Hong Kong J Radiol. 2015;18:46-50 | DOI: 10.12809/hkjr1414270

CASE REPORT

Evolution of Caseating Granuloma from Tuberculous Cerebritis in the Corpus Callosum

SH Kim, SS Choi, MJ Kang

Department of Radiology, Dong-A University Medical Center, Busan, Korea

ABSTRACT

Central nervous system tuberculosis usually presents as tuberculous meningitis, intracranial tuberculoma, tuberculous brain abscess, or tuberculous mass. Tuberculoma originates as a conglomerate of microgranulomata in an area of tuberculous cerebritis that joins to form a non-caseating tuberculoma. We report a patient with central nervous system tuberculosis that showed evolution of caseating granuloma from cerebritis in the corpus callosum.

Key Words: *Magnetic resonance imaging; Meningitis, listeria; Tuberculoma, intracranial; Tuberculosis*

中文摘要

胼胝體的結核性腦炎演變成乾酪樣肉芽腫

金尚泫、崔舜燮、姜明辰

中樞神經系統結核往往表現為結核性腦膜炎、顱內結核球、結核性腦膿腫或結核性腫塊。結核性腦炎病變區域的微小肉芽腫融合為非乾酪性結核球，進而形成乾酪性結核球。本文報告一名中樞神經系統結核病患者，其胼胝體的結核性腦炎演變成乾酪樣肉芽腫。

INTRODUCTION

Tuberculosis continues to be an important public health problem, primarily in developing countries. Central nervous system (CNS) tuberculosis usually present as tuberculous meningitis, intracranial tuberculoma, tuberculous brain abscess, or tuberculous mass.

We report a patient with CNS tuberculosis that showed evolution of caseating granuloma from cerebritis in the corpus callosum.

CASE REPORT

In July 2012, a 64-year-old man was admitted to the Dong-A University Medical Center, Busan, Korea, with disorientation, confusion, ataxia, dysarthria, and aphagia. His family reported that he had received empirical treatment for fever, chills, myalgia, and headache for the previous 7 days. He had a history of hypertension. On physical examination, he had blood pressure of 140/80 mm Hg and body temperature of 36.8°C. Blood tests showed a white blood cell count

Correspondence: *Dr Sunseob Choi, Department of Radiology, Dong-A University Medical Center, 1,3-ga, Dongdaeshin-dong, Seo-gu, Busan 602-715, Korea.*
Email: sschoi4048@gmail.com

Submitted: 16 Sep 2014; Accepted: 15 Oct 2014.

(WBC) of 11.4 x 10^9/l (reference range [RR], 4.5-11.0 x 10^9/l) and C-reactive protein of 50.47 nmol/l (RR, 0-4.76 nmol/l). Cerebrospinal fluid (CSF) study on admission showed an infection (WBC 8.9 x 10^9/l with 91% lymphocytes), protein 4.21 g/l (RR, 0.15-0.45 g/l), adenosine deaminase (ADA) 12.0 U/l (RR, 6.8-18.2 U/l), CSF glucose 2.22 mmol/l (RR, 2.8-4.4 mmol/l), and serum glucose 7.216 mmol/l (RR, 3.9-6.1 mmol/l). Acid-fast bacilli smear was negative and also CSF-negative *Mycobacterium tuberculosis* by polymerase chain reaction (PCR). CSF for *M. tuberculosis* culture, fungal culture, and viral titre were negative.

Magnetic resonance imaging (MRI) of the brain on admission showed increased signal intensity along the corpus callosum on fluid-attenuated inversion recovery images and diffusion-weighted image (DWI). The apparent diffusion coefficient map showed hypointense signal intensity. Postcontrast T1-weighted images showed no contrast enhancement around the hypointense area. Relative cerebral blood volume map demonstrated no evidence of increased vascularity (Figure 1). MR angiography showed normal arterial vasculature.

Taking into account the CSF study and MRI findings, the diagnosis was most likely to be tuberculous meningoencephalitis. The patient was given an antituberculosis drug regimen consisting of isoniazide 400 mg/day, rifampin 600 mg/day, ethambutol 800 mg/day, and pyrazinamide 1500 mg/day with steroids.

On day 5, the patient became lucid and his CSF study showed decreases in WBC and protein level: WBC 2.4 x 10^9/l with 87% lymphocytes, protein 1.33 g/l, ADA 14.0 U/l, and CSF glucose 2.998 mmol/l, and serum glucose 6.661 mmol/l.

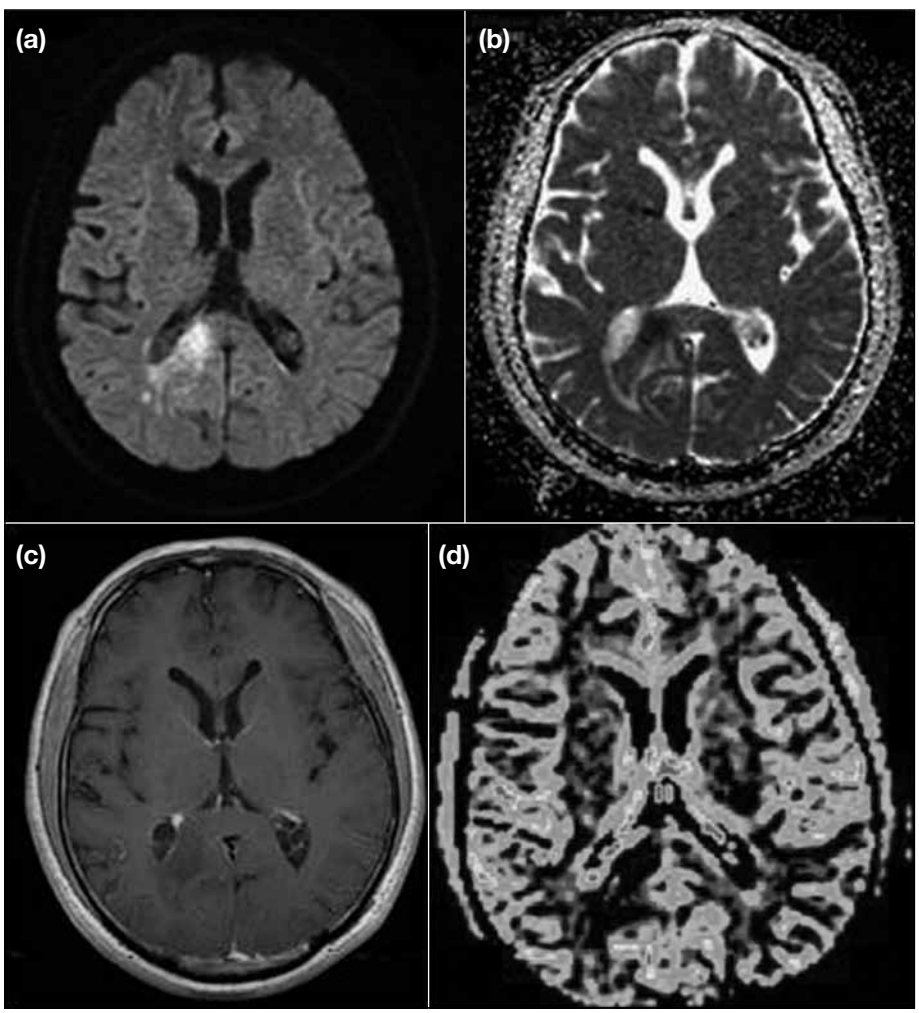

Figure 1. Magnetic resonance images on admission showing cerebritis. (a) A diffusion-weighted image shows hyperintense signal in the splenium of the corpus callosum, (b) apparent diffusion-coefficient map shows corresponding hypointense signal intensity, (c) postcontrast T1-weighted image shows no contrast enhancement around a hypointense area, and (d) relative cerebral blood volume map shows no evidence of increased vascularity in the splenium of the corpus callosum, which is suggestive of a non-neoplastic lesion.

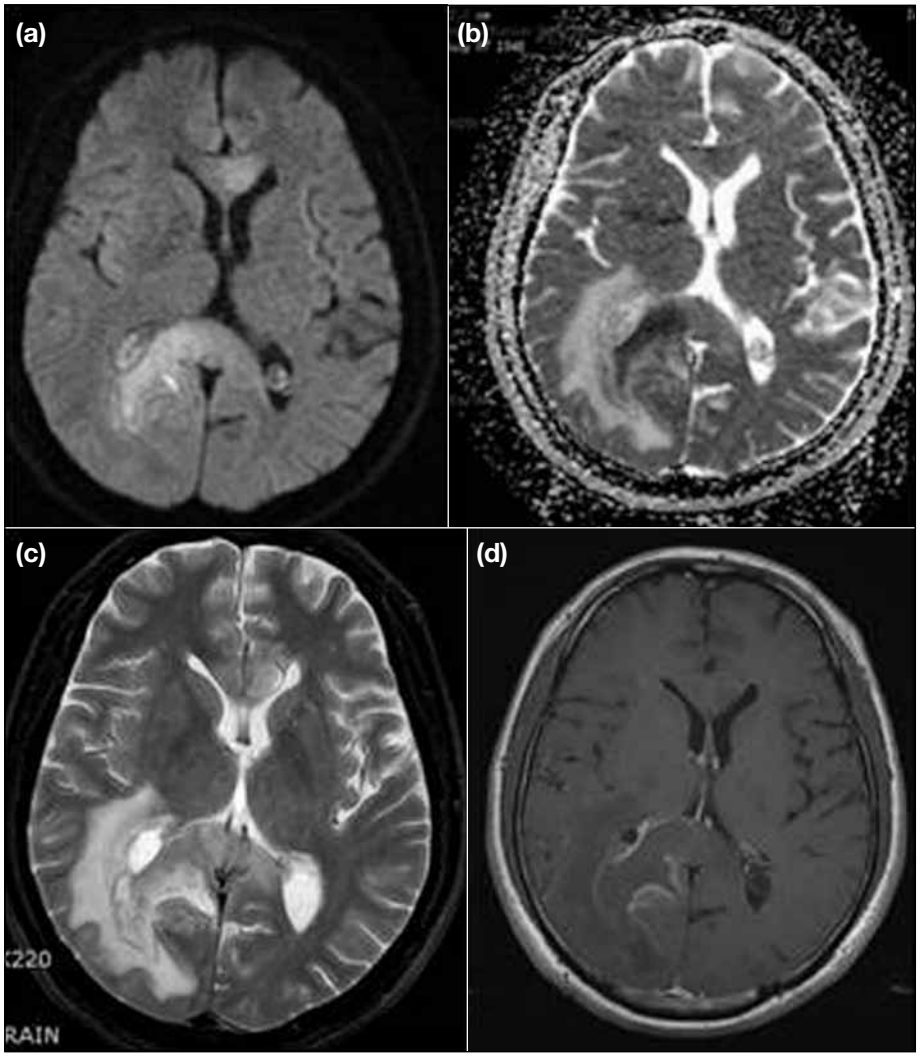

Figure 2. Magnetic resonance images on day 20 showing a tuberculoma. (a) A diffusion-weighted image shows increased extent of hyperintense lesion in the genu and splenium of the corpus callosum, (b) hypointensity on the apparent diffusion-coefficient map is consistent with restricted diffusion, (c) T2-weighted image shows central intermediate signal with perilesional oedema, and (d) postcontrast T1-weighted image shows peripheral enhancing rim around the lesion.

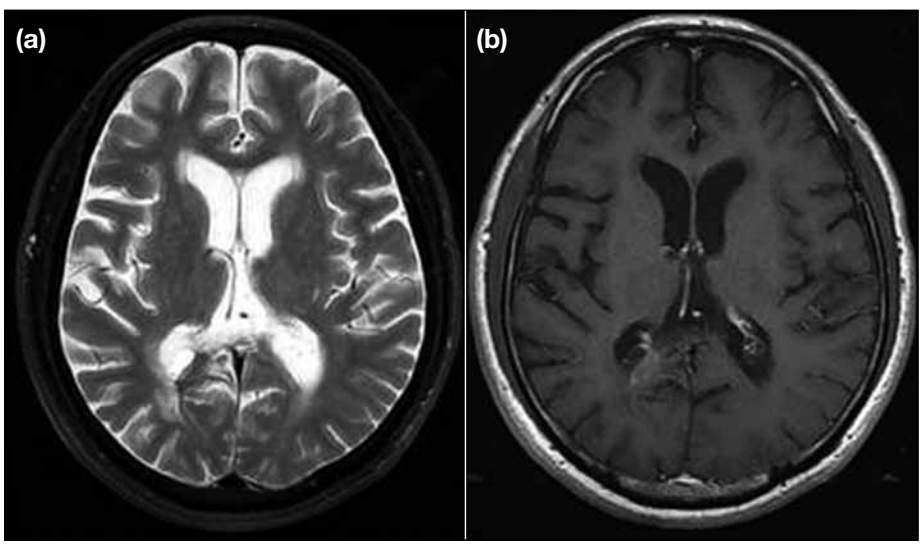

Figure 3. Magnetic resonance images after 2 years of antituberculous treatment. (a) T2-weighted and (b) postcontrast T1-weighted images show marked reduction in size and signal intensity, and near-complete resolution of the tuberculoma in the genu and splenium of the corpus callosum.

On day 20, MRI demonstrated increased extent of the lesion in the corpus callosum with perilesional oedema. Contrast-enhanced MRI showed peripheral enhancing rim around the lesion in the corpus callosum with perilesional oedema, consistent with caseating granuloma (Figure 2). CSF study showed improvement: WBC 9 x 10^9/l, protein 0.82 g/l, ADA 6.0 U/l, CSF glucose 3.719 mmol/l, and serum glucose 7.327 mmol/l. The increased extent of the lesion in the corpus callosum was thought to be a paradoxical reaction to antituberculous treatment because the patient did not show clinical or laboratory deterioration such as fever and leukocytosis, and CSF study showed improvement. He was maintained on antituberculous agents and the clinical symptoms improved. He was discharged on day 50.

The patient received 2 years of antituberculous treatment. At the 24-month follow-up, MRI showed marked reduction in the size and signal intensity and near resolution of the lesion in the corpus callosum (Figure 3). He was well except for intermittent bilateral lower-extremity allodynia.

Although the results of the microbiological investigations were negative, both protein and ADA levels in the CSF decreased and the glucose level was corrected. The clinical and radiological success of the antitubercular agents strongly suggested infection with *M. tuberculosis*.

DISCUSSION

M. tuberculosis involving the CNS is one of the most serious forms of tuberculous infection and usually presents as meningitis, tuberculoma, or abscess. In Korea, extrapulmonary tuberculosis accounts for 21% of all cases of *M. tuberculosis* and 0.8% of extrapulmonary *M. tuberculosis* is CNS tuberculosis.[1]

Cerebritis is the earliest manifestation of a cerebral infection, but it is unusual for patients to present at this stage. On T1-weighted MRI, an ill-defined isointensity or hypointensity and subtle mass effect may be seen, and contrast enhancement is absent or minimal.[2] Restricted water diffusion on DWI may be seen in cerebritis.[3-5] In the absence of purulent fluid, restricted water diffusion in early cerebritis might be attributed to hypercellularity. In response to the infecting microbe, an ill-defined area of coagulative necrosis forms, with profuse infiltration of the necrotic centre by polymorphonuclear leukocytes.[6,7] Vascular proliferation does not occur until several days later.[6] The initial MRI findings in this patient are in agreement with the described findings. We think that this case is important because it shows that early cerebritis should be added to the differential diagnosis of ill-defined lesions that may be associated with restricted water diffusion. The differential diagnosis includes ischaemia, and demyelinating, neoplastic, infectious, or inflammatory diseases. In this patient, MR angiography showed normal arterial vasculature. Postcontrast T1-weighted MRI showed no contrast enhancement and relative cerebral blood volume map demonstrated no evidence of increased vascularity, which is suggestive of a non-neoplastic lesion.

Tuberculoma originates as a conglomerate of microgranulomata in an area of tuberculous cerebritis that joins to form a non-caseating tuberculoma. Different types of tuberculous lesions reflect a continuum of the same disease. In most cases, subsequent central caseous necrosis develops that is initially solid but, in some instances, may eventually liquefy.[8] The radiological presentation depends on whether the granuloma is non-caseating, caseating with a solid centre, or caseating with a liquid centre.[9]

Caseating granuloma with a solid centre appears as low or intermediate signal intensity on T1-weighted images and intermediate or low signal intensity on T2-weighted images. T1-weighted gadolinium demonstrates rim enhancement of the lesion compared with caseating granuloma with a liquid centre, or tuberculous abscess, which shows a hyperintense signal on T2-weighted image. Furthermore, tuberculomas may show characteristics generally described for tuberculous brain abscess, including larger size (>3 cm in diameter), thin walls, presence of a single lesion, and multiloculation.[10] The differential diagnosis for tuberculoma includes mainly other granulomatous diseases (sarcoidosis, fungal lesions, and parasitic disease), primary neoplasms, and metastatic neoplasm. Imaging characteristics of tuberculoma may be non-specific. If this is the case, suspicion of a diagnosis of tuberculosis can be confirmed by the history and clinical findings, use of serological investigations and, especially, follow-up with anti-tuberculous therapy.[11,12]

CONCLUSION

MRI findings demonstrated the evolution of caseating granuloma with a solid centre from tuberculous cerebritis in the corpus callosum. Tuberculous cerebritis

should be considered in the clinical setting when an ill-defined focal brain lesion in the corpus callosum is associated with restricted diffusion.

DECLARATION

No conflicts of interests were declared by authors.

REFERENCES

1. Korean Center for Disease Control and Prevention. Annual report on the notified tuberculosis patients in Korea, 2010. Seoul: Korean Center for Disease Control and Prevention; 2011.
2. Falcone S, Post M. Encephalitis, cerebritis, and brain abscess: pathophysiology and imaging findings. Neuroimaging Clin N Am. 2000;10:333-53.
3. Höllinger P, Zürcher R, Schroth G, Mattle HP. Diffusion magnetic resonance imaging findings in cerebritis and brain abscesses in a patient with septic encephalopathy. J Neurol. 2000;247:232-4. cross ref
4. Tung GA, Rogg JM. Diffusion-weighted imaging of cerebritis. AJNR Am J Neuroradiol. 2003;24:1110-3.
5. Hirotani M, Yabe I, Hamada S, Tsuji S, Kikuchi S, Sasaki H. Abnormal brain MRI signals in the splenium of the corpus callosum, basal ganglia and internal capsule in a suspected case with tuberculous meningitis. Intern Med. 2007;46:505-9. cross ref
6. Flaris NA, Hickey WF. Development and characterization of an experimental model of brain abscess in the rat. Am J Pathol. 1992;141:1299-307.
7. Wispelwey B, Dacey RJ, Scheld W. Brain abscess. In: Scheld W, Whitley R, Durack D, editors. Infections of the central nervous system. 2nd ed. Philadelphia: Lippincott-Raven; 1997. p 463-93.
8. Gupta RK. Tuberculosis and other non-tuberculous bacterial graunulomatous infections. In: Gupta RK, Lufkin RB, editors. MR imaging and spectroscopy of central nervous system infection. New York: Kluwer Academic/Plenum Publishers; 2001. p 95-145. cross ref
9. McGuinness. Intracranial tuberculosis. In: Clinical imaging in nonpulmonary tuberculosis. Berlin, Heidelberg, New York: Springer; 2000. p 5-25.
10. Vidal JE, Hernández AV, Oliveira AC, de Souza AL, Madalosso G, Silva PR, et al. Cerebral tuberculomas in AIDS patients: a forgotten diagnosis? Arq Neuropsiquiatr. 2004;62:793-6. cross ref
11. Jinkins JR, Gupta R, Chang KH, Rodriguez-Carbajal J. MR imaging of central nervous system tuberculosis. Radiol Clin North Am. 1995;33:771-86.
12. Just M, Higer HP, Kramer G, Golla G, Betting O, Holper H, et al. Magnetic resonance imaging in infections of the brain: findings in tuberculosis, listeriosis, toxoplasmosis, subacute sclerosing panencephalitis, and multiple sclerosis. Neurosurg Rev. 1987;10:185-90. cross ref