LETTER TO THE EDITOR

Reply to the Letter to Editor Regarding “An Unusual Case of Critical Illness Polyneuromyopathy”

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Abstract

Critical illness myopathy (CIM), critical illness polyneuropathy (CIP), and critical illness polyneuromyopathy (CIPNM) are the group of disorders that are commonly presented as neuromuscular weakness in intensive care unit (ICU) settings. They are responsible for prolonged ICU stay and failure to wean off from mechanical ventilation.¹ We report one such case of young female who was admitted with undiagnosed type I diabetes mellitus with diabetic ketoadsosis with severe hypokalemia with sepsis developed acute-onset quadriplegia and diaphragmatic palsy within 72 hours of ICU admission. Detailed investigation led to the diagnosis of critical illness polyneuromyopathy. In view of high morbidity, mortality, and poor prognosis, a guided approach to diagnoses and treatment in earliest possible duration might give better improvement and outcome of the illness. Despite all the odds, our patient showed good clinical improvement and finally got discharged.

Keywords: Critical illness myopathy, Critical illness polyneuromyopathy, Diabetic ketoacidosis (DKA).

Indian Journal of Critical Care Medicine (2020): 10.5005/jp-journals-10071-23499

Sir,

At first, we are at immense pleasure to thank you and your team for publishing our case report “An Unusual Case of Critical Illness Polyneuromyopathy” in your Journal IJCCM. We are glad to receive and answer to some of the concerns being raised by your readers.

We want to emphasize critical illness polyneuromyopathy (CIPNM), which is an important entity to suspect in critically ill patients with multisystem involvement and develops acute-onset flaccid paralysis. So, we have decided to publish this case report to discuss our views in approaching a case of ICU-acquired weakness and the differential diagnoses.

Reply to the major concerns raised:

• Our patient presented with altered sensorium and severe dehydration; upon investigation, she was diagnosed to have diabetic ketoacidosis with severe hypokalemia (pH 6.9, K⁺: 1.9 mEq/dL). She almost had required large amounts of potassium (300 mEq/day) for correction, and normal serum potassium levels were achieved in 3 days. Thereafter, it was constantly within normal limits, despite management of DKA. Her initial Magnesium and phosphorus levels were also within normal limits (Mg: 2.4 mg/dL, PO4<sup>−</sup>: 3.8 mg/dL). Normal levels: Mg—1.6–2.6 mg/dL, PO4<sup>−</sup>: 2.5–4.5 mg/dL. Despite the normal levels, we have supplemented magnesium along with calcium and vitamin D3.

• Since our patient had severe hypokalemia, we initially thought of hypokalemic periodic paralysis as our differentials in this case, but the patient continued to have quadriplegia for almost 2 weeks after presentation, despite early electrolyte correction.

• According to the literature, to cause acute flaccid paralysis/quadriplegia in a case of hypophosphatemia, serum phosphate levels should be <1 mg/dL.¹ In our patient, serum phosphate levels (PO4<sup>−</sup>: 3.8 mg/dL) are absolutely normal throughout her ICU stay. Hence, hypophosphatemia was ruled out.

• We have taken neurophysician’s opinion for quadriplegia and have done electromyographical studies that turned out to be motor, sensory axonal polyneuropathy, and myopathy.

We sent total CPK levels which was very high, 1,171 U/L. Other causes of acute-onset flaccid paralysis like GB syndrome and myasthenia gravis² were ruled out.

• We had gone through many literatures regarding CIPNM. As per the present guidelines, we decided to give intravenous immunoglobulins (IVIg),³ and then patient started showing improvement in power and tone. The patient was quadriplegic for many days. Only when she got IVIg, she was weaned off mechanical ventilation completely.

Thus, we diagnosed this patient to have had CIPNM after ruling out all other possible causes of acute-onset flaccid paralysis in ICU. If the patient had developed paralysis due to hypokalemia, patient would have had improved after correcting large potassium deficits. Only after patient got infused with IVIg, she showed drastic neurological improvement in power, tone, and was weaned off mechanical ventilation in the following days. Despite having bad prognosis in most of the cases of CIPNM, our patient survived with almost complete recovery.

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Source of support: Nil

Conflict of interest: None

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REFERENCES

