



Paradoxical Choroid Plexitis during Treatment for Tuberculous Meningoencephalitis

Hyo Eun Bae^a
Keun Tae Kim^a
Yong Won Cho^a
Kon Chu^b
Soon-Tae Lee^b

^aDepartment of Neurology,
Keimyung University School of Medicine,
Daegu, Korea

^bDepartment of Neurology,
Comprehensive Epilepsy Center,
Seoul National University Hospital,
Seoul, Korea

Dear Editor,

The clinical or radiologic worsening of already existing lesions or the emergence of new lesions after beginning treatment in patients with tuberculosis is referred to as a paradoxical reaction (PXR).¹ Cases of PXR in pulmonary tuberculosis and tuberculous meningitis were first reported in 1955 and 1980,^{2,3} respectively. Since PXR may be misdiagnosed as a drug-resistant state or treatment failure, and cause confusion in the diagnosis of tuberculosis. Herein we report a case of PXR that presented as choroid plexitis in order to raise awareness of this situation.

A 30-year-old woman presented with headache that first appeared 6 days previously. She had been taking steroids for Kikuchi's disease for about 2 months. The initial examination revealed that her body temperature was 37.9°C. She was confused, her orientation was impaired, and had stiffness in her neck but no focal neurologic deficit. Brain magnetic resonance imaging (MRI) showed diffuse leptomeningeal enhancement (Fig. 1A–C). In addition, milary tuberculosis was confirmed in chest computed tomography. The initial laboratory test revealed slight elevation of C-reactive protein, at 0.44 mg/dL (<0.5 mg/dL). Cerebrospinal fluid (CSF) analysis revealed pleocytosis at 95/high-power field (HPF), predominantly lymphocytes (70%), elevated protein (121.5 mg/dL), a normal adenosine deaminase level (4.6 U/L), and low glucose (34 mg/dL; 97 mg/dL in serum). Stain and culture findings for bacteria, mycobacteria, and fungi were negative, but polymerase chain reaction (PCR) of CSF showed positivity for *Mycobacterium tuberculosis*, confirming the presence of tuberculous meningoencephalitis. The patient improved after starting treatment with dexamethasone, rifampicin, ethambutol, isoniazid, pyrazinamide, and moxifloxacin. She was discharged after 2 weeks, when all of the symptoms had disappeared except for a mild headache.

Three weeks later, she made an unscheduled visit due to a severe headache. The patient appeared normal in a neurologic examination. She was afebrile and got a perfect score on the Mini Mental State Examination, however, brain MRI revealed left choroid plexitis (Fig. 1D–F). Follow-up CSF analysis revealed pleocytosis at 140/HPF, predominantly lymphocytes (70%), an aggravated protein level (218.5 mg/dL), an increased adenosine deaminase level (16.3 U/L), and low glucose (38 mg/dL; 94 mg/dL in serum). Dexamethasone administration was restarted, and her headache improved after 2 weeks. Two weeks after the headache improved, the initial susceptibility results for tuberculosis confirmed the presence of susceptibility for all of the tested drugs. Dexamethasone was used for a total of 4 weeks, and then gradually reduced before being discontinued. Brain MRI in the sixth month from the initiation of antituberculosis medication revealed a noticeable improvement of choroid plexitis (Fig. 1G–I). Based on these observations, the headache and neuroradiologic manifestations in this patient were compatible with PXR.

PXR is known to occur as a bimodal distribution, at 1 and 10 months after initiation of antituberculosis medication,⁴ and has been reported to occur in approximately 30% of patients

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Correspondence

Keun Tae Kim, MD
Department of Neurology,
Keimyung University
School of Medicine,
1095 Dalgubeol-daero, Dalseo-gu,
Daegu 42601, Korea
Tel +82-53-258-4379
Fax +82-53-258-4380
E-mail 6k5upa@gmail.com

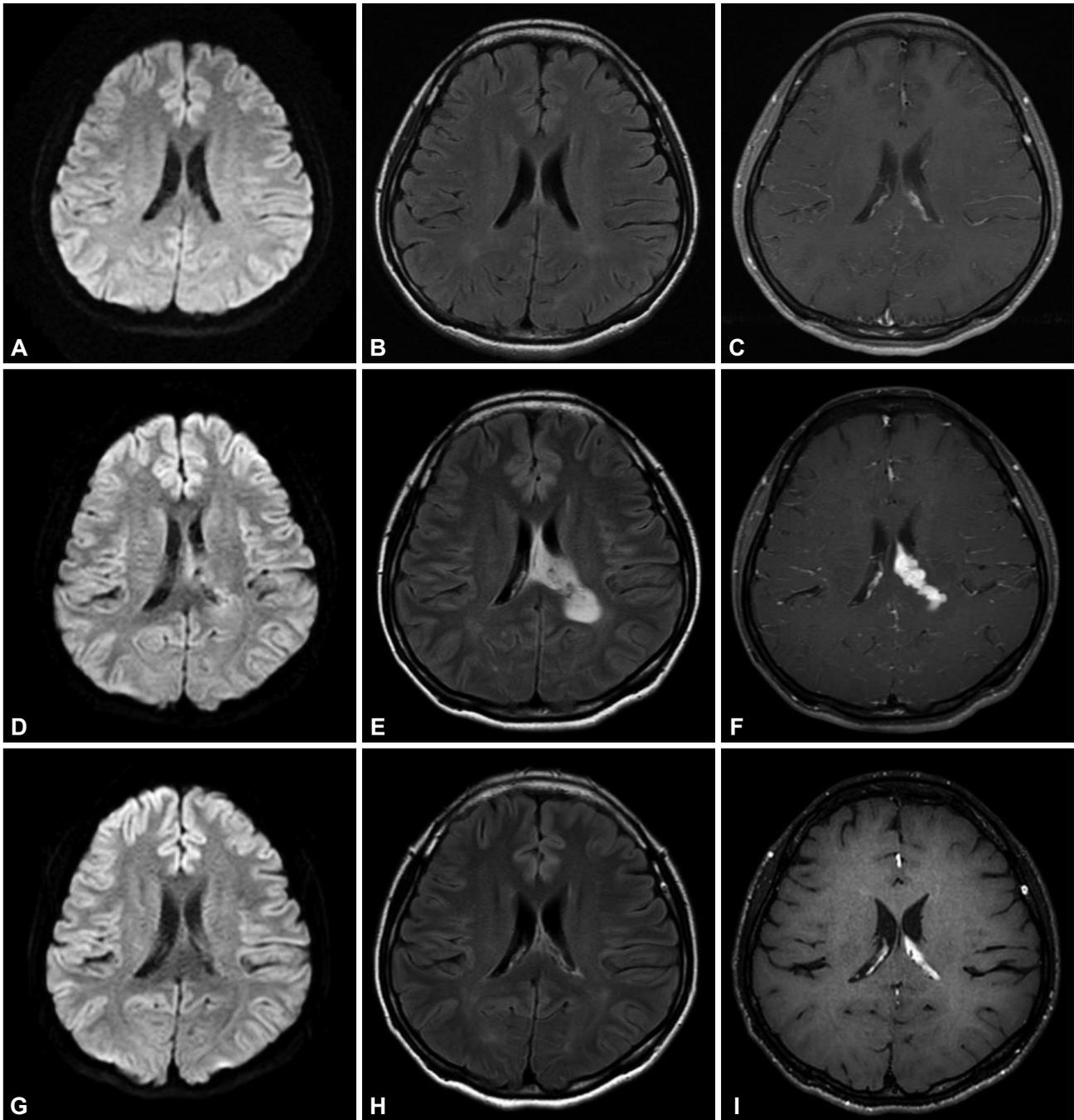


Fig. 1. MRI changes in paradoxical choroid plexitis. Serial diffusion-weighted imaging, FLAIR, and gadolinium-enhanced T1-weighted images from an axial view. A–C: Initial images showing diffuse leptomeningeal enhancement. D–F: Follow-up neuroimaging for severe headache performed 5 weeks after initiation of antituberculosis medication demonstrates left choroid plexitis. G–I: Follow-up images obtained in the sixth month, indicating marked improvement of the lesion and confirmation of a paradoxical reaction.

with tuberculous meningitis.⁵ The causative organism in our case was tuberculosis, with positivity confirmed in CSF PCR. If we did not consider the possibility of PXR, we might have considered the choroid plexitis with the deterioration of headache as a treatment failure and started unnecessary treatment options. Although there is a lack of evidence, it is known that steroids can be used for PXR. In the present patient, the clinical

symptom (headache) and the choroid plexitis in neuroimaging had both improved after administration of dexamethasone.

Primary choroid plexitis has been reported in CNS infections with cryptococcosis, tuberculosis, and nocardiosis,⁶ but it has never been reported as PXR. To the best of our knowledge, this is the first report of choroid plexitis as PXR. It is important to differentiate PXR from treatment failure due to resistant tu-

berculosis and irregular drug use, and so physicians need to be aware of PXR of tuberculosis and its protean manifestations.

Author Contributions

Conceptualization: Keun Tae Kim, Soon-Tae Lee. Data curation: Keun Tae Kim, Hyeon Bae. Writing—original draft: Hyeon Bae, Yong Won Cho, Keun Tae Kim. Writing—review & editing: Keun Tae Kim, Yong Won Cho, Kon Chu, Soon-Tae Lee.

ORCID iDs

Hyeon Bae	https://orcid.org/0000-0003-2351-0460
Keun Tae Kim	https://orcid.org/0000-0002-7124-0736
Yong Won Cho	https://orcid.org/0000-0002-6127-1045
Kon Chu	https://orcid.org/0000-0001-5863-0302
Soon-Tae Lee	https://orcid.org/0000-0003-4767-7564

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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