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Restricted Diffusion Abnormalities on Magnetic Resonance Imaging in a Patient with Tuberculous Pachymeningitis

Kyusik Kang[a]
Ra Gyoung Yoon[b]
Byung-Kun Kim[a]

[a]Departments of Neurology and
[b]Radiology, Nowon Eulji Medical Center,
Eulji University, Seoul, Korea

Dear Editor,

A 69-year-old male with a history of hypertension, diabetes, and cerebellar infarct was referred to our hospital for headache. Seven days prior to admission he became anorexic and developed occipital headache and daytime sleepiness. He visited his nearest hospital and underwent brain diffusion-weighted imaging (DWI) (Fig. 1A and B) and T2-weighted magnetic resonance imaging (MRI) (Fig. 1C), which revealed multiple hyperintensities in the right temporal convexity, left temporal pole, interhemispheric fissure, and right dorsolateral prefrontal cortex. These lesions appeared hypointense on maps of the apparent diffusion coefficient (Fig. 1B).

Upon admission, his body temperature was 36.7°C and the findings of a neurological examination were normal. He underwent brain MRI, which revealed the right dorsolateral prefrontal lesion with contrast enhancement and bilateral temporal and falcine pachymeningeal enhancement (Fig. 1D and E). T2-weighted MRI revealed a curvilinear hypointensity along the right temporal convexity (Supplementary Fig. 1 in the online-only Data Supplement). A brain magnetic resonance angiogram was normal. He underwent a lumbar puncture, for which the opening pressure was 130 mmH_2O and the cerebrospinal fluid (CSF) was clear. Analysis of the CSF revealed a white blood cell count of 9×10^6/L, red blood cell count of 2×10^6/L, glucose level of 3.66 mmol/L, and protein level of 0.262 g/L. Laboratory analysis of the blood revealed a white blood cell count of 8.08×10^9/L, sodium level of 117 mEq/L, whole-blood glucose level of 10.6 mmol/L, C-reactive protein level of 2.7 mg/L, and procalcitonin level of <0.01 μg/L. A CSF acid-fast bacilli (AFB) smear was negative. The patient was placed on empirical antibiotics including antimycobacterials. The CSF was negative for Mycobacterium tuberculosis by polymeric chain reaction (PCR), but the sputum was positive. All other CSF workups including evaluations of fungal agents and bacteria produced negative findings. The patient was diagnosed as having tuberculous meningitis using the Lancet consensus scoring system (diagnostic score=13).[1] His symptoms disappeared and he was discharged on hospital day 11. Repeat DWI performed 18 days after discharge revealed resolution of the restricted diffusion abnormalities. Repeat brain MRI performed 4 months later demonstrated resolution of most of the radiological changes (Supplementary Fig. 2 in the online-only Data Supplement).

Our patient had several symptoms and signs that led to a diagnosis of tuberculous meningitis and tuberculoma after performing a comprehensive workup.[1-4] The Lancet consensus scoring system reliably distinguishes between tuberculous meningitis and other infectious etiologies, with a diagnostic score of ≥13 known to be virtually diagnostic of tuberculous meningitis because it has 100% specificity.[5] Despite recent advances, central nervous system (CNS) tuberculosis remains difficult to diagnose.[1] AFB smear and culture and PCR for *M. tuberculosis* deoxyribonucleic acid on CSF lack sensitivity.[1] Therefore, empirical treatment should be started in the presence of strong clinical suspicion even if the results of these tests

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Correspondence
Byung-Kun Kim, MD, PhD
Department of Neurology,
Nowon Eulji Medical Center,
Eulji University,
68 Hangeulbiseok-ro, Nowon-gu,
Seoul 01830, Korea
Tel +82-2-970-8311
Fax +82-2-974-7785
E-mail kimbkun1403@gmail.com

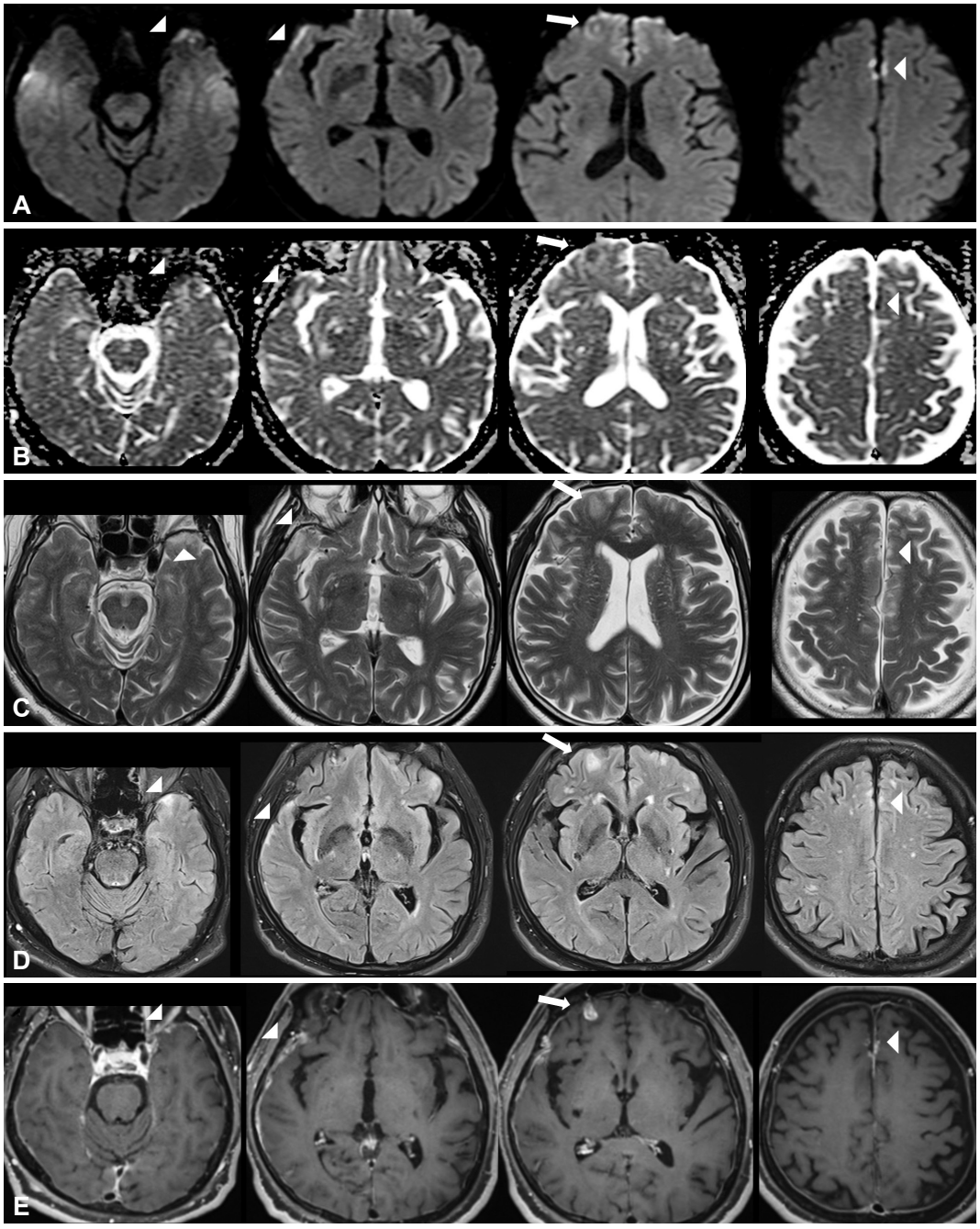

Fig. 1. Diffusion-weighted images (A) obtained 6 days after symptom onset reveal multiple hyperintensities in the left temporal pole, right temporal convexity, interhemispheric fissure (arrowheads from left to right), and right dorsolateral prefrontal cortex (arrow). These lesions appear hypointense on the corresponding maps of the apparent diffusion coefficient (B). T2-weighted images obtained simultaneously with the diffusion-weighted images (C) and fluid-attenuated inversion-recovery images (D) obtained 1 day later show hyperintensities in the corresponding regions and the adjacent cortices. Contrast-enhanced T1-weighted images (E) show the right frontal tuberculoma (arrow) and pachymeningeal thickening with enhancement along the left temporal pole, right temporal convexity, and falx cerebri (arrowheads from left to right).

are negative or pending.[6] The application of MRI has facilitated the diagnosis and treatment of CNS tuberculosis,[7] with DWI being particularly helpful.[7-9] An intra-axial DWI restriction can be tuberculoma, tuberculous abscess, or ischemic stroke caused by vasculitis,[7,9] whereas an extra-axial DWI restriction could indicate tuberculous subdural empyema.[8] The tuberculous pachymeningitis usually appears hypo- or isointense with the brain on T2-weighted MRI, and isointense on T1-weighted MRI being surrounded by edema.[10] It was particularly interesting in our case that the areas of restricted diffusion appeared in or near the thickened gadolinium-enhanced dura (Fig. 1A and E). Because the areas of restricted diffusion

were small and located near the cortices, it was difficult to confirm whether they were intra-axial or extra-axial.

We offer three possible explanations of the restricted diffusion in or near the dura. The first explanation is that the areas were intra-axial and represented ischemic stroke caused by infectious vasculitis.[11] However, the areas were not in the so-called medial tuberculosis zone[11] and did not corresponded to any arterial territory. The second explanation is that the areas were intra-axial and could have been tuberculous cerebritis. However, tuberculous cerebritis is very rare,[11] and how this appears in DWI is currently unclear. The third explanation is that the areas were extra-axial and caused by pachymeningitis. In accordance with this explanation, meningitis due to bacteria or fungi is known to cause subarachnoid DWI hyperintensities.[12]

The findings in the present patient indicate that tuberculous pachymeningitis should be considered in the differential diagnosis of DWI hyperintensities in or near the dura.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.3988/jcn.2021.17.1.147.

Author Contributions

Conceptualization: Kyusik Kang. Investigation: all authors. Writing—original draft: Kyusik Kang, Ra Gyoung Yoon. Writing—review & editing: Byung-Kun Kim.

ORCID iDs

Kyusik Kang https://orcid.org/0000-0002-4021-4439
Ra Gyoung Yoon https://orcid.org/0000-0003-3203-921X
Byung-Kun Kim https://orcid.org/0000-0003-1053-4682

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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