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We read with great interest the description by U-King-Im et al¹ in the February issue of the *American Journal of Neuroradiology* (AJNR) of 4 patients with “acute hyperammonemic encephalopathy” on diffusion-weighted imaging (DWI). In the September 2010 issue of AJNR, we used a similar term of “acute hepatic encephalopathy” (most of the patients had hyperammonemia), and the terms could perhaps be considered interchangeable (notably, both would result in the acronym AHE).² We thank them for describing their findings, which are similar to the cases we described that, in our opinion, lie at the severe end of the spectrum of AHE. Of particular note is that 2 of their 4 patients died. In our study, 3 of 5 patients died; they had a similar distribution on DWI, which we termed “diffuse cortical involvement.”

U-King-Im et al¹ limited this description to the cingulate and insular gyri, but review of their available images demonstrates the abnormalities to be more extensive than those 2 regions. We do not point this out for the purpose of criticism; rather, we agree that these findings should alert the radiologist to the possibility of AHE. Thus, the combination of these 2 studies would indeed suggest (though preliminarily) that “diffuse cortical involvement” or alternatively “cingulate and insular involvement” may portend a poor outcome, though AHE is potentially reversible with therapy (such as lactulose).

They also similarly noted thalamic brain stem involvement in 1 patient, which we noted in most of our 20 patients. Hence, it would be of interest to us whether subtle involvement of the thalamic or dorsal brain stem existed on further review of fluid-attenuated inversion recovery (FLAIR) or DWI in these 4 patients or if they may have other patients who may have been eventually excluded from their study or not included due to stringent criteria, for example due to confounding diagnoses with preliminarily negative findings on MR imaging (we have noted that such situations uncommonly occur). Such confirmation of milder cases limited to the thalamic, for example, would help solidify our findings that AHE occurs along a spectrum, with multifocal diffuse cortical findings being at the severe end.

We proposed the terminology “acute hepatic encephalopathy” rather than “acute hyperammonemic encephalopathy” for several reasons: 1) Although some correlation likely exists, the degree of correlation between serum ammonia levels and AHE severity (the “ammonia hypothesis”) is still controversial. 2) Ammonia levels may be mildly elevated in patients with chronic cirrhosis without symptoms of AHE. 3) Severely encephalopathic patients may have normal ammonia levels. 4) Serum ammonia has been shown to be a poor pre-

dictor as a single test for the presence of AHE.^{3,4} More recent evidence suggests rather that there is a synergistic effect between ammonia and various other inflammatory cytokines that results in excess glutamine within astrocytes, leading to osmotic swelling of the astrocytes and the subsequent brain edema as well as other neurocytotoxic effects.⁵

Ultimately, the diagnosis of AHE still remains a clinical one, where serum ammonia and other ancillary tests are supplementary. Indeed, our study did not find a significant correlation between plasma ammonia levels and the initial clinical severity of AHE, though we found that the initial clinical severity, the plasma ammonia level, and the extent of FLAIR and DWI abnormalities on MR imaging all significantly correlated with patient outcome.² Thus, while ammonia is inextricably linked to the pathogenesis of AHE, it may be a bit misleading to use the term “hyperammonemic encephalopathy,” because ammonia is but 1 (albeit quite important) precursor to the development of encephalopathy. Both our study and that of U-King-Im et al¹ would suggest that prospective monitoring of serum ammonia levels and MR imaging findings, along with other clinical and laboratory tests, could further delineate the pathogenesis of this potentially reversible disorder.

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