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## Reply:

D.G. Na and E. Kim

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### Reply:

We thank Drs. Zuccoli, Pipitone, and Cruz for their interest in our recent article regarding MR imaging features of metronidazole-induced encephalopathy (MIE).<sup>1</sup> Although the exact mechanisms of MIE and Wernicke encephalopathy (WE) are not elucidated, we agree with the opinion that MIE may be mediated by pathogenic pathways similar to those thought to be operating in WE.

In our article, we reported that the brain lesions of MIE were typically located at the cerebellar dentate nucleus, midbrain, dorsal pons, medulla, and splenium of the corpus callosum. The recently published case reports of WE show lesions in the cerebellar dentate<sup>2,3</sup> and focal tegmental lesions of low pons,<sup>3,4</sup> which we described as characteristic lesion locations of MIE and controversial atypical MR features of WE in the article.<sup>1</sup> These recent reports<sup>2-4</sup> suggest that the MR imaging feature of bilateral symmetric involvement of dentate nuclei and cranial nerve nuclei may be found in WE, as well as in MIE. However, in these WE cases,<sup>2-4</sup> there was involvement of at least one of medial thalamus, mammillary body, or pericentral cortices, which were well-documented MR features in WE but were not reported in MIE. Therefore, although 2 diseases of MIE and WE may share the similar pathogenic pathways for metabolic pathology and similar MR

features in the cerebellum and brain stem, the analysis of lesion distribution on MR images may still be useful in distinguishing MIE from WE.

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