



Received: 20-08-2025
Accepted: 30-09-2025

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Only Diagnose EBV-Related Myositis in a Patient with Mitochondrial Myopathy after Detecting the Virus in the Muscle

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DOI: <https://doi.org/10.62225/2583049X.2025.5.5.5027>

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Letter to the Editor

We read with interest the article by Kyriakidou *et al.* about a 33-year-old man with maternally inherited diabetes and deafness (MIDD), arterial hypertension, renal insufficiency (proteinuria), trigeminal neuralgia, brachial plexus paralysis, and hyperlipidemia, who was admitted with fever, fatigue, and generalized myalgia ^[1]. The tests revealed elevated creatine kinase (908 U/L), elevated transaminases, and elevated IgM antibodies against the Epstein-Barr virus (EBV), which led to a diagnosis of EBV-associated myositis ^[1]. The patient recovered within one month after the start of symptomatic treatment ^[1]. The study is noteworthy, but some points require discussion.

The first point is that the diagnosis of MIDD was not confirmed ^[1]. Neither the family history nor the mutation underlying MIDD were reported ^[1]. Since MIDD is attributable to the m.3243A>G variant in MT-TL1 in the majority of cases, it would have been essential to confirm that the index patient carried this mutation or to test the index patient and his first-degree relatives for it.

The second point is that we disagree with the diagnosis of MIDD ^[1]. MIDD is characterized by maternally inherited diabetes and deafness, but the patient clearly suffered from myopathy, trigeminal neuralgia, arterial hypertension, hypothyroidism, and hyperlipidemia in addition to diabetes and hearing impairment ^[1]. Therefore, the patient should be diagnosed with MIDD plus ^[2] rather than MIDD, assuming he actually carried the m.3243A>G variant.

The third point is that the diagnosis of “myositis” is not substantiated ^[1]. The index patient did not undergo a muscle biopsy, muscle magnetic resonance imaging (MRI) with contrast agent, or needle electromyography. As long as a muscle biopsy does not clearly detect EBV particles in the myofibers, the diagnosis of EBV-associated myositis remains speculative. Abortive rhabdomyolysis triggered by the viral infection was not considered in the index patient. Muscle pain is a classic symptom of rhabdomyolysis ^[3]. In particular, in a patient with mitochondrial myopathy who is taking statins, a viral infection can trigger rhabdomyolysis. There are even cases of rhabdomyolysis due to EBV ^[4].

The fourth point is that no even cases of rhabdomyolysis was provided on serum lactate levels ^[1]. Assuming that the patient did indeed carry the m.3243A>G variant and that this variant is phenotypically characterized by lactic acidosis, it can be speculated that the viral infection further increased serum lactate and was responsible for the fatigue and muscle pain. Lactic acidosis is known to manifest itself with myalgia ^[5].

The fifth point is that it is not clear why a patient with a mitochondrial disease was given statins ^[1]. Statins and fibrates are known to cause myopathies or rhabdomyolysis, which are more likely and more severe in patients with pre-existing myopathy. Therefore, these types of drugs should be administered with caution and creatine kinase levels should be monitored regularly. In summary, before diagnosing EBV-associated myositis, all other causes of elevated creatine kinase levels associated with myalgia should be thoroughly ruled out. The diagnosis of EBV-associated myositis requires detection of the virus in muscle tissue by muscle biopsy.

Declarations

Ethical approval: Not applicable.

Consent to participation: Not applicable.

Consent for publication: Not applicable.

Funding: None received.

Availability of data and material: All data are available from the corresponding author.

Completing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SM: contributed to literature search, discussion, correction, and final approval.

Acknowledgements: None.

Keywords: Maternally Inherited Diabetes and Deafness, Epstein Barr Virus, Myositis, Creatine-Kinase, Rhabdomyolysis

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