Case Report

Acute respiratory failure revealing Guillain-Barré syndrome

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ABSTRACT

This article reports the occurrence of Guillain-Barré syndrome in a critically ill patient manifesting as a "difficult-to-wean" patient. This uncommon but possible etiology is worth noting by physicians in the intensive care unit.

Keywords: Respiratory failure, Guillain-Barré syndrome, intensive care unit.

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Guillain-Barré syndrome (GBS) is an acute autoimmune polyradiculoneuropathy with prototype clinical presentation including generalized flacid paralysis, areflexia, variable sensory disturbance, and elevated cerebrospinal fluid protein without pleocytosis.

In GBS, respiratory failure is a life–threatening complication, and its occurrence in critically ill patients with multi-organ failure is very rare and may be difficult to predict.[1] Respiratory failure occuring in GBS patients is estimated at 25%.[2-4] It can present with atypical features particularly while attempting weaning from the mechanical ventilation, the so-called "difficult to wean patient". In these patients the diagnosis requires a high index of suspicion and meticulous careful work-up to confirm the diagnosis. It should include detailed history and physical examination, cerebrospinal fluid (CSF), and electrophysiological studies, course of illness, frequency of mechanical ventilation and attempted failure of weaning from mechanical ventilation.[5,6] This article attempts to alert physicians in the intensive care unit (ICU) to be aware of GBS in critically ill patients among other causes of "difficult to wean patients".

Case Report. A 65-year-old Saudi female, referred to the ICU at King Fahd Hospital of the University, Al Khobar, Kingdom of Saudi Arabia, as a case of bilateral bronchopneumonia in acute hypoxic respiratory failure. Her past history is significant of diabetes with all its complications and hypertension which is controlled on insulin and captopril. The general physical examination revealed a middle-aged female with endotracheal tube in place, significant pallor but no cyanosis and engorged neck veins, bilateral rales, loud S1 and S2 with summation gallop, no murmur. Abdominal examination was not remarkable. In the lower limb edema was present. The neurological examination showed hypersomnolance, no papilledema, and mild generalized weakness. Muscle stretch reflexes were all absent and the planter responses were flexor. The patient was managed by sedation and pancuronium as the paralyzing agent, which was withdrawn after 24 hours. She was continued on sedation-analgesia with (fentanyl and midazolam) in addition to tazocin 4.5g every 6 hours, insulin drip, furosemide, and renal dose of dopamine. On the 14th day, she was extubated successfully with satisfactory condition,

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and transferred 48 hours later to the general medical ward. Ten days later, she was transferred back to the ICU due to possible aspiration pneumonia with oxygen saturation <80% in acute respiratory failure. Her clinical and radiological evaluation was suggestive of right lower lobe pneumonia. The patient was intubated again and sedated. After improvement of her chest infection, repeated attempts to wean her off the ventilator failed. She was noted to be progressively weak. A neurological consultation revealed mild bilateral lower motor neuron facial weakness with flaccid areflexic weakness suggestive of the diagnosis of GBS, however critical care illness neuropathy and myopathy needed to be ruled out. The CSF results showed clear, colorless fluid. White cell count was 3.0. Cerebrospinal fluid protein was 85 mg/dl. The CSF glucose was 173 mg/dl and serum glucose was 350 mg/dl. The nerve conduction studies revealed prolonged distal motor latencies (DML) and slow motor conduction velocities (MCV) (namely ulnar nerve DML=5.0m/sec; MCV=3.4m/sec) with dispersion of the compound muscle action potential (CMAP) characteristic of acute inflammatory demyelinating polyneuropathy or GBS with evidence of axonal involvement on electromyography **(Figure 1)** The patient was started on immunoglobulin therapy with 0.5g/kg/day for 4 days, physiotherapy and supportive therapy. She was successfully extubated after 7 days and transferred to the general medical ward where she continued on physiotherapy with steady improvement.

Discussion. The differential diagnosis of generalized weakness of peripheral origin in the ICU in critically ill patients with multi-organ failure syndrome (MOFS), illnesses such as diabetes mellitus[7,8] and multiple drug therapies includes a wide range of causes namely neuropathies, neuromuscular junction defects and myopathies[9] **(Table 1).** However, the differentiation between these disorders requires careful clinical examination and detailed neurophysiological studies to attempt localization and correct diagnosis **(Table 2).** The differential diagnosis of non-pulmonary causes of the "difficult-to-wean" patients from mechanical ventilation, prolonged ventilation or both include neuropathies namely; acute inflammatory

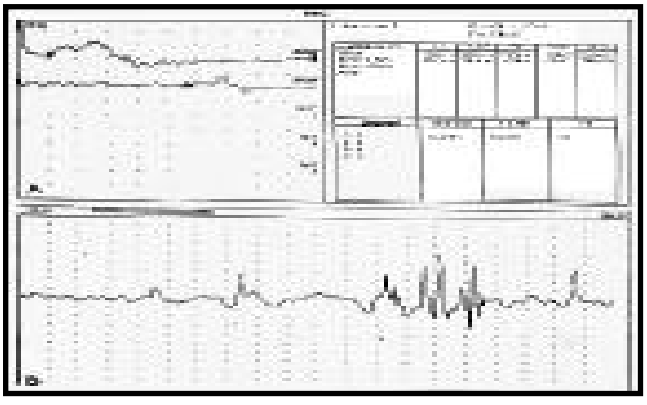

Figure 1 - Electromyographic study of a patient showing delayed distal motor lagency, slow conduction velocity, dispersed, low amplitude **a)** Compound muscle action potential with block and **b)** polyphasic, long duration motor unit potentials.

Table 1 - The differential diagnosis of acute generalized weakness in the intensive care unit.

Peripheral nerve	Neuromuscular Junction	Muscle
Critical illness polyneuropathy	Persistent neuromuscular blockade, namely by Amino glycoside	Acute quadriplegic myopathy
Guillain-Barre syndrome	Myasthenia gravis	Hypokalemia
Acute intermittent porphyria	Hypermagnesemia	Periodic paralysis*
Vasculitis	Botulism	Metabolic/ inflammatory myopathy
Acute massive intoxication	Hypokalemia	Disuse atrophy Rhabdomyolysis Critical illness myopathy
* Seldom causes respiratory failure		

Table 2 - Clinical features of common causes of generalized weakness in the intensive care unit.

Lesions	Weakness	Cranial Nerve Involvement	Reflexes	Sensory Deficits
Brainstem	Assymetric quadriparesis	Common	Increased	Common
Spinal cord	Paraparesis	None	Reduced early increase later	Below lesion
Guillain-Barre syndrome	Distal	Common	Absent	Common
Critical illness polyneuropathy	Distal	Rare	Absent	Present
Myasthenia Gravis	Proximal	Common	Normal	None
Acute quadriplegic myopathy	Diffuse	Rare	Normal or reduced	None

polyneuropathy (GBS), which occasionally develops in the setting of pre-existing critical illness, systemic presentation of diseases such as porphyria and vasculitis, amyotrophic lateral sclerosis, polyneuropathy of critical illness, both motor and sensory neuropathy due to sepsis, MOFS, burns and trauma. The neuromuscular junction defects include patients with myasthenia gravis exacerbated by infection or medication. Myopathies include inflammatory and glycogen storage disorder, periodic paralysis and weakness due to electrolyte disturbances, acute quadriplegic myopathy (AQM).[10] This patient demonstrated GBS as a cause of respiratory failure, which has been masked by the presence of diabetes and sepsis.[11] Reaching this correct diagnosis has facilitated early intervention in the form of appropriate therapy, tracheotomy, extensive pulmonary and general rehabilitation, which reduced or shortened the morbidity and possibly prevent mortality.[12]

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