



## Response: Acute Diffusion MRI Findings in Metabolic Encephalopathies are Diverse

Se Jeong Jeon<sup>1</sup>, See Sung Choi<sup>1</sup>,  
Ha Yon Kim<sup>2</sup>, In Kyu Yu<sup>2</sup>

<sup>1</sup>Department of Radiology, Wonkwang University Hospital, Iksan, Korea; <sup>2</sup>Department of Radiology, Eulji University Hospital, Daejeon, Korea

We thank you for your in-depth comments and concerns about our article [1]. First, we regret that there are limited cases in the article among various acquired metabolic encephalopathies. We wanted to conduct the review based on the cases seen at our institutions, which are not uncommonly encountered in actual clinical practice. It was not possible to handle various types of metabolic encephalopathies, and our lack of experience with other encephalopathies, as you commented, also influenced the content of our review.

Missing in the review are DWI abnormalities of the oxidative metabolism disturbances; the oxidative metabolism in the mitochondria is a superordinate and subcellular concept to edema. In addition, DWI reflects the Brownian motion of water, which is usually used to assess edema (including cytotoxic, vasogenic, and excitotoxic). Our article did not review other metabolic MR techniques such as MR spectroscopy and pH imaging [2,3]. However, our review highlights an edema issue, which is considered as the mechanism of several metabolic encephalopathies.

Most mitochondrial diseases (MIDs), but not always,

are rare hereditary diseases related to a genetic defect (mitochondrial DNA or nuclear DNA). Relatively well-known mitochondrial stroke-like lesions (Fabry disease, mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes; MELAS) are hereditary diseases [4]. There are also acquired MIDs, but we have not had enough experience with them.

Hashimoto's encephalopathy was not considered in this review. As we mentioned above, this article was compiled based on the cases encountered at our institutions. We have not had any experience with Hashimoto's encephalopathy; therefore, we did not include it in the article. We would have reviewed it if we had experience with it.

Lastly, you asked whether the family history was truly negative in all the cases whose images were presented and whether genetic causes were truly excluded through genetic investigation. The response is that all cases whose images were presented did not have any familial history. Even though we did not exclude genetic causes by genetic investigation, the cases did not have any genetic disease on EMR, but had an apparent cause.

We sincerely appreciate your attention and comments once again and hope to soon see another article on the other types of metabolic encephalopathies that are not covered in our article.

### Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

### ORCID iDs

Se Jeong Jeon

<https://orcid.org/0000-0002-1134-2757>

See Sung Choi

<https://orcid.org/0000-0002-4442-9410>

Ha Yon Kim

<https://orcid.org/0000-0002-7139-8410>

In Kyu Yu

<https://orcid.org/0000-0003-1587-2840>

### Funding Statement

None

**Received:** November 5, 2021 **Accepted:** November 5, 2021

**Corresponding author:** In Kyu Yu, MD, Department of Radiology, Eulji University Hospital, 95 Dunsanseo-ro, Seo-gu, Daejeon 35233, Korea.

• E-mail: midosyu@eulji.ac.kr

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

**REFERENCES**

1. Jeon SJ, Choi SS, Kim HY, Yu IK. Acute acquired metabolic encephalopathy based on diffusion MRI. *Korean J Radiol* 2021;22:2034-2051
2. Straathof M, Meerwaldt AE, De Feyter HM, de Graaf RA, Dijkhuizen RM. Deuterium metabolic imaging of the healthy and diseased brain. *Neuroscience* 2021;474:94-99
3. Hattingen E, Magerkurth J, Pilatus U, Mozer A, Seifried C, Steinmetz H, et al. Phosphorus and proton magnetic resonance spectroscopy demonstrates mitochondrial dysfunction in early and advanced Parkinson's disease. *Brain* 2009;132:3285-3297
4. Niyazov DM, Kahler SG, Frye RE. Primary mitochondrial disease and secondary mitochondrial dysfunction: importance of distinction for diagnosis and treatment. *Mol Syndromol* 2016;7:122-137