Mild Encephalopathy with Reversible Lesion in the Splenium of the Corpus Callosum and Bilateral Frontal White Matter

Jeong-Seon Cho, M.D., Sang-Won Ha, M.D., Young-Su Han, M.D., Sang-Eun Park, M.D., Ki-Moo Hong, M.D., Jeong-Ho Han, M.D., Eun-Kyoung Cho, M.D., Doo-Eung Kim, M.D., Jea-Geun Kim, M.D.

Departments of Neurology and Radiology, Seoul Veterans Hospital, Seoul, Korea

A 59-year-old man visited an emergency room due to the sudden onset of severe dysarthria with a drowsy mental status. MRI demonstrated T2 prolongation and restricted diffusion involving the splenium of the corpus callosum and bilateral frontal white matter neurological signs and symptoms were mild, and the recovery was complete within a week. Follow-up MRI performed one month later revealed complete resolution of the lesions. The clinical and radiological courses were consistent with previously reported reversible isolated splenial lesions in mild encephalitis/encephalopathy except for the presence of frontal lesions. This case suggests that such reversible lesions can occur outside the splenium.

J Clin Neurol 3(1):53-56, 2007

Key Words : Reversible Lesion, Splenium, Corpus Callosum, Encephalitis/Encephalopathy

Reversible MRI lesions with transiently restricted diffusion in the splenium of the corpus callosum (SCC) without any accompanying lesions have been reported in patients receiving antiepileptic drugs and experiencing trauma and high-altitude disease.1-5 This unusual MRI finding also has been reported in patients with clinically mild encephalitis/encephalopathy.1-4,6,7 Some authors have thus called this condition mild encephalitis/encephalopathy with a reversible isolated SCC lesion (MERS).2 We recently experienced a 59-year-old man with clinically mild encephalopathy, in whom brain MRI showed reversible lesions with transiently restricted diffusion in the SCC and bilateral frontal white matter. The clinical and radiological courses were identical to those of the reversible isolated SCC lesions previously reported in MERS.

CASE REPORT

A 59-year-old man visited an emergency room due to the sudden onset of severe dysarthria with a drowsy mental status. A neurologic examination revealed no other focal neurological deficits. Despite his drowsy mental state, he could fully understand questions, but answered them using letters. He had been receiving hypertensive medication for 7 years and antiplatelet medication for 3 years. He stated that he was not using any other drugs or alcohol, nor had toxin exposure or experience of weight loss. Three days before the
admission, he had a mild headache, general myalgia, and chilling sensation, but had taken no medication. At admission, his body temperature was 37.7°C; except for the slight elevated body temperature, his vital signs (blood pressure, respiration rate, and pulse rate) were within the normal ranges. On the admission day, brain diffusion-weighted imaging (DWI) showed high-intensity signals in the SCC and symmetric bilateral frontal white matter. A low apparent diffusion coefficient (ADC) was noted in the same areas. On the next day, T2-weighted MRI showed high-intensity signals, whereas T1-weighted MRI showed no signal changes in the same regions. Brain contrast-enhanced MRI revealed no enhanced lesions (Fig. 1). There were no inflammation signs or abnormalities in complete blood count, blood chemistry, urine analysis, cerebrospinal fluid, and a chest X-ray. No specific therapy was applied, and 2 days later he showed alert mentality and improved
dysarthria. Another 3 days later he had only slight
dysarthria, and his clinical symptoms were com-
pletely resolved within 7 days. Follow-up brain MRI
was performed 1 month later. No abnormal signal
changes were noted in diffusion-weighted, ADC, T1-
weighted, or T2-weighted MRI. Brain contrast-en-
hanced MRI showed no enhanced lesions (Fig. 2), and
lesions in the SCC associated with symmetric bilateral
frontal white-matter changes were completely resolved.

**DISCUSSION**

The SCC exhibits irregular water component and low
homogeneity. Myelin sheaths in the SCC exhibit a
relatively high water component, and the SCC is more
susceptible to cytotoxic edema than other brain areas.4,6

Encephalitis is defined as an acute onset of brain
dysfunction associated with inflammatory changes of
the CSF. When there is no evidence of inflammatory
changes, as in the case of our patient, the condition can
be diagnosed as encephalopathy.

In any encephalitis/encephalopathy patient with lesions
in the white matter, Acute disseminated encepha-
lomyelitis (ADEM) should be considered in the differential
diagnosis.1-3,6 However, in ADEM, recovery occurs within
weeks, and in our case the symptoms recovered within
7 days without specific treatment. MRI in ADEM
usually shows multiple foci of T1 and T2 prolongation
in the subcortical white matter that typically is bilateral
and asymmetric. The lesions usually evolve over weeks
to months and disappear only after several months. After
contrast-agent infusion, ADEM lesions will show
variable enhancement depending on their acuity. No
enhanced MRI lesions were detected in the present
case.

Reversible MRI lesions in the SCC have been re-
ported in patients with epilepsy receiving antiepileptic
drugs1,8 and in patients with MERS.1,6 However, our
patient was not receiving antiepileptic drugs.

All reported patients with MERS have had mild
clinical courses, recovering completely without any
sequelae. The previous reports of MERS excluded those
patients with parenchymal lesions in addition to the
reversible SCC lesion.1,3,6 More recently, symmetric
reversible lesions with transiently restricted diffusion
were seen in the peripheral frontoparietal white matter
and SCC of some patients with encephalitis/encepha-
lopathy.1,2 The clinical signs and symptoms of these
patients were mild, and their recovery complete. The
signal characteristics, reversibility of the lesions, and the
clinical features of the patients were identical to those
seen in MERS.2 We consider our patient to be similar
to those described as MERS.

Reversible brain lesions have been attributed to the
transient development of intramyelinic edema due to the
separation of myelin layers, which is a possible mecha-
nism for the transiently decreased diffusion of the SCC
lesion.2,3,6 However, a reversible SCC lesion with
reduced diffusion has been observed in a 12-day-old
neonate.9 At that age the SCC is still completely un-
myelinated, and hence mechanisms other than intra-
myelinic edema must be responsible for at least some of
the cases of reversible SCC lesion. Another possible
explanation is the development of an inflammatory
infiltrate. The influx of inflammatory cells and mole-
cules, possibly combined with related cytotoxic edema,
might have decreased the ADC. However, the symmetry
and absence of contrast enhancement in our patient
makes an inflammatory etiology less likely, since inflam-
mation is rarely completely symmetric and often causes
transient impairment of the blood–brain barrier. Thus the
reason for the callosal involvement in these cases
remains purely speculative.

The clinical and radiological courses of our patient
were identical to those of previously reported reversible
lesions isolated to the SCC in MERS. This suggests that
such reversible lesions are not necessarily restricted to
the SCC. Only a few Korean cases of reversible SCC
lesions in encephalitis/encephalopathy have been re-
ported,6,6 and here we have presented the first case
of reversible SCC lesions with bilateral white-matter
changes in encephalitis/encephalopathy in Korea.

**REFERENCES**

1. Takanashi J, Barkovich AJ, Shiihara T, Tada H, Kawatani
M, Tsukafara H, et al. Widening spectrum of a reversible splenial lesion with transiently reduced diffusion. *AJNR Am J Neuroradiol* 2006;27:836-838.

2. Takanashi J, Hirasawa K, Tada H. Reversible restricted diffusion of entire corpus callosum. *J Neurol Sci* 2006;247: 101-104.

3. Tada H, Takanashi AJ, Barkovich H, Oba H, Maeda M, Tsukahara H, et al. Clinically mild encephalitis/encephalopathy with a reversible splenial lesion. *Neurology* 2004;63: 1854-1858.

4. Seo HJ, Kim SY, Kim WM, Hong YJ, Sohn JH, Lee SM, et al. A case of encephalitis with a reversible splenial lesion on a diffusion weighted MRI image. *J Korean Neurol Assoc* 2006;24:507-510.

5. Uchino A, Takasa Y, Nomiyama K, Egashira R, Kado S. Acquired lesions of the corpus callosum: MR imaging. *Eur Radiol* 2006;16:905-914.

6. Hong JM, Park MS, Jun DC. Transient splenial lesion of the corpus callosum in patients with infectious disease. *J Korean Neurol Assoc* 2005;23:667-669.

7. Kobata R, Tsukahara H, Nakai A, Tanizawa A, Ishimori Y, Kawamura Y, et al. Transient MR signal changes in the splenium of the corpus callosum in rotavirus encephalopathy: value of diffusion-weighted imaging. *J Comput Assist Tomogr* 2002;26:825-828.

8. Shin YE, Cho YW, Kim HA, Sohn SI, Lee HL, Lim JG, et al. Two cases of transient focal lesion in the splenium of the corpus callosum after aggravated seizures. *J Korean Neurol Assoc* 2005;23:111-1139.

9. Takanashi J, Maeda M, Hayashi M. Neonate showing a reversible splenial lesion. *Arch Neurol* 2005;62:1481-1482.