Case Report

NEUROPATHIES IN SEPSIS: A DIFFICULT SITUATION TO WEAN FROM VENTILATOR.

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ABSTRACT:

Neuromuscular weakness in critically ill patients is diagnostic challenge. Septic Polyneuropathy is an important cause of failure to wean from artificial ventilation. We studied patient of septic polyneuropathy to highlight the importance of regular neurological examination in the early diagnosis of this conditions. Availability of facilities for bed side electrophysiological study & histopathology of muscle are important to diagnose these entity. A 56 years old lady was admitted in gastro-enterology unit with complains of abdominal pain & fever, subsequently she was diagnosed as a case of burst appendix with septicemia in Surgery Unit. Appendicectomy and surgical toileting was done under general anaesthesia. In the early post-operative period the patient developed respiratory failure and was transferred to ICU. She was on ventilator for a long time with all other investigation electrophysiological study of nerve conduction showed septic polyneuropathy. On 21st POD the patient could be withdrawn from ventilator and after T-piece trial extubation was done on the next day. Neuropathies in sepsis, an important cause of failure to wean from ventilator, a high index of suspicion and regular bed side neurological & electrophysiological examination is required to make an early diagnosis.

INTORDUCTION:

A number of disorders producing generalized neuromuscular weakness specifically associated with critical illness, these include neuropathies, myopathies & combinations of both[1].

Sepsis, neuromuscular blocking drugs (NMBA), disuse atrophy, asthma, corticosteroids, & the multiorgan failure have all been implicated. Two major subgroups are outlined. One is critical illness polyneuropathy (CIP) which is acute, diffuse mainly motor neuropathy is the commonest of these disorders, another is critical illness myopathy which is linked with asthma and with the use of corticosteroids,

NMBA and less convincingly, aminoglycosides and beta-adrenergic agonists.

In spite of existing knowledge of the condition, it is rarely diagnosed. Availability of facilities for bedside electrophysiological studies & histopathology of muscle are important to diagnose this entity[2].

We discuss the presentation, diagnosis and outcome of a patient with Septic Polyneuropathy with an aim to highlight the importance of regular neurological examination in the early diagnosis of this condition, as it might have a bearing on the management and prognosis of critically ill patients.

Case Report:

A 56 years old lady named Mrs. Tahera Malek, C/O Mr. G M Faruk, Mohammadpur, Dhaka was brought to Dhaka Medical College Hospital under gastroenterology unit on 10.04.08 with the complains of abdominal pain for 10 days and fever for seven days. In gastroenterology unit the patient was diagnosed as acute pancreatitis with hypokalamia with pseudocyst and treated accordingly. She was a known case of DM and hypertension. Aspirate from abdominal cavity examined for cytology & bacteriology. Gm(+ve), gm(-ve), bacteria and pus cell (+++) were present. No malignant cell was found. Injectable antibiotics were given with all other management.

But pain did not subside and then consulted with gynae & obs. unit and also with surgical unit. Patient was reffered to surgical unit where she

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diagnosed as intrabdominal abscess and decided for laparotomy. Patient was critically ill and was in septic condition. Just before operation patient was semiconscious, BP-160/90mmHg, pulse-132/min, R/R -20/min, RBS-9.5mmol/L, SPO_2-83%, ASA-4E. Then O_2 given by face mask @ 6 L/min and found SPO_2 98%, pulse 120/min BP-140/90mmHg. On 20.04.08. Induction of anaesthesia was done with inj. Propofol, Fentanyl & vecuronium & was maintained with 50% N_2O with O_2, vecuronium & halothene.

During laparotomy it was found that there was collection of pu33s in the illio-cecal region, appendix was burst and gangrenous condition. Appendisectomy and surgical toileting was done and intrabdominal drainage was given. During operative procedure haemodynamic condition was stable.

Unfortunately patient developed respiratory failure in the early post operative period. Patient was transferred to ICU with following condition: Pulse 123/min, BP- 200/90mmHg, heart-NAD, lungs-clear, SPO_2 95% with 100% O_2 with Bain circuit & controlled ventilation with muscle relaxant.

At ICU patient was managed as continuous Controlled ventilation with inj. PCB then gradually weaning by assessing the capability of the patients to maintain SPO_2 with less external support. On 1st POD patient was unconscious on assist controlled ventilation, pulse -70/min, BP- 170/90mmHg, Temp-98° F. On 2nd POD patient come conscious, gain respiratory effort, ventilator set with SIMV mode with FiO_2 .7, SPO_2 95%, pulse–100/min, BP- 120/70mmHg. But on 3rd POD patient develop respiratory distress, suction given, 100% O_2 given through Bain circuit & nebulization done. Respiratory distress dose not subside so put on ventilator with A/C mode again. Investigation CBC, serum creatinine, serum Urea, serum Electrolytes, ABG, RBS, Blood for MP (thick & thin film), ICT for p. falciparum, ECG, EMG, USG of whole abdomen, CXR done. All investigation was with in normal limit except hypokalamia which was corrected & electrophysiological study of nerve condition shows septic polyneuropathy. On 10th POD temp suddenly raised to 104° F. All investigation done repeatedly on every alternate day which was with in normal limit. Temp become normal on next day (11th POD) but ventilatory support can not be with drawn for many days. On 15th POD ventilatory support was with drawn, O_2 was given through T-piece with spontaneous respiration. Respiratory distress in spont. resp. through T-piece so patient again put on ventilator with SIMV.

Ventilatory support off on 21st POD & patient was kept spontaneous respiration through T-piece with O_2 5 L/min and extubation done on 22nd POD (10.05.08), O_2 given by face mask.

Next 3 days followed up at ICU on 24th POD patient was transferred to surgical word with following parameters:

Patient was conscious
Respirate spontaneously,
R/R-14/min
SPO_2 97%
BP- 100/70 mmHg

DISCUSSION:

Neuropathies in sepsis, an important cause of failure to wean from assisted ventilation are often missed due to lack of suspicion and initiative to undertake regular bedside neurological and electrophysiological examinations in critically ill patients. A high index of suspicion is required to make an early diagnosis [(2)]. Sepsis neuromuscular blocking agents (NMBA), disuse atrophy, asthma, corticosteroids and the multiorgan dysfunctions syndrome (MODS) have all been implicated. In CIP there is flaccid paralysis of all the four limbs and absent deep tendon jerk [(3)].

The frequency of CIP is approximately 70% in patient with sepsis. Prospective studies have explored the causality and clinical outcome of CIP. Clinical outcome of patients and CIP includes difficulty weaning from mechanical ventilation, increased length of stay, prolonged recovery and an over all mortality rate of 26-71%.

Hyperglycemia, sepsis and decreased serum albumin concentration are associated with decrease in peripheral nerve function. Cytokines secreted in sepsis increases microvascular permeability leading to endoneural oedema which causes hypoxia leading to axonal degeneration.

In 1984, Botton *et al* reported five cases who were critically ill and had difficulty in weaning from mechanical ventilation. These patients had sensory motor weakness and electrophysiological studies revealed primary axonal neuropathy.

Neurological evaluation of such patient is often difficult because of ventilatory support and other equipment attached to the patient. The present case had difficulty in weaning from mechanical ventilation and developed hypercarbia when put on a T-piece trial inspite of fulfilling all other criteria for weaning.

From India two case report of CIP found. One in a patient with asthma and the other in a patient with renal failure. Primary axonal degeneration has been reported in 70% of critical ill patients and sepsis & multiorgan failure of which 30% had weakness of clinical examination.

Electrophysiological studies were consistent with predominantly motor axonal polyneuropathy and were very helpful in confidently making the diagnosis of CIP. In one study of survivors of at least 28 days of ICU treatment, nearly all patients displayed electrophysiological evidence of chronic partial denervation 05 (five) years after ICU discharge.

Another prospective evaluation confirmed the association between multiorgan failure, sepsis and CIP. According to clinical and electrophysiologic testing Ninety four percent of septic patients diagnosed with CIP.

No specific therapies are available but most patients improve after a period of supportive care. The condition is reversible, hence intensive physiotherapy and extended rehabilitation shoud be continued until the neuropathy improves adequatly

REFERENCES:

1. G Skowronski Neuromuscular diseases in intensive care. Oh's Intensive Care Manual, 5th edition ;47;537-47.
2. G.C. Khilnani ,Ravi Bansal ,O.P. Malhotra How offen do we Diagnose It? Indian J.Chest Dis Allied Sei 2003; 45:209-213.
3. Nates JL,Cooper DJ Day B, Tuxen DV 1997. Acute weakness syndrowes in critically ill Patients – a reappraisal. Anaesth Intens Care. 25:502-13.
4. Sliwa JA 2000. Acute weakness syndromes in the critically ill patient. Arch Phys Med Rehabil. 81: 545-52.
5. Will NJ. Zochodne DW. Bolton CF *et al* 1991. Peripheral nerve function in sepsis and multiple organ failure, Chest: 99; 176-84.
6. Bolton CF, Gilbert JJ, Halin AF, Sibbald WJ 1984. Pollyneuropathy in critically ill patients. J, Neurol Neurosurg Psychiat. 47: 1223-31.
7. DR. John Griffiths. Critical illness poly-neuropathy and myopathy. Critical care UK Editor.
8. Bolton CF, Brown JD, Sibbad WJ 1983. The electrophysiologic investigation of respiratory paralysis in critically ill patients. Neurology; 33;2: 186.
9. Sandra L. Kane, Pharm D, Jaseph F, Dastan MS 2002. Clinical Outcomes of critical illness Polyneuropathy. Pharmacotherapy, 22; 373-9.
10. Visser LH 2006. Critical illness polyneuropathy and myopathy: clinical feature risk factors and prognosis. Eur J Neurology, 13(11): 1203-12.