

Critical illness myopathy

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Abstract

Critical illness myopathy is one of the causes for failure to wean from ventilator. Although associated factors of dyselectrolytemia is to be ruled out and other causes for failure to wean is to be ruled out before diagnosing critical illness myopathy. Several factors play role in development of this condition. Here we present a case report of a post partum patient where we had encountered failure to wean despite several attempts and at last was successfully weaned and discharged from Intensive care unit.

Key words: critical illness, myopathy, weaning

Twenty year old primigravida who had undergone cesarean section for fetal distress had post partum hemorrhage. She was managed conservatively with blood and intravenous fluids. There was continued bleeding, so uterine compression and internal iliac artery ligation were done, after which bleeding was stopped. Intra operative blood loss was estimated around 2 liters which was replaced with blood.

Still Blood pressure was low and she was then started on injection Dopamine 6µ/kg/min. As the procedure was done under general anesthesia and blood pressure was not picking up, we planned for post operative mechanical ventilation.

Vasopressors were continued, but still the BP was low. Dopamine was increased and nor-adrenaline was started. Gynecological examination revealed no obvious abnormality and no active bleeding.

She was put on controlled mode ventilation as residual Neuro-Muscular Blockade was not reversed. Then on start of spontaneous ventilation she was kept in Synchronised Intermittent Mandatory Ventilation (SIMV) mode. BP was still maintained with inotropes. Patient was restless and was sedated. Despite sedation with adequate dose of Fentanyl, Midazolam, and Lorazepam, she was still fighting with the ventilator. As BP was still low and was on inotropes, we had planned for paralysis and controlled ventilation. On 3rd day on ventilator, paralysis was stopped and noradrenalin was titrated and stopped. She was managed with intravenous sedation, where she tolerated mechanical ventilation well. On 4th day, dopamine was also titrated

and stopped. She had good respiratory efforts so trial of Continuous Positive Airway Pressure was done which revealed good tidal volume. The next day, trial of T piece was given, she tolerated it well. She was extubated uneventfully. Three hours later, she started having tachypnea and respiratory distress. Other causes were ruled out; she was re intubated and started on mechanical ventilation, she had good tidal volume on SIMV mode and tachypnea subsided. To rule out dyselectrolytemia including Magnesium, it was sent and was normal. Arterial blood gas (ABG) revealed normal values. On the 5th day similar trial of T piece was done and patient extubated, she had similar event following extubation.

Other causes for failure to wean including nutritional cause were ruled out and patient was diagnosed to have critical illness myopathy. She was kept on mechanical ventilation for total of 10 days where she made spontaneous recovery and was then discharged from ICU.

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Table 1: Illustrating different causes of muscular weakness in ICU setting².

Medications	steroids, neuromuscular blockers (pancuronium, vecuronium), zidovudine, amiodarone
Spinal cord disease	ischemia, compression, trauma, vasculitis, demyelination
Illness	Critical illness myopathy, polyneuropathy
Loss of muscle mass	cachectic myopathy, rhabdomyolysis
Undiagnosed neuromuscular disorder	myasthenia, LEMS, inflammatory myopathies, mitochondrial myopathy, acid maltase deficiency
Electrolyte disorders	hypokalemia, hypophosphatemia, hypermagnesemia
Systemic illness	porphyria, AIDS, vasculitis, paraneoplastic, toxic

Discussion

‘Spontaneous weaknesses indicate disease’-Hippocrates, 460–377 B.C.

Neuromuscular weakness is often encountered in patients in the intensive care unit. The syndrome of severe, acute, intensive care unit (ICU)-acquired neuromuscular weakness poses a common and serious diagnostic, prognostic, and therefore management issue. It goes by various names, some of which presuppose a mechanism: acute necrotizing myopathy of intensive care, acute quadriplegic myopathy, critical care myopathy, critical illness myopathy (CIM), critical illness neuromuscular disease, critical illness neuromyopathy, critical illness polyneuromyopathy, critical illness polyneuropathy (CIP)¹.

Causes of neuromuscular weakness in ICU may be attributed to Medicines, Spinal cord diseases, Critical illness myopathy, loss of muscle mass, Electrolyte disorders, systemic illness.

Risk factors for critical illness myopathy

Sepsis, multi-organ dysfunction syndrome, multi-organ failure, female sex, use of corticosteroids, severe asthma, ionic abnormalities, malnutrition and immobility are frequently cited causes of Critical illness myopathy in humans³⁻⁴.

Pathogenesis

Critical illness myopathy is likely multifactorial and may be partly related to neuromuscular toxicity from drugs such as corticosteroids, aminophylline, and paralytic agents. Neuromuscular blocking agents have been implicated in prolonged weakness^{5, 6, 7, 8}, possibly by persistent disruption of synaptic transmission or a toxic myopathic effect.

Diagnosis

The clinical diagnosis of critical illness myopathy is challenging. Although muscle biopsy is indicated in the diagnosis, it's time consuming and invasiveness of the procedure makes it more complex. It usually becomes apparent when patient cannot be weaned from the ventilator. Flaccid weakness of the extremities and loss of tendon reflex are associated findings⁹.

Treatment

No specific treatment is known to hasten recovery from myopathies associated with critical illness. The benefit of physical rehabilitation is unknown, but may be minimal, at least during the early stages of recovery.

Recovery of muscle strength is expected in survivors of critical illness, independent of whether the acquired muscle weakness is caused by neuropathy, myopathy, or prolonged blockade of neuromuscular junctions. After muscle and/or nerve injury, functional recovery generally occurs within two weeks to several months.

Conclusion

CIM is not so uncommon disorder. With advent of better ventilator management and diagnosis of this disorder, CIM can be managed conservatively. The incidence of CIM can be decreased by decreasing the use of corticosteroids and non depolarizing muscle relaxant in ICU settings.

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