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Case report

CRITICAL ILLNESS MOTOR NEUROPATHY IN A PUERPERAL WOMAN WITH SEPTIC ABORTION: A CASE REPORT

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Abstract

A puerperal woman presented to us with sepsis, multiple organ dysfunction and paraparesis with a history of abortion done at home by a dai. On evaluation, she was found to have critical illness motor neuropathy which was established on electrophysiologic studies and her paraparesis resolved gradually on treating her infection and she regained near normal power. This case has been reported to highlight the importance of recognition of this common but rarely diagnosed condition as if the underlying cause is treated successfully, full recovery from critical illness neuropathy can occur, which usually takes a few weeks to months depending upon the severity of the cause.

Keywords: Motor Neuropathy, Puerperal Woman, Case Report

INTRODUCTION

Critical Illness Polyneuropathy(CIP) is an acute axonal neuropathy that develops during a severe illness. Clinical manifestations include muscle weakness, which is usually more pronounced distally than proximally and is often accompanied by atrophy, delayed weaning (if on a ventilator), and delayed recovery (mobility). The pathogenesis is not clearly understood but most people believe that the inflammatory cascade which mediates the systemic inflammatory response and multiple organ failure plays an important role. The electrophysiologic studies (EPS)reveal a predominantly motor[1] and sometimes a sensory axonal polyneuropathy. The incidence of CIP is high, with often more than 50% of patients in major medical and surgical critical care units. The neurologic effects are most likely mediated by inflammatory mediators like cytokines and free radicals which affect the microcirculation of the central and peripheral nervous system[2].Multiorgan failure, sepsis and CIP have a mortality rate of approximately 50%.[3]

CASE REPORT

A 25 year old puerperal lady presented with acute onset of pain,swelling and weakness of both lower limbs for 3 days. The patient was para 3 living 1 abortion 2 and had underwent an abortion from local dai 3 days prior to admission. There was no history of bladder and bowel

involvement, trauma, difficulty in swallowing, back pain, tingling and numbness in limbs. She had no previous hospitalizations or significant medical illness in past. On examination, she was conscious and well oriented. There was pedal oedema, pallor and icterus. The patient was febrile with a temperature of 100^{o}F.Blood pressure was 140/80 mm of Hg in right arm supine position, pulse rate was 92/min, and oxygen saturation was 94% on room air. Examination of lower limb showed that both limbs had pitting pedal edema and allperipheral pulses were felt. There was hypotonia of lower limbs and power(right and left hip flexor 3/5,extensor2/5,right knee flexor 2/5, left knee flexor 2/5,right and left knee extensors 1/5,ankle flexion 1/5, and extension 1/5 in both limbs). Deep tendon reflexes were absent. Plantar was bilaterally not elicitable. There was no associated sensory deficit.Chest auscultation revealed bilateral basal crepitations. Cardiovascular system examination was within normal limits and per abdomen examination revealed ascites with no palpable organomegaly.

Her Hb was 9.3gm/dl,WBC 19600/mm^3($N_{90}L_{08}E_1M_1$),platelet count was 1,20,000/mm^3, serum sodium was 134mEq/l and potassium was 8mEq/l. The hyperkalemia was treated by parenteral infusion of calcium gluconate and insulin in 25%dextrose and nebulization with salbutamol. However, there were no improvement of symptoms and within the next 24 hours the potassium level came to be normal(K^+=4.8).Blood urea was 30mg/dl and serum creatinine 2.5mg/dl,serum bilirubinT/D/I were 7.9/7.2/0.7 mg/dl, SGOT was 40 IU/ml, SGPT was 45IU/ml. Prothrombin time (PT) of the patient was 18.4 seconds and INR was 1.28.The activated partial thromboplastin time (aPTT) was 32.5 seconds. The routine urine examination showed no sugar or protein. Nerve conduction study was done which showed marked decrease in compound muscle action potentialin bilateral common peroneal nerves while the sensory nerve action potentials were normal in both upper and lower limbs. The findings were therefore suggestive of bilateral common peroneal nerve axonal motor neuropathy.Venous Doppler of lower limbs was found to be normal, ultrasound of abdomen showed moderate ascites, rightsided pleural effusion and an involuting uterus. She was treated with parenteral piperacillin-tazobactam and metronidazole in appropriate doses along with supportive treatment. The patient recovered and she was discharged 15 days later with advice to continue exercise at home. The patient has had regular follow upfor 3 years after discharge and she has regained near normal power in her lower limbs.

DISCUSSION

CIP is an acute reversible neuropathy that develops during a critical illness[4]. This newly acquired neuromuscular cause of weakness has been found in 46%critically ill patients with sepsis, multiple organ failure or prolonged mechanical ventilation[5]. Our patient presented with sepsis and multiple organ failure. Laboratory findings showed hyperkalemia, which can cause weakness that should improve after correcting hyperkalemia[6]but our patient showed no improvement. Electrophysiological findings revealed an axonal degeneration affecting only motor nerve fibres[7].The recovery in CIP patients is gradual[8]andour patient is showing symptomatic improvement on regular follow up and has near normal power in both her lower limbs.

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