

## Cortical Involvement of Marchiafava-Bignami Disease: A Case Report<sup>1</sup>

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Marchiafava-Bignami disease is a rare complication of chronic alcoholism and this malady typically manifests as callosal lesion. I report here on one patient with Marchiafava-Bignami disease (MBD) who has symmetric restricted diffusion in both lateral-frontal cortices, in addition to the callosal lesion.

**Index words :** Alcoholism  
Brain, diffusion  
Corpus Callosum

Marchiafava-Bignami disease (MBD) develops in patients suffering with chronic alcoholism. Diffuse high signal intensity during the acute stage and focal low signal intensity during the chronic stage in the corpus callosum, on T2 weighted and fluid-attenuated inversion recovery sequences, are the hallmarks of MBD on magnetic resonance imaging (MRI) (1, 2). Cortical involvement of MBD accompanied with callosal lesion has recently been reported, but there have been only a few such cases. I report here on a patient with cortical involvement of MBD and this was accompanied with callosal lesion.

### Case Report

A 51-year-old man with a 30 year history of alcohol abuse was admitted to our hospital's emergency center because of his comatous mentality. Before he arrived at our center, he was admitted to another hospital due to

his one week period of altered mentality. He had no history of trauma or any hypoxic status. Before he was admitted to our emergency center, he had never suffered from electrolyte imbalance. His Glasgow coma scale score was three. The pupils were isocoric, and there was no spontaneous eye movement; the light reflex was sluggish. Routine blood tests, including serum electrolyte and cerebrospinal fluid studies, revealed no ab-



Fig. 1. Non-contrast CT scan shows low density in the splenium of the corpus callosum (arrow).

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normalities. On computed tomography (CT), low density was seen in the splenium of the corpus callosum (Fig. 1). The T2-weighted and diffusion weighted image (DWI) showed abnormal high signal intensities in the splenium of the corpus callosum and in both lateral-frontal cerebral cortices with apparently reduced diffusion coefficients (ADC) (Fig. 2). The patient was given intravenous high-dose vitamin B complex, including thiamin. Despite the treatment, the patient eventually expired.

### Discussion

Patients suffering with MBD show typical callosal le-

sion that consists of central necrosis and peripheral demyelination. CT scanning shows non-specific low density in the corpus callosum (3). MRI shows high signal intensity during the acute stage and focal low signal intensity during the chronic stage on T2-weighted images (1). This acute MRI finding has also been noted for tumors such as lipoma, glioblastoma multiforme, lymphoma and juvenile pilocytic astrocytoma, for demyelinating diseases such as multiple sclerosis and progressive multifocal leukoencephalopathy, for vascular diseases such as infarction and arteriovenous malformation, and for trauma such as diffuse axonal injury (4). The unusual cortical involvement of MBD has recently been reported. These cases showed bilateral high signal intensities

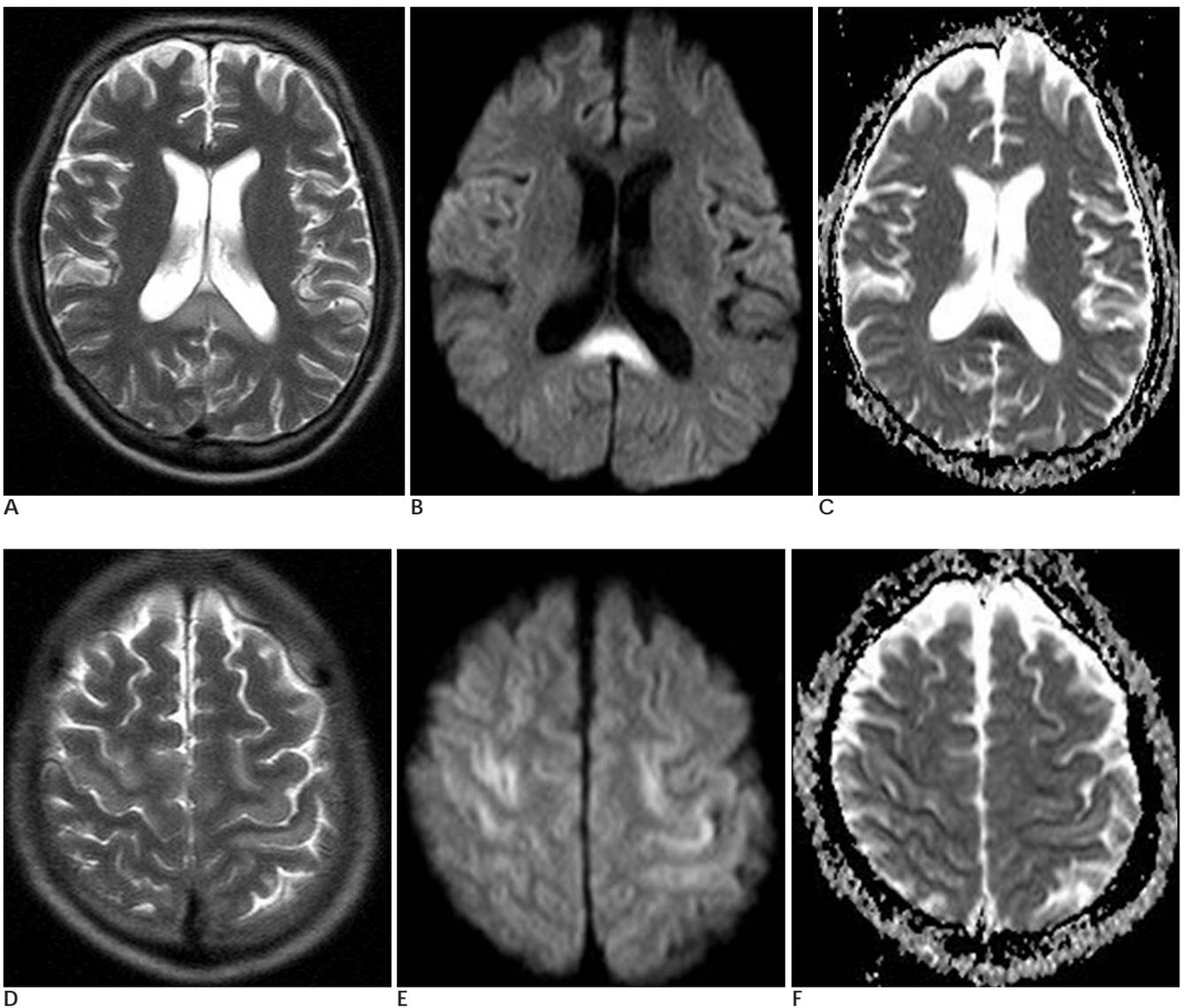


Fig. 2. High signal intensity is noted in the splenium of the corpus callosum on the T2-weighted image (A) and the DWI (B), and the splenium of the corpus callosum also shows decreased ADC values (C). (Ed note: put in exactly what shows decreased ADC values.) The T2-weighted image (D) and DWI (E) show high signal intensity in both lateral-frontal cortices with reduced ADC values (F).

on the DWI with reduced ADC values in the lateral-frontal cortices, and callosal lesion was also observed. Yet these cases do not show midline lesions such as the medial thalami, the mamillary bodies and the periaqueductal gray matter as are usually seen in Wernicke's encephalopathy (5, 6). The pathology of this cortical lesion, which is called Morel's laminar sclerosis, is laminar necrosis and gliosis, and this is mainly located in the third layer (7). Some authors have postulated that the early stage of Morel's laminar sclerosis might be caused by cytotoxic edema (5). Functional neuroimaging such as [18F]-2-fluoro-2-deoxy-D-glucose positron emission tomography (FDG PET) also shows cortical lesions due to the decreased glucose metabolism (8). This finding suggests that a disconnection of the commissural fibers as well as the extracallosal projections and association fibers that are involved in the cortico-cortical and cortico-subcortical connectivity, which causes transneuronal depression of the cortical metabolism and function (9). The prognosis is poor for the patients with cortical involvement (5, 6) and diffuse involvement of the corpus callosum (10).

In conclusion, MBD can show cortical lesion on MRI in the lateral-frontal lobe, in addition to that in the corpus callosum, and especially on DWI. These features can help the physician predict the patient's prognosis.

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