CASE REPORT

Japanese encephalitis

Zia Arshad*, Sulekha Saxena**, Ritu Verma*, Haidar Abbas***, GP Singh****, Jai Shree Bogra*****

**Assistant Professor, **Senior resident, ***Professor, ****Professor & In-charge, Trauma Ventilatory Unit , *****Professor and Head of Department Department of Anaesthesiology, King George's Medical University, Shah Mina Road, Chowk, Lucknow, Uttar Pradesh 226003, (India).*

Correspondence: Dr.Sulekha Saxena, Senior resident, Department of Anaesthesiology, King George's Medical University, Shah Mina Road, Chowk, Lucknow, Uttar Pradesh 226003, (India); Cell: 09359618480; E-mail: dr.sulekha2008@rediffmail.com

ABSTRACT

We present a case of Japanese encephalopathy, which posed serious problems during diagnosis as well management. Our patient had to be nursed in ICU and mechanically ventilated for a prolonged period (more than 50 days) and was successfully weaned, but with difficulty and with sustained and careful monitoring and flexible planning. A multispecialty approach made all the difference between life and death.

Key words: Japanese encephalitis; Hospitalization; Critical illness polyneuropathy; Critical illness myopathy; Mechanical ventilation; ICU

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INTRODUCTION

Japanese encephalitis (JE) is one of the leading causes of acute encephalopathy in the tropics. It mainly affects children <15 years and is mostly asymptomatic.[1] When symptomatic, it usually resolves within weeks and ventilator support is not commonly required. Hospitalization is mainly due to neurological symptoms and respiratory involvement may or may not occur. After a prolonged ICU stay weaning from ventilator can be difficult and can have multiple causes. Critical illness polyneuropathy is the most common peripheral neuromuscular disorders encountered during difficult weaning in ICU setting.[2]

CASE REPORT

A 17 year old boy presented with generalised headache, evening rise of temperature, altered sensorium and un-coordination of movement for 5 days. He later developed difficulty in breathing for which he was referred to our centre.

On admission, he was febrile with a temperature of 101°F, pulse of 134/min blood pressure of 88/47. On pulse oximetery 91% saturation with signs of respiratory distress. The peripheries were cool, pale and clammy. Arterial blood gas analysis revealed PO_2- 58.7, PCO_2-59.7, oxygen saturation of 88%. Tracheal intubation was performed to aid mechanical ventilation; SIMV mode with PS-18, PEEP of 5 cmH_2O and FiO_2 of 80% and RR-14.

Systemic inflammatory response syndrome was provisionally diagnosed and treatment was initiated as recommended by the surviving sepsis guidelines. A central venous catheter (CVC) was inserted via right internal jugular vein and a MAP of >70 mmHg was achieved with the help of fluid resuscitation and norepinephrine infusion. The patient was started on empirical antimicrobials which were later modified according to culture sensitivity.

On neurological examination his Glasgow Coma Scale (GCS) was E3VtM2 with rigid limbs. There were no signs of meningism. The rest of the systemic examination was within normal limit.

His baseline routine investigations were;

On day 2, MRI showed patchy T2 and Flair hyperintense involving basal ganglia and thalami with patchy restriction on DWL specific of JE. CSF examination showed features 0f viral encephalitis and a fourfold increase in IgM antibodies against JE. Antibodies against dengue, chikungunya virus (CHIKV), hepatitis virus and herpes simplex were negative, confirming the diagnosis of acute Japanese encephalitis with sepsis.

After 2 weeks there was improvement in his GCS, vital parameters, hemodynamic status and lab values indicating sepsis control; weaning was considered and the patient was given spontaneous breath trial. His ill-sustained efforts showed no improvement in saturation and ABG's. The patient underwent tracheostomy on day 15 after failing

multiple attempts at spontaneous breathing trials. He was continued on SIMV mode of ventilation for another 3 weeks and his nutrition was stepped up keeping in view his hypercatabolic state and increased demand due to increase in work of breathing.

Over a period of next 20 days patient was weaned off to spontaneous mode of ventilation; gradually decreasing the pressure support from 20 cmH_2O to 8 cmH_2O and increasing his trigger sensitivity. Improvement was evident in vital parameters, oxygen saturation, ABG's and chest x-ray. During this weaning period he was put off the sedation, whenever he developed a temperature of 38.5°C or more, systolic blood pressure greater than 140 mmHg, pulse rate of at least 130 beats per minute, respiratory rate of at least 20 breaths per minute, agitation, diaphoresis, and dystonia. These symptoms continued for more than three times a day pointing to clinical diagnosis of sympathetic storm after traumatic brain injury. Serum levels of epinephrine during episodes were not done but patient responded well to clonidine 0.1 mg. T-piece trial was considered after patient responded to clonidine and his hemodynamics became stable. It was initially given for 10-15 sec per day. Later over a period of 50 days the trail duration was increased up to 24 hours maintaining saturation and adequate P/F ratio in ABG's.

During his weaning period routine ICU care, chest and limb physiotherapy, and adequate nutritional support continued throughout. Anabolic steroids were given every 21 days to build his muscle mass and aid his weaning.

DISCUSSION

Japanese encephalitis (JE) virus has been known to be the leading cause of viral neurologic disease and disability in Asian countries. More than 100 days of mechanical ventilation in a patient of acute encephalitis makes it a case of prolonged weaning. The causes can be multiple in this patient and common causes in context would be discussed. Reduced central drive due to unresolved encephalopathy may be present. Patients do not exhibit any ventilatory activity upon discontinuation from the ventilator, and this persists despite hypercapnia and hypoxaemia. However, Japanese encephalitisis usually resolves in days to weeks and residual neurological disease could be present. Improvement in GCS and no development of any other neurological signs makes this diagnosis unlikely. Reduced drive can be due to metabolic causes like alkalosis and electrolyte disturbances. Hypophosphatemia, hypomagnesaemia and hypokalaemia all can cause muscle weakness.[3] Hypothyroidism and hypoadrenalism may also contribute to difficulty in weaning.Recent ABG and and electrolyte were well within normal limit for this patient. Malnutrition causes reduction of muscle mass, endurance, and muscle strength. It also causes decreased immunity, predisposing the patient to further infections. Nutrition repletion in critically ill patients showed improved respiratory forces and facilitated weaning.[4] Our patient was started on enteral nutrition from day 5 with anti-oxidant and trace element supplementation in accordandance to his needs for a hypercatabolic state and sepsis. Myopathies can be steroid or Neuromuscular myopathy and critical illness myopathy. Steroid induced myopathy in icu setting have been associated with long term use of steroid and neuromuscular agents along with aminoglycosides.[5] However, 5 days use of 100 mg hydrocortisone and normal levels of creatinine phosphokinase (CPK), AST, ALT ruled out steroid induced myopathy. Critical illness polyneuropathy are the most common peripheral neuromuscular disorders encountered in the ICU setting and usually involve both muscle and nerve[3]. Risk factors can be severity of illness, multiple organ dysfunction, SIRS, exposure to corticosteroids, presence of hyperglycaemia and prolonged ICU stay.[6,7] The signs and symptoms are distal muscle weakness, reduced or absent deep tendon relxess, weakness of respiratory muscle leading to difficulty in weaning.[8,9] It is an acute axonal sensorymotor polyneuropathy so electroneurography is the gold diagnostic standard[10] and neurologic examination is neither sensitive nor specific.[11] To confirm the diagnosis and differentiate it from other illnesses, electromyography (EMG) and nerve conduction studies were undertaken in this patient, which showed reduction of amplitude and duration of muscle compound action potentials and ± decreased amplitude of sensory nerve action potentials which is common in both critical illness myopathy and critical illness polyneuropathy. CPK levels were relatively normal (140 units/L) pointing more towards neuropathy. Fatigue of patients undergoing weaning from MV is a major factor in failure to wean. Ventilator associated diaphragm dysfunction may be one of the causes. 18 to 69 hours of complete diaphragmatic inactivity and mechanical ventilation results in marked atrophy of human diaphragm myofibers.[12] These findings are consistent with increased diaphragmatic proteolysis during inactivity. Oxidative stress due to critical illness can also be contributing, however, tocopherol, vitamin and trace element supplemantion have shown a role in reducing these risks.[13-15]

Difficult weaning as seen in this patient can be due to multiple causes and attributing to a single cause is difficult. However, as evidenced by the NCV critical illness polyneouropathy can be the main cause. Few of the risk factors were present in this case. Treatment is nonspecific and includes supportive care, personal hygiene, limb physiotherapy. Early mobilization of these patients may lead to a better outcome.[16]

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