

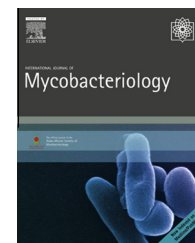


Asian - African society
Of Mycobacteriology

Available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/IJMYCO



Case Report

Tuberculous ventriculitis: A rare complication of central nervous system tuberculosis

Siavash Vaziri^a, Saeed Soleiman-Meigooni^{b,*}, Jalil Rajabi^c, Ali Asgari^c

^aDepartment of Infectious Diseases, Faculty of Medicine, Kermanshah University of Medical Sciences, Kermanshah, Iran

^bInfectious Diseases Research Center, AJA University of Medical Sciences, Tehran, Iran

^cDepartment of Infectious Diseases, Faculty of Medicine, AJA University of Medical Sciences, Tehran, Iran

ARTICLE INFO

Article history:

Received 24 December 2015

Received in revised form

19 February 2016

Accepted 3 March 2016

Available online 12 March 2016

Keywords:

Tuberculoma

Tuberculosis

Ventriculitis

ABSTRACT

Tuberculous ventriculitis is an inflammatory infection of the ventricular system of the brain, and is caused by *Mycobacterium tuberculosis*. We herein present the case of an immunocompromised patient with brain tuberculomas who developed ventriculitis during treatment. The patient was successfully treated with a high dose of steroid, long-term anti-tuberculosis drugs, and aggressive supportive care.

© 2016 Asian-African Society for Mycobacteriology. Production and hosting by Elsevier Ltd.

All rights reserved.

Introduction

In 2014, there were 9.6 million cases of tuberculosis (TB) worldwide, with reports estimating 1.5 million deaths globally due to this infection [1]. Central nervous system TB (CNS-TB) accounts for 1% of all TB cases, and has the highest mortality than other forms of the disease [2]. The pathogen *Mycobacterium tuberculosis* may invade the CNS following primary infection by inhalation, filtering by regional draining lymph nodes, low-level bacteremia, and metastatic seeding. Reactivation of the organisms in the CNS results in different syndromes, with meningitis being the most prevalent presentation of CNS-TB [3]. A proportion of patients with CNS-TB have also been reported to experience complications

during the various phases of the disease. Common complications of CNS-TB are cranial nerve paresis and hydrocephalus [4]. One of the rare and devastating complications of the disease is ventriculitis. Tuberculous ventriculitis occurs following enlargement, extension, and rupture of a tubercle in the ventricular system [5]. We herein describe the case of an immunocompromised patient with intracranial tuberculomas, who developed ventriculitis during treatment.

Case report

A 37-year-old man was admitted to our hospital for evaluation of photophobia, diplopia, and deterioration of mental status, which he was experiencing for the last 2 days. He

* Corresponding author at: Infectious Diseases Research Center, AJA University of Medical Sciences, E'temad Zadeh Avenue, Fatemi Street, Tehran 1411718541, Iran.

E-mail address: dr.saeed.meigooni@gmail.com (S. Soleiman-Meigooni).

Peer review under responsibility of Asian African Society for Mycobacteriology.

<http://dx.doi.org/10.1016/j.ijmyco.2016.02.008>

2212-5531/© 2016 Asian-African Society for Mycobacteriology. Production and hosting by Elsevier Ltd. All rights reserved.

was treated for Behcet's disease for the last 5 years and pulmonary TB for the last 3 weeks in another hospital. TB was documented by detection of *M. tuberculosis* in two separate sputum smear staining. On admission, he looked ill (blood pressure 135/88 mmHg, pulse 96 heart beats/min, respiratory rate 26 breaths/min, temperature 36.7 °C). Systemic examination was unremarkable, except for Cushing's face and crackles in both lungs. Neurological examination showed drowsiness, unclear speech, ataxia, and decreased deep tendon reflexes in four limbs. Laboratory finding was normal except for anemia (hemoglobin 10.3 mg/dL) and a high erythrocyte sedimentation rate (107 mm/h; normal range: up to 20 mm/h). Brain contrast-enhanced magnetic resonance imaging (MRI) showed meningeal enhancement and two ring-enhanced lesions in the right occipital lobe and the left insular lobe (Fig. 1). We investigated for other possible diagnoses such as pyogenic and fungal brain abscess, toxoplasmosis, and endocarditis by serologic and routine microbiological assays. In this situation, intravenous broad-spectrum antimicrobial treatment and steroid therapy were initiated. Owing to the documentation of pulmonary TB by sputum smear staining and endemicity of TB in our region, the patient received anti-TB drugs with a standard dose, which consisted of isoniazid (5 mg/kg), rifampin (10 mg/kg), pyrazinamide (25 mg/kg), and ethambutol (15 mg/kg), for cranial lesions.

The clinical condition of the patient improved for a few days. Further examinations for other possible diagnoses were all negative. On hospitalization day 26, he was developed a sudden loss of consciousness, high-grade fever, generalized tonic-clonic seizure, and respiratory distress. At that time, his vital signs were as follows: blood pressure 183/85 mmHg, pulse rate 84 heart beats/min, respiratory rate 28 breaths/min, and temperature 39 °C. Results of neurological examinations revealed a light comatose state, papilledema, nuchal rigidity, and lower limbs hypotonia. The second brain MRI showed mild hydrocephalus, enlargement of the posterior lesion with extension into the right occipital horn of the lateral ventricles, ependymal enhancement with some ventricular sludge, and periventricular edema, compatible with ventriculitis (Fig. 2). Hence, we started treatment with anti-convulsants, high dose of intravenous steroid, which was 24-mg daily divided dose of dexamethasone with any required supportive care. He also developed an episode of hospital-acquired infection (HAI) during this period, which was managed by broad-spectrum antimicrobials. Improvement occurred gradually over the ensuing weeks. The patient was discharged with relative amelioration on hospital day 44. The patient continued to receive anti-TB drugs and low-dose oral steroid for 1 year. In the 2nd-year follow-up, the patient was completely cured with no sign of reactivation.

Discussion

Ventriculitis, also called "ependymitis", is an inflammation of the ventricular system of the brain. It usually develops after neurosurgical interventions, and *Staphylococcus* species are the predominant microorganisms, identifiable in more than 90% of the cases. There are also rare reports of unusual causes

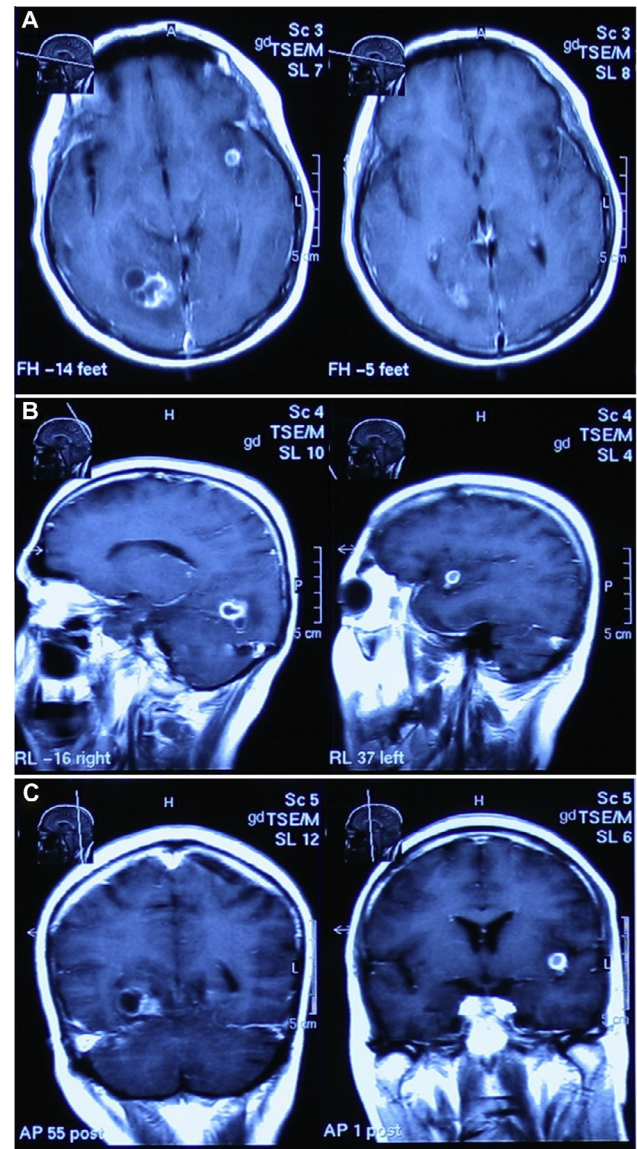


Fig. 1 – Contrast-enhanced T1-weighted brain magnetic resonance imaging shows meningeal enhancement, a ring-enhanced lesion in the right occipital lobe with cystic formation, and peripheral edema and another lesion in the left insular lobe in the (A) axial, (B) parasagittal, and (C) coronal views on admission.

of ventriculitis, such as those caused by *M. tuberculosis* [6]. Enlargement of brain tuberculomas during treatment is reported in about one third of patients [7] as a sign of paradoxical TB-associated immune reconstitution inflammatory syndrome [8], which has a 13% mortality rate [9]. In very exceptional cases, an enlarged tubercle may extend or rupture into the ventricular system, resulting in ventriculitis. Despite endemicity of TB in many countries, only limited reports of this complication have been found in the literature.

Ventriculitis is accompanied by vigorous neurological symptoms and devastating clinical condition. Symptoms of ventriculitis in our patient were seizure, papilledema, meningismus, and worsening of the underlying disease. These symptoms are comparable to the report presented by

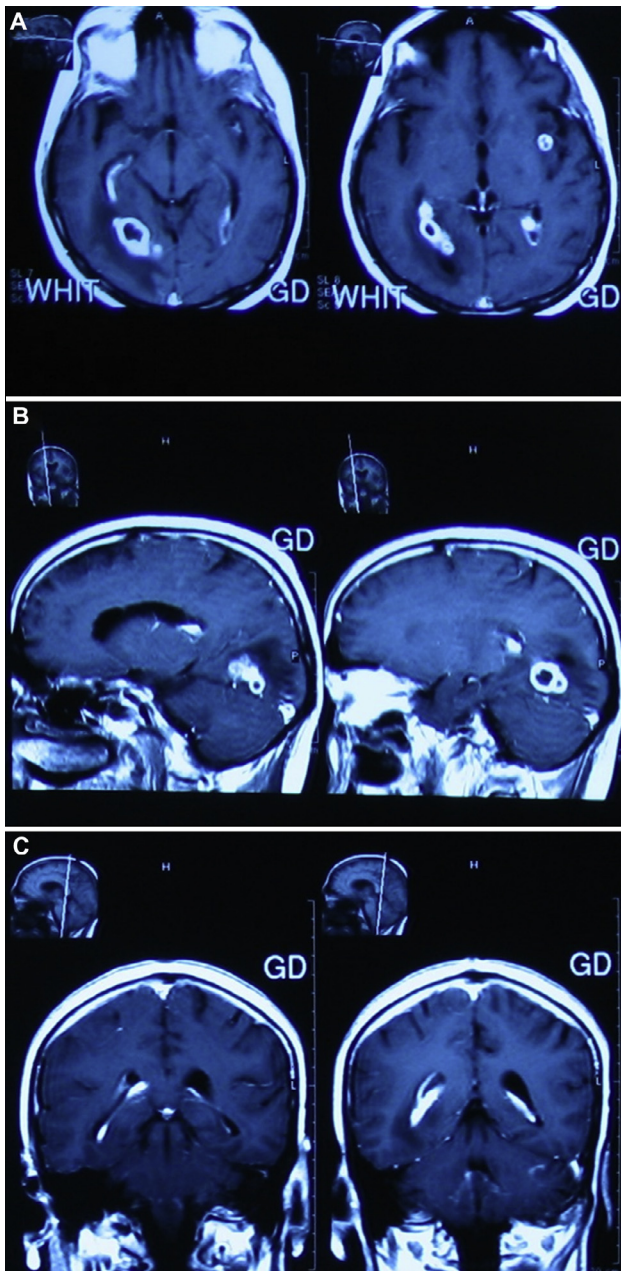


Fig. 2 – After 4 weeks of treatment, the second brain magnetic resonance imaging shows mild hydrocephalus, enlargement of the posterior lesion with extension into the right occipital horn of the lateral ventricles, resulting in ependymal enhancement with some ventricular sludge, which is compatible with ventriculitis in the (A) axial, (B) parasagittal, and (C) coronal views.

Paliwal et al. [10] who described five patients with tuberculous ventriculitis. Furthermore, another report of fatal tuberculous ventriculitis in an infant has also been presented [11]. A recent study [12] reported the case of an old patient with silent tuberculous ventriculitis presenting with symptoms of memory loss and drowsiness [12]. Common radiological findings in our patient and in all of these reports were

hydrocephalus, ventricular sludge, and ependymal and meningeal enhancement.

The main treatment regimen in our patient and in the patient described by Guler et al. [12] was medical management based on anti-TB drugs. However, in two other reports [10,11], five of six patients underwent operative intervention for severe complications, such as hydrocephalus. High-dose steroid is another principal component of the treatment in all the patients, but it raises the concern of developing a secondary infection. The final prognosis in our patient was recovery; however, among the seven previously reported cases, two patients died [10–12]. We think that the essential keys for the successful management of our patient were control of intracranial inflammation, management of HAI, long-term TB treatment, plenary supportive care, and avoidance of unnecessary operative intervention.

Conflicts of interest

All contributing authors declare no conflicts of interest.

Acknowledgments

We would like to thank our patient for contribution to follow-up and permission to publish his disease and treatment course. This study was supported by the Infectious Diseases Research Center, AJA University of Medical Sciences, Tehran, Iran.

REFERENCES

- [1] World Health Organization, Global Tuberculosis Report 2015, World Health Organization, Geneva, Switzerland, 2015. Available from: http://www.who.int/tb/publications/global_report/en/9789241565059_eng.pdf [Last accessed: February 29, 2016].
- [2] H.M. Peto, R.H. Pratt, T.A. Harrington, et al, Epidemiology of extrapulmonary tuberculosis in the United States, 1993–2006, *Clin. Infect. Dis.* 49 (2009) 1350–1357.
- [3] R.B. Rock, M. Olin, C.A. Baker, et al, Central nervous system tuberculosis: pathogenesis and clinical aspects, *Clin. Microbiol. Rev.* 21 (2008) 243–261.
- [4] Y. Amin, A. Shaukat, B.A. Mian, Intracranial manifestations of tuberculosis: an imaging study, *Biomedica* 20 (2004) 1–4.
- [5] S. Marais, G. Meintjes, D.J. Pepper, et al, Frequency, severity, and prediction of tuberculous meningitis immune reconstitution inflammatory syndrome, *Clin. Infect. Dis.* 56 (2013) 450–460.
- [6] A. Agrawal, R. Cincu, J. Timothy, Current concepts and approach to ventriculitis, *Infect. Dis. Clin. Pract.* 16 (2008) 100–104.
- [7] N. Farinha, K. Razali, H. Holzel, et al, Tuberculosis of the central nervous system in children: a 20-year survey, *J. Infect.* 41 (2000) 61–68.
- [8] N. Hejazi, W. Hassler, Multiple intracranial tuberculomas with atypical response to tuberculostatic chemotherapy: literature review and a case report, *Acta Neurochir. (Wien)* 139 (1997) 194–202.
- [9] D.J. Pepper, S. Marais, G. Maartens, et al, Neurologic manifestations of paradoxical tuberculosis-associated

- immune reconstitution inflammatory syndrome: a case series, *Clin. Infect. Dis.* 48 (2009) e96–e107.
- [10] P. Singh, V.K. Paliwal, Z. Neyaz, et al, Clinical and magnetic resonance imaging characteristics of tubercular ventriculitis: an under-recognized complication of tubercular meningitis, *J. Neurol. Sci.* 342 (2014) 137–140.
- [11] S. Kumar, R. Kumar, B. Radotra, et al, Tubercular ventriculitis: an uncommon entity, *Indian J. Pediatr.* 81 (2014) 608–610.
- [12] S.K. Guler, N. Güneş, B.G. Cokal, et al, Tuberculous ventriculitis as a relapse following central nervous system tuberculoma, *Neurol. Sci.* 36 (2015) 167–169.