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Journal of Society of Anesthesiologists of Nepal

Case Report

ICU acquired weakness or something else?

Subhash Acharya, Sabin Koirala, Anjan Shrestha, Diptesh Aryal

***Tribhuvan University Teaching Hospital, Institute of medicine, Maharajgunj Medical Campus, Institute of Medicine, Maharajgunj, Kathmandu, Nep**al.*

ARTICLE INFO

Article history

Received 20.08.2014

Accepted 02.12.2014

Published 26.02.2015

Abstract

An elderly female presented to Intensive Care Unit with generalized weakness and altered sensorium with past history of adenocarcinoma of larynx. She was managed with Radiation Therapy and Chemotherapy in the past. Current admission to the intensive care unit was for aspiration pneumonitis and respiratory failure requiring mechanical ventilation. She then progressively developed muscle weakness, which was provisionally diagnosed and managed as critical illness polyneuromyopathy (ICU acquired weakness) and muscle biopsy was sent, as there were no obvious neurological signs and she was not recovering from her muscle weakness as well. She then developed Catheter related sepsis and refractory septic shock, and then she passed away. Her muscle biopsy report that was available postmortem revealed that she had severe toxoplasma infestation that remained in shadow.

Keywords: Critical illness polyneuropathy; myopathy; ICU acquired weakness; Toxoplasmosis

How to cite this article: Acharya SP. ICU acquired weakness or something else? JSAN 2015;2:25-27.

Case Report

A 71 years female with past medical history of Adenocarcinoma of Hypopharynx two years back, treated with radiotherapy and chemotherapy, presented to Emergency Department with altered sensorium. She was drowsy, afebrile, with respiratory rate of 22 per minute, heart rate of 89 bpm, and blood pressure of 90/60 mmHg. Her oxygen saturation on was 49% in room air, which increased to more than 88% with oxygen via facemask. On auscultation of her chest, she had bilateral crepitations, more on right than left. There were no other significant clinical findings. Blood investigations showed Hyponatremia (Na: 114 mEq/L) and raised Creatinine (118 micromol/L). Chest X-ray showed minimal bilateral infiltrates. She was started on IV fluids and was admitted to ICU with the clinical diagnosis of Hyponatremia with Aspiration pneumonia. She was then managed with intravenous (IV) fluids and IV antibiotics along with management of hyponatremia. Patient's condition deteriorated on second day of admission. She became hypotensive with drop in oxygen saturation, for which she got intubated and kept in mechanical ventilator and vasopressor (Norepinephrine) was started.

Corresponding author:

Subhash P Acharya, MD, FACC, FCCP

Intensivist and Clinical Coordinator, TUTH ICU

Lecturer, Department of Anaesthesiology, Institute of Medicine (IOM)

Telephone: 977-9851147242

Email: drsuvash@gmail.com

At this time, her Glasgow Coma Scale (GCS) was E4 M6 VET; she was awake and was following commands. All the reflexes were normal; tone and power were also normal on all her four limbs. She was intubated with 7.5 mm ID cuffed endotracheal tube and was fixed at 20 cm at incisors, and was kept on Volume Assist Control mode and was breathing 5 – 6 breaths above the ventilator. She was afebrile and had heart rate of 112 per min, was in sinus rhythm and she was requiring Norepinephrine support at 0.2 mcg/kg/min for a target MAP of more than 70 mmHg. She was then managed symptomatically and her status started to improve over the next two days. Hyponatremia was corrected, chest infection was better and Norepinephrine was only at 0.05mcg/kg/min but could not be off. Then weaning was started on Pressure Support with PEEP (10/5/40%). From the fourth day of ICU admission, her neurological status deteriorated further with decrease in GCS (E3M3VT), for which Neurology consultations was made and her fall in GCS was attributed to PRN Fentanyl that she was receiving on need basis. Laboratory tests showed increase in total leukocyte counts (16,100/mm3), thrombocytopenia (69000/mm3) and hypokalemia (2.2 mEq/L). Hypokalemia was managed with potassium replacement and there were no evidence of hypomagnesaemia. She also developed fever upto 39 degree Celcius. Surprisingly, on 6th day in ICU, her GCS improved to E4M5VET, but she couldn't move her all four limbs. Power was 1/5 in all four limbs, and there was no evidence of increased tone and reflexes. Neurology consultation advised for CT scan of the Head, which was done and was normal. Neurology team then diagnosed her as Critical illness polyneuromyopathy (CINM) as she has been ill before admission too. Nerve conduction studies were thought of but could not be done, as they are not available at bedside. The ICU team was not convinced with the diagnosis of Critical illness polyneuromyopathy and thus decided to send muscle biopsy. Surgical consult was done and Right Deltoid Muscle Biopsy was done under local anesthesia in the ICU and was sent for histopathology. She was further continued on conservative management but on the 8th day, she was further hypotensive requiring more Norepinephrine and Vasopressin was also added. Her GCS again dropped (E3M2VT) and was back on Assist control ventilation. Blood culture from Right Internal Jugular Central venous catheter grew *Burkholderia cepacia complex* and thus Right IJ catheter was removed and another CV catheter was inserted in Left Subclavian Vein and antibiotics were changed as per sensitivity. On the 9th day, her BP dropped and she went into bradycardia and asystole and CPR was done for 3 minutes and was reverted back to sinus tachycardia. She was coagulopathic with platelet count of 38000 per cubic mm with some evidence of endotracheal tube bleeding. Therapeutic hypothermia was not induced and she was being managed with Platelets transfusion. From the 12th day, her GCS was E4M1VT, TLC decreased 3200, platelets were persistently low and on 15th day she again went into Asystole and had ROSC after 5 minutes of CPR. On 16th day, GCS dropped to E1M1VT with continued conservative management with inotropes, blood product transfusion. Epinephrine was added for refractory hypotension and on 17th day, she didn't respond to any inotrope or vasopressor and then arrested and could not be revived. Her family members cremated her and autopsy was not performed. But a week later after her death, histopathology report of deltoid muscle biopsy was available, which revealed muscle fibers with bradyzoite cysts consisting of numerous bradyzoites consistent with muscular Toxoplasmosis.

Discussion

Critical illness polyneuromyopathy (CINM) or ICU acquired weakness is a primary axonal degeneration of motor and sensory fibers that lead to generalized skeletal muscle weakness. It is not simply the result of physical inactivity, but is due to imbalances in the structure and function of peripheral nerves and skeletal muscle.[1]

There are inconsistencies in reporting, testing and terminology of CINM, so incidence is difficult to ascertain. Prospective studies using neurophysiological testing reveal that CINM is present in 33% to 55% of patients admitted in ICU for more than 7 days.[2,3] The incidence may be even higher in patients with sepsis and SIRS: 68% to 100%.[4]

Our patient was subjected to a number of differential diagnoses for muscle weakness including electrolyte imbalances (hypokalemia, hyponatremia); ICU acquired weakness (ICU-AW) due to sepsis, use of steroids, mechanical ventilation and use of muscle relaxants. Stroke and Cervical spinal cord injury was ruled out clinically and with CT scan.

CINM has many impacts on ICU outcomes, including, difficulty weaning and prolonged mechanical ventilation, increased ICU length of stay, and increased morbidity and mortality. In fact, after cardiac and pulmonary causes have been ruled out, it is the most common cause for difficulty weaning from mechanical ventilation.[5,6] Apart from difficulty in weaning and prolonged mechanical ventilation, most significant clinical significance lies in its association with other secondary complications like pneumonia, deep vein thrombosis and pulmonary embolism.[7] We faced a similar situation in our patient.

There are numerous risk factors for development of CINM and there can be numerous differential diagnoses. The common risk factors are: systemic inflammation, medications (corticosteroids, muscle relaxants), poor glycemic control and immobility.[8]

The differential diagnosis for the development of neuromuscular weakness in the ICU includes:[1,2]

CNS Disorders: encephalopathy, Stroke, Spinal Cord Injury, Anterior horn cells disorder (amyotrophic lateral sclerosis)

Neuromuscular junction disorders: Myasthenia Gravis, Lambert-Eaton myasthenic syndrome

Peripheral Neuropathies: Gullian Barre Syndrome (GBS), Diabetic neuropathy, Thiamine deficiency, drug-induced neuropathies (chemotherapy, anti-retro virals)

Muscular Disorder: muscular dystrophies, myotonic dystrophy, polymyositis, poliomyelitis, steroid-induced myopathy, uremia, glycogen storage disorders

Physical examination, electrophysiological studies and finally muscle biopsy is needed for diagnosing ICU-Acquired Weakness. Our patient, on physical examination, had a symmetric weakness and even on deep painful stimuli there was no motor response, apart for some facial grimacing. Whenever these findings are present, CINM should be suspected.[9] Electrophysiologic studies could not be done in our patient because some degree of voluntary muscle contraction is required for these studies. Muscle biopsy is the method of choice for the diagnosis of structural abnormalities of the peripheral muscles. Muscle biopsy was done in this case, and it showed Toxoplasmosis.

Toxoplasmosis is caused by the intracellular protozoal parasite, Toxoplasma gondii, usually acquired by the ingestion of infectious oocysts from the environment, ingestion of tissue cysts in meat from an infected animal or can be transmitted vertically from mother to fetus or via blood transfusion or organ donation.[10] Ingestion of undercooked meat is responsible for the majority of toxoplasmosis cases in some parts of the world including Nepal, Europe and USA.[11,12] Usually immunocompetent hosts are asymptomatic. When symptomatic infection occurs, the most common manifestation is bilateral, symmetrical cervical lymphadenopathy. Constitutional symptoms like fever, chills and sweats may be present. However, extremely rare cases of pneumonitis, ARDS, myocarditis, pericarditis, polymyositis, hepatitis and encephalitis have been reported.[13-15] This case presented as a rare case of Toxoplasmosis with involvement of muscle, CNS and respiratory system.

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