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I.H. Lee, D.M. Kim and C.J. Song

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REPLY:

We thank Dr Das and colleagues for their interest in our article and their comments.

In our article,[1] we tried to evaluate the specific MR imaging findings in patients with clinically diagnosed uremic encephalopathy. Therefore, we enrolled 9 of 10 patients with chronic renal failure (CRF) and the lentiform fork sign (LFS) on brain MR imaging and 1 patient with acute renal failure (ARF) without the LFS. In 9 patients with CRF, despite regular hemodialysis (3 times a week in all patients), the various neurologic symptoms developed between regular hemodialysis sessions and the LFS was shown on brain MR imaging. Thus, they were included. One patient with ARF was first clinically diagnosed with uremic encephalopathy and then was included in the study, though there was no LFS on brain MR imaging. In our article, we suggested that the expansile T2 high signal intensity of the bilateral basal ganglia (LFS) may be a reliable finding of uremic encephalopathy.

Before brain MR imaging for a diagnosis and then additional intensive hemodialysis as a treatment for uremic encephalopathy, 6 of 10 patients underwent arterial blood gas analysis and only 2 of them showed metabolic acidosis (1 with CRF and 1 with ARF). As Dr Das and colleagues have suggested, there may be a relationship between the LFS and metabolic acidosis in patients with underlying CRF, but we did not find a specific one. In addition, regular hemodialysis may not always prevent various neurologic complications, including uremic encephalopathy from uremia.

Nine patients showed the LFS on T2WI or FLAIR. However, unfortunately, FLAIR was not available in 3/3 patients imaged with a 1.5T scanner and 2/7 patients imaged with a 3T scanner. In general, 9 patients with CRF and the LFS showed diffusely increased signal intensity ($n = 4$) and normal signal intensity ($n = 5$) in the basal ganglia on DWI, but 3 of 9 patients showed focal restricted diffusion in the globus pallidus or putamen—that is, various patterns of cytotoxic or vasogenic edema may be present in patients with uremic encephalopathy. Thus, the LFS on T2WI does not mean increased signal intensity on DWI in all patients.

Renal failure may be attributed to uremic encephalopathy and posterior reversible encephalopathy syndrome (PRES). In addition to the pathophysiologic findings such as vascular autoregulatory dysfunction,[2] imaging findings may overlap between PRES and uremic encephalopathy. Also, as PRES can be divided into 2 types, the cortical or subcortical type and the central variant type, uremic encephalopathy can be divided into 3 patterns according to the involved area: basal ganglia, cortical or subcoritcal area, and white matter. Considering the clinical condition and imaging findings, we classified the 2 patients showing cortical and basal ganglia involvement as having uremic encephalopathy. As Dr Das and colleagues have suggested, SWI is helpful for detecting microhemorrhage in some conditions such as PRES and is more sensitive than the gradient recalled-echo (GRE) sequence of 1.5T and 3T scanners. However, in our study only GRE sequence was available in 2/3 patients with a 1.5T scanner and 4/7 patients with a 3T scanner, and we did not find any microhemorrhages related to uremic encephalopathy.

Many various etiologies may play an important role in CRF, but diabetes mellitus–related CRF may be related to the LFS in the patients with uremic encephalopathy, especially Asian individuals, considering previous studies[3-7] as well as our study.

Once again, although we did not verify the sensitivity or specificity of the LFS due to the small number and selection bias in our study, it is important to consider the relationship between uremic encephalopathy and LFS in patients with CRF or ARF.

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I.H. Lee
D.M. Kim
C.J. Song
Department of Radiology
Chungnam National University Hospital
Chungnam National University School of Medicine
Daejeon, South Korea