. A head computer tomography (CT) was obtained, which demonstrated a cystic mass in the left cerebellum with perilesional edema [Figure 1]. Further evaluation with contrast enhanced MRI was performed that showed a well circumscribed left cerebellar hemisphere ring enhancing mass on T1 sequences with restricted diffusion, mass effect, and perilesional edema [Figure 1]. Differential diagnosis included pyogenic abscess (bacterial or fungal), neurocysticercosis, cystic glioma, or other cystic malignant/metastatic process.

The patient underwent a left posterior fossa biopsy and drainage. The lesion was well encapsulated with a frankly purulent center.Numerous acid fast bacilli were seen on acid fast stain, and cultures returned positive for mycobacterium tuberculosis [Figure 2]. Cerebrospinal fluid (CSF) cultures were also obtained, which were negative. Microbiology revealed pan sensitive mycobacterium tuberculosis and the patient was started on rifampicin, isoniazid, pyrazinamide, ethambutol (RIPE), and B6.

Follow-up MRIs were obtained 1, 4, 9, 11, and 14 months [Figures 3 and 4]. During this time, the patient was seen monthly, and had continued to show clinical improvement with treatment. After 6 months, ethambutol had been removed from the treatment regimen. Treatment with rifampicin, isoniazid, and pyrazinamide was continued in order to maximize CNS penetration, and it was noted on the 9-month MRI that the tuberculosis abscess had grown in size from 2.0 × 2.0 × 2.2 to 2.5 × 3.3 × 2.3 cm [Figure 3]. While the abscess had grown in size at 9 months, the amount of perilesional edema had decreased [Figure 4b]. At this time the patient's neurologic examination and symptoms were stable, and given the continued clinical improvement, and pan sensitive cultures, the drug regimen was not changed. Due to the increase in size, vigilant surveillance with frequent clinic visits was pursued. The 11-month follow up MRI showed that the abscess size was decreased to 1.5 × 2.2 × 1.2 cm, and on month 14, the abscess had nearly resolved [Figure 3].

DISCUSSION

CNS tubercular abscess is a rare form of extra-pulmonary tuberculosis in which the typical granulomatous reaction associated with tuberculosis is not present.[4] This case fits the criteria for an abscess by having positive macroscopic evidence of abscess formation with frank pus centrally and cavity formation, and positive mycobacterial tuberculosis cultures and acid fast organism staining.[4]CNS tuberculous abscesses are generally seen in immunocompromised individuals and generally occur in the supratentorial compartment.[6] TB abscesses, less commonly, as in our case, occur within cerebellum.[6] There are few reports of true cerebellar abscesses, and

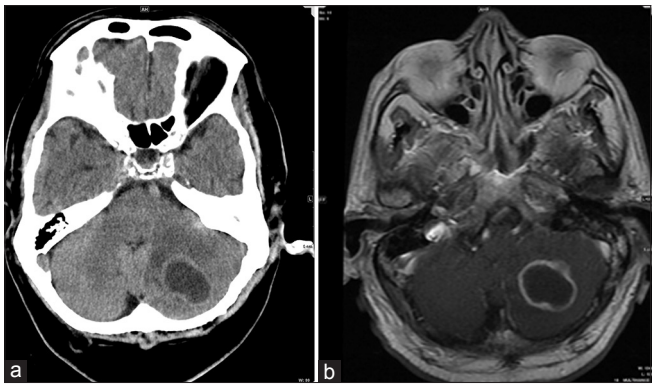

Figure 1: (a) Noncontrast head CT shows a cavitary lesion with perilesional edema and mass effect upon the 4th ventricle. (b) Contrast enhanced MRT1 sequence shows a well circumscribed left cerebellar hemispheric lesion with prominent rim enhancement. There is associated vasogenic edema compressing both the fourth ventricle and medulla

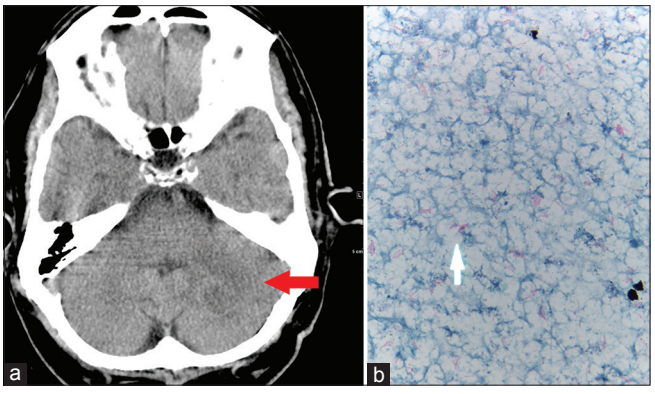

Figure 2: (a) Noncontrast head CT status post abscess drainage shows improvement in abscess size and mass affect.There is minimal residual hypodensity present. (b) Acid fast stain shows numerous stained bacilli, which correlated with culture showing pan sensitive mycobacterium tuberculousis

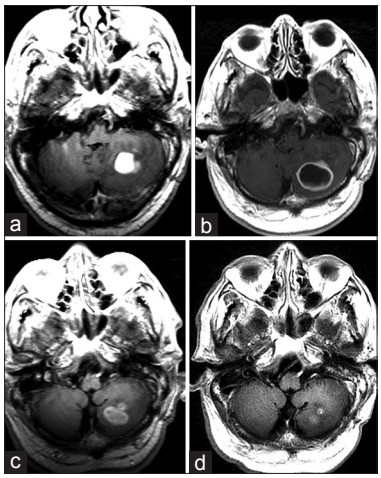

Figure 3: Evolution of TB Cerebellar Abscess. (a) Contrast enhanced axial T1 at 4-month follow up shows enhanced thickened and nodular cavity wall, which was unchanged in size or appearance from the postoperative 1 month follow up MR (not shown). (b) Axial T2 FLAIR at 9-month follow up showed increased cavitation size, but with decreased capsule thickness and nodularity. (c) Contrast enhanced axial T1 at 11-month follow up showed decreased size of the lesion, without change in treatment. (d) Contrast enhanced axial T1 at 14-month follow up shows significant reduction in size of previously noted cavitary lesion

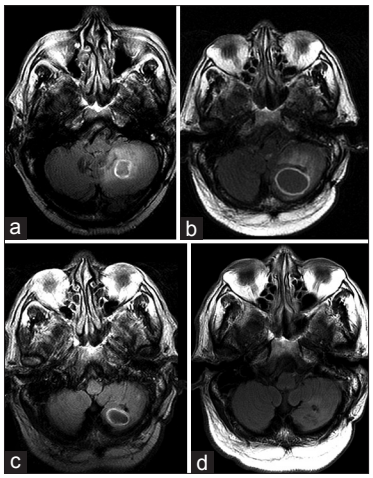

Figure 4: Evolution of TB Cerebellar Abscess on T2 FLAIR Imaging. (a) Contrast enhanced axial T2 FLAIR at 4-month follow up shows significant perilesional edema. (b) Contrast enhanced axial T2 FLAIR at 9-month follow up showed residual perilesional edema, which had decreased since prior study, even though the cavitation size had increased. (c) Contrast enhanced axial T2 FLAIR at 11-month follow up and 14 month follow up (d) showed minimal to no perilesional edema

those cases mostly involved younger patients, as opposed to the 71-year-old in our case. Due to the rarity of CNS tuberculosis abscess, more cases need to be evaluated to further assess if there is a statistically significant geographic distribution.

The radiologic appearance of CNS TB abscess on CT generally demonstrates a ring enhancing cystic lesion with perilesional edema, while MRI demonstrates a hyperintense central area with a hypontense rim on T2-weighted images, and hypointense central area with peripheral rim enhancement on T1 sequences.[4] Our case had the aforementioned characteristics. These lesions can be difficult to distinguish from pyogenic abscesses based on radiological appearance.

The consequences of untreated neurotuberculosis are devastating, and choosing the proper treatment regimen is imperative.[7] One of the most important components of treatment consists of long-term antituberculosis drug therapy.[7] Surgical drainage and excision, as in our case, can be used to obtain a microbiological diagnosis and to relieve mass effect caused by large lesions.[6,7] Most commonly, treatment of tuberculosis lesions involves a pattern of decreasing abscess size on anti-TB drug treatment.[5] Rarely reported are enlarging tuberculomas of the brain, and very rarely, as in our case, has there been reports of enlarging tuberculous abscess after both surgical drainage and RIPE treatment in an adult.[2] The enlarging tubercular abscess can present a dilemma for the treating clinician as this may potentially signify a failure of treatment/drug resistance, infection with a secondary organism, underlying neoplasm, or simply inflammatory changes.[2] Prior review of enlarging CNS tuberculosis lesions has shown the time to enlargement to occur up to 27 months, however, in most cases the enlargement occurred at 3 months from the start of therapy.[8] In our particular case, the abscess evolved during treatment from no change in size but increasing wall thickness and nodularity, to a paradoxically enlarging thin walled abscess at 9 months, to decrease in size at 11 months and subsequent near resolution at 14 months. It is interesting to note that although the abscess had increased in size at 9 months, the amount of perilesional edema had decreased from prior study [Figure 4], which may have been a sign that the abscess was following a pattern of resolution. Clinically, the patient remained stable with no focal neurological deficits and symptoms, and no mass effect on imaging, therefore treatment was not changed. Most cases of enlarging CNS tuberculosis lesions, resolve with no change in anti-TB drug therapy, however, some cases may require the addition of steroids, change in antituberculous drug therapy, and, in rare instances, surgical decompression.[1,8] Of the CNS tuberculosis lesions, CNS abscesses have been notoriously difficult to treat.[3]

While there is no agreed upon explanation as to the cause of tuberculosis abscess enlargement, several theories exist.[5,8] One theory suggested is the restoration of the blood–brain barrier during treatment possibly reduces drug penetration, allowing latent foci to reactivate.[1] It is unclear whether the cerebellar location of the abscess, the size of the lesion, or the patient's immune status affected the response to treatment. Another explanation is the possibility of the host immune response eliciting a delayed hypersensitivity/inflammatory response to mycobacterial products resulting in an enlarging abscess.[1,8] It is also

possible that the abscess had become aseptic with permanence and even increment of pus and debris within the cavity. Both corticosteroids and thalidomide (tumor necrosis factor modulating drug) are being researched as possible adjuncts to treatment for enlarging abscesses.[3] It remains unclear why some tuberculosis abscesses enlarge, as such, close clinical and imaging follow up are strongly emphasized in these patients.

CONCLUSION

In conclusion, we present a rare case of tuberculosis abscess of the cerebellum in an adult, which enlarged after treatment with both antituberculosis drugs and surgical drainage and reached near resolution with no change in antitubercular therapy. This case highlights the importance of close clinical and imaging follow up, as one of the uncommon patterns of abscess evolution may include growth even on proper treatment. This pattern does not necessarily signify treatment failure, as our abscess resolved without change in treatment. Additional research is needed to determine factors that predispose to enlarging abscesses.

REFERENCES

1. Afghani B, Lieberman JM. Paradoxical enlargement or development of intracranial tuberculomas during therapy: Case report and review. Clin Infect Dis 1994;19:1092-9.
2. Chambers ST, Hendrickse WA, Record C, Rudge P, Smith H. Paradoxical expansion of tuberculomas during chemotherapy. Lancet; 1984;2:181-3.
3. Ersoy Y, Ates O, Onal C, But AD, Cayli SR, Bayindir Y, et al. Cerebellar abscess and syringomyelia due to isoniazid-resistant Mycobacterium tuberculosis. J Clin Neurosci 2007;14:86-9.
4. Gurjar HK, Joshua SP, Agrawal D, Mahapatra AK. Large pontine tubercular abscess treated surgically. Br J Neurosurg 2013;27:134-6.
5. Kumar R, Prakash M, Jha S. Paradoxical response to chemotherapy in neurotuberculosis. Pediatr Neurosurg 2006;42:214-22.
6. Menon S, Bharadwaj R, Chowdhary A, Kaundinya D, Palande D. Tuberculous brain abscesses: Case series and review of literature. J Neurosci Rural Pract 2011;2:153-7.
7. Roopesh Kumar VR, Gundamaneni SK, Biswas R, Madhugiri VS. Tuberculous cerebellar abscess in immunocompetent individuals. BMJ Case Rep 2012 (2011): 1535-7.
8. Schoeman JK, Fieggen G, Seller N, Mendelson M, Hartzenberg B. Intractable intracranial tuberculous infection responsive to thalidomide: Report of four cases. J Child Neurol 2006;21:301-8.