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CASE REPORT

A Rare Case of Acute Axonal Motor Neuropathy in a Puerperal Woman

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ABSTRACT

We report the case of a puerperal woman who presented to us with sepsis with multiorgan dysfunction and motor weakness of both lower limbs. On detailed evaluation, patient was found to have axonal neuropathy establishing the diagnosis of critical illness polyneuropathy. A high index of suspicion is required to arrive at the diagnosis as this condition is not only associated with high mortality and morbidity rates but also can affect the quality of life of the individual in the long term. This case has been reported to highlight the importance of recognition of this common, but rarely diagnosed condition as it can help us to portend the prognosis.

Keywords: Axonal polyneuropathy, Critical care, Sepsis.

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INTRODUCTION

Critical illness polyneuropathy (CIP) is an acute disorder of neuromuscular system affecting severely ill patients. Complex pathogenetic mechanisms have been hypothesized involving metabolic, inflammatory, and bioenergetic alterations supporting microvascular changes in peripheral nerves, though its etiology remains unclear.[1] Usually, the motor nerve fibers are affected. Sensory fibers and cranial nerves are generally preserved.[2] Multiorgan failure, sepsis and critical illness polyneuropathy have a mortality rate of 50%.[3] We describe a patient who developed sepsis and went into multiorgan dysfunction with lower limb weakness following delivery which prompted us to diagnose CIP.

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CASE REPORT

A 25-year-old puerperal lady presented with acute onset of pain, swelling and weakness of lower limbs for 3 days. The patient was a para 1 living 1 abortion 2 and had underwent an emergency cesarean section for fetal distress 3 days prior to admission. Patient had history of fever 1 week prior to delivery. She had no previous hospitalizations or significant medical illness in the past. On examination, she was conscious and well oriented. She was febrile with a temperature of 100°F. There was pedal edema, pallor and icterus. Pulse was 92/min and BP was 140/80 mm Hg. Per abdominal examination revealed an involuting uterus. Examination of the lower limbs showed that both limbs had pitting pedal edema. Peripheral pulses were felt in both lower limbs. There was hypotonia of the lower limbs (hip flexors, extensors 1/5, quadriceps 2/5, distally 3/5). Deep tendon reflexes were absent. There was no sensory deficit. The routine urine examination showed no sugar or protein. Her Hb was 9.3 gm/dl, WBC 19600/mm^3 and platelets 1,20,000/mm^3. The serum sodium was 134 mEq/l and potassium was 8 mEq/l. The hyperkalemia was corrected by parenteral infusion of calcium gluconate and 25% dextrose and insulin. The blood urea was 30 mg/dl and creatinine 2.5 mg/dl. The serum bilirubin was 7.9 mg/dl, direct bilirubin 7.26 mg/dl, SGOT was 40 U/I and SGPT was 35 U/I. Prothrombin time was 18.4 seconds and activated partial thromboplastin time was 32.5 seconds and INR was 1.28. She was treated with parenteral piperacillintazobactam and metronidazole. Nerve conduction study was done and the electrophysiological studies (EPS) showed marked decrease of compound muscle action potentials in bilateral common peroneal nerve. The sensory nerve action potentials were normal in the upper and lower limbs. The findings were suggestive of bilateral common peroneal nerve axonal motor neuropathy. Venous Doppler of the lower limbs was found to be normal. Ultrasound of the abdomen showed moderate ascites and right pleural effusion and involuting uterus. With supportive

treatment, patient recovered and she was discharged 15 days later with the advice to continue exercises at home. The patient has had regular follow-up for 3 years after discharge and she has regained near normal power in her lower limbs.

DISCUSSION

By definition, CIP is an acute reversible neuropathy that develops during the treatment of critically ill patients.[4] This newly acquired neuromuscular cause of weakness has been found in 46% (95% confidence interval 43 to 49%) critically ill patients with sepsis, multiorgan failure or prolonged mechanical ventilation.[5] Our patient presented with sepsis and multiorgan failure. Laboratory investigations are nonspecific.[6] Electrophysiologic findings are those of a pure axonal dege-neration, affecting motor than sensory fibers.[7] In this patient, the electrophysiological studies showed marked decrease of compound muscle action potentials in bilateral common peroneal nerve and normal sensory nerve action potentials. Sepsis, hyperglycemia and decreased serum albumin concentrations are asso-ciated with decrease in peripheral nerve function.[8] The serum albumin in this patient was low (1.3 gm/dl). Treatment is supportive, initially consisting of aggressive pulmonary hygiene and prevention of secondary complications of immobility, such as skin breakdown, deep venous thrombosis and superimposed compressive neuropathies.[7] Our patient recovered with symptomatic treatment. The recovery in patients with CIP is spontaneous but gradual.[9] Critical illness polyneuropathy or critical illness myopathy is associated with increased ICU and hospital stays and elevated mortality rates, although other data suggest that patient selection may partially explain this.[10] Our patient is symptomatically better on follow-up and regained normal power in both her lower limbs.

CONCLUSION

This case has been presented to highlight the importance of considering this possibility in patients who present with weakness of lower limbs in the presence of severe sepsis as this can help us to prognosticate the disease.

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