Case Report

Guillain-Barre syndrome complicated by acute fatal rhabdomyolysis

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Abstract

Guillain-Barre syndrome (GBS) is a heterogenous group of peripheral-nerve disorders with similar clinical presentation characterized by acute, self-limited, progressive, bilateral and relatively symmetric ascending flaccid paralysis, which peaks in 2-4 weeks and then subsides. The usual complications, which occur in a patient of GBS are pneumonia, sepsis, pulmonary embolism, respiratory insufficiency and cardiac arrest. The clinical course of GBS complicated by acute rhabdomyolysis is extremely rare. We present the case of GBS with marked elevation in serum creatine kinase, serum myoglobin levels and persistent hyperkalemia as a result of associated acute rhabdomyolysis.

Keywords: Acute kidney injury, creatine kinase, Guillain-Barre syndrome, hyperkalemia, rhabdomyolysis

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Introduction

Guillain-Barre syndrome (GBS) is an acute immune-mediated polyradiculopathy that presents as a rapidly progressive, areflexic, symmetric ascending motor paralysis with or without sensory disturbances. The clinical course of GBS can be complicated by pneumonia, sepsis, pulmonary embolism, respiratory paralysis or cardiac arrest.[1] Diagnosis of GBS is usually based on clinical examination and is supported by nerve conduction studies and cerebrospinal fluid (CSF) examination.[2] Mild elevation in serum creatine kinase (CK) level have been seen in early stage of GBS, but marked elevation of CK in GBS is an extremely rare finding.[3] We present the case of GBS with marked elevation in serum CK, serum myoglobin levels and persistent hyperkalemia as a result of associated acute rhabdomyolysis, which is a potentially life-threatening condition.

Case Report

The present case report is about a 24-year-old Indian man who presented with 20 days history of weakness of both lower and upper extremities. The weakness began suddenly in both lower extremities, which progressed to both upper extremities over 2 days. He was confined to bed for 15 days prior to admission in hospital. Five days of fever preceded the onset of weakness by 10 days. He had no sensory or bladder symptoms. He had no history of trauma, toxin exposure, illicit drug or alcohol abuse. On general examination, he was afebrile, with a pulse rate of 106/min, blood pressure of 110/70 mmHg and respiratory rate of 18/min. There were no pressure sores or any muscle tenderness present on physical examination. On neurological examination, he had flaccid, areflexic, pure motor quadriparesis with strength graded as 2/5 in both lower extremities and 3/5 in both upper extremities. Bilateral plantar reflexes were not elicitable.

His laboratory tests on hospital day 1 revealed hyperkalemia, elevated liver enzymes (aspartate aminotransferase, 274 IU/L; alanine aminotransferase, 476 IU/L), leukocytosis (14,100/mm^3) and deranged renal profile [Table 1]. His electrocardiogram showed tall T-waves, absent P-waves and widening of QRS complexes consistent with severe hyperkalemia [Figure 1]. He was treated with intravenous infusion of insulin-dextrose and calcium gluconate for hyperkalemia.

His CSF examination revealed a protein of 68 mg/dL, glucose of 90 mg/dL and a CSF cell count

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Table 1: Serum laboratory test results during patient's hospital course

Lab tests	Day 1	Day 2	Day 3
Creatinine (mg/dL)	1.9	2.4	4.5
Urea (mg/dL)	48	76	188
Potassium (mEq/L)	7.3	8.1	8.8
Creatine kinase (U/L)		279	7002

of 2 leukocytes/mm^3. Electrophysiological studies confirmed the presence of acute motor and sensory axonal neuropathy. It showed low amplitude of compound muscle action potential and nerve action potential, with near normal conduction velocity and the presence of fibrillation and positive sharp wave potentials consistent with axonal neuropathy. His serological tests were negative for leptospira, dengue, human immunodeficiency virus, antinuclear antibodies, hepatitis B virus and hepatitis C virus. His blood and urine cultures were sterile. Thyroid stimulating hormone and free thyroxine levels were within the reference range.

Immunoglobulins could not be given to him for the treatment of GBS due to financial constraints. On hospital day 2, he developed hypotension. Inotropic support (noradrenalin and dopamine) was started after adequate fluid repletion. On hospital day 3, he was not able to lift his head off the pillow and cough out his secretions. He was put on a mechanical ventilator for respiratory failure. In view of rising renal parameters, oliguria, proteinuria (2+ on urine dipstick test) and persistent hypotension, peritoneal dialysis was started [Table 1].

A possibility of coexisting chronic renal insufficiency or acute rhabdomyolysis was kept in view of the presence of persistent hyperkalemia, hyperphosphatemia (10.8 mg/dL), hyperuricemia (14.2 mg/dL), hypocalcemia (7.4 mg/dL) and high anion gap metabolic acidosis. His renal profile prior to admission was normal and there was no evidence of chronic kidney disease on abdominal ultrasonography. His biochemical analysis on day 3 of admission revealed serum CK of 7002 U/L (n. 38-174), lactate dehydrogenase (LDH) of 1250 U/L (n. 91-180), serum myoglobin levels were above 10,000 µg/L (n. 10-46) and urine myoglobin levels were 25.7 µg/L (n. 0-1000). His serum CK was 279 U/L and 44 U/L on day 2 of admission (in our hospital) and 3 days prior to admission (in other private hospital from where he was referred) respectively [Table 1]. No evidence of muscle necrosis was seen in muscle biopsy. A final diagnosis of GBS complicated by acute kidney injury (AKI) and rhabdomyolysis was made. His hyperkalemia persisted despite all the conservative measures. On day 4, he developed a cardiac arrest, from which he could not be revived. There was no history suggestive of inherited muscle disease or recurrent episodes of rhabdomyolysis.

Discussion

Rhabdomyolysis is a syndrome which results from skeletal muscle injury with the release of intracellular muscle cell contents including electrolytes, myoglobin and other muscle enzymes (creatinine kinase, aldolase, LDH, aminotransferases) into the systemic circulation.[4,5] The common causes of rhabdomyolysis are trauma, vigorous exercise, burns, alcohol, seizure, metabolic abnormalities, drugs, toxins and infections.[4]

Acute viral myositis accompanied by rhabdomyolysis has been reported in the literature.[1,6,7] The mechanisms underlying rhabdomyolysis due to infection are direct muscle invasion, toxin production and myotoxic cytokines released in response to viral infection.[8]

Post-viral immune-mediated damage to muscle cells leading to acute rhabdomyolysis may be the possible etiology in our case of GBS. Negative history of trauma, illicit drug or alcohol abuse, toxin consumption or recurrent episodes of rhabdomyolysis in the past ruled out the other possible causes of rhabdomyolysis.

Volume depletion as a result of influx of extracellular fluid into injured muscles resulting in renal ischemia, tubular obstruction due to heme pigment casts and direct nephrotoxic effects of liberate muscle cell constituents may all contribute to the development of AKI in our patient. The risk of AKI in patients with rhabdomyolysis is lower in patients with CK levels at presentation less than 15,000 U/L. The risk factors for AKI in patients with lower CK values include dehydration, sepsis and acidosis.[5] Persistent hyperkalemia not responding to conservative measures in our case might be due to an additive effect of massive release of cellular potassium as a result of rhabdomyolysis, impaired excretion of potassium as a consequence of AKI and reduced sodium and water delivery to the sites of potassium secretion in the distal nephron due to reduced effective arterial blood volume.

Mild to modest raised levels of CK have been documented in the early stages of GBS, the cause of which is uncertain.[3] The possible mechanism proposed in the literature is rapid extensive denervation due to severe axonal degeneration of motor nerve terminals can result in the release of muscle enzymes (CK).[9] Acute

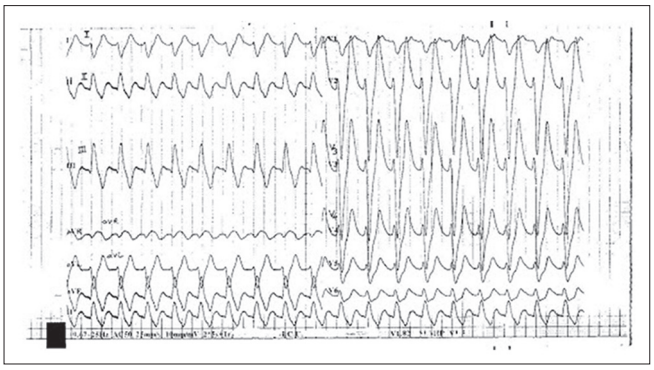

Figure 1: Electrocardiogram showing tall T-waves, absent P-waves and widening of QRS complexes

rhabdomyolysis leading to markedly raised CK levels in a GBS patient is extremely rare.[10]

Conclusion

Acute rhabdomyolysis should be searched for a possible cause in a patient of GBS presenting with hyperkalemia. Its early recognition and institution of the appropriate management could prevent the fatal complications such as AKI, compartment syndrome, disseminated intravascular coagulation, multi-organ failure and death.

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