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An Interesting case of Acute Polyneuropathy in the Context of Acute Pancreatitis

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ABSTRACT

Acute polyneuropathy in the critically ill is frequently encountered in the ICU setup, but it is rarely seen as a sequelae of acute pancreatitis. The clinical entity critical illness polyneuropathy occurs almost exclusively in patients in critical care units and has been characterised as a complication of sepsis and multiple organ failure. Moreover the need to distinguish it from Guillain barre syndrome is all the more challenging. The clinical features elicited by both the polyneuropathies frequently overlap making it to be a diagnostic challenge. To obtain the diagnosis as early as possible affects the treatment course as both of them have entirely different modes of treatment .Here we discuss a case of a 33 year old male who developed acute polyneuropathy in the context of acute pancreatitis.

Keywords: Critical illness neuropathy (CIP), Guillain barre‘ syndrome (GBS), acute pancreatitis.

INTRODUCTION

The term “Critical illness polyneuropathy” was introduced in 1984 by Bolton et al, who attributed characteristic axonal loss of motor and sensory fibres to the toxic effect of sepsis. Critical illness myopathy, which has been increasingly recognized from the 1990s, can be elicited from excessive dosages of intravenous corticosteroids. Increasing evidence in intensive care units has shown that critical illness polyneuropathy and myopathy frequently occur concomitantly[1].

Also when a patient develops axonal polyneuropathy in the presence of critical illness and sepsis differentiation between CIP and axonal GBS may be clinically difficult. In both, polyneuropathy runs a monophasic course, the onset being relatively acute but with subsequent improvement in most instances. The clinical features also are similar. Comprehensive studiesdone at necropsy and nerve biopsies of patients with CIP showed the presence of primary axonal degeneration of the motor and sensory fibers, mainly distally, with no evidence of inflammation therefore concluded that the two types of polyneuropathy most probably are separate entities[2].

CASE REPORT

A 33 year old male who was diagnosed with CKD-Stage 5 for 5 years on medical management presented with abdominal distension, pain abdomen and loose stools for 2 weeks and reduced urine output for 1 day. He is a known hypertensive for 15 years. He had grossly deranged renal functions (serum urea-312mg/dl, serum creatinine-24.5mg/dl) on presentation following which he was taken up for haemodialysis. Later he was diagnosed to have acute pancreatitis (serum amylase-857mg/dl, serum lipase-1293mg/dl) and sepsis. He was initiated on broad spectrum parentral antibiotics such as intravenous meropenem according to renal dosing as well as intravenous metronidazole.

But during the course of treatment he developed ascending muscle paralysis of limbs followed by respiratory failure over a period of 2 days. There was symmetrical muscle weakness (muscle power-grade-0), generalized hypotonia with are flexia. It was also associated with facial muscle paralysis Patient also had features of dysautonomia in the form of tachycardia alternating with bradycardia. He was mechanically ventilated due to respiratory failure. MRI Brain and cervical spine was normal. Nerve conduction studies showed inelicitable potentials from upper and lower limbs suggestive of advanced neuropathy. Electromyography could not be done due to technical reasons. CSF was within normal limits (total count-nil, sugar-51mgldl, protein-16mg/dl). Immunoserology for HIV, HBs Ag, HCV and syphilis was negative. Patient was initiated on plasmapheresis.

During treatment patient developed electrolyte disturbances, several hypoglycaemic episodes, seizures and septic shock. A nerve biopsy was planned but was not done as the patient expired.

DISCUSSION

CIP is a predominantly motor, axonal polyneuropathy occurring as a complication of the sepsis syndrome. In two prospective studies, it has occurred in 50–70% of patients with sepsis.

The clinical and electrophysiological criteria employed in various studies on CIP include limb weakness, failure to wean from ventilator and abnormalities in nerve conduction and needle EMG consistent with axonal motor and sensory neuropathy. Sepsis syndrome is defined as a syndrome characterized by sepsis with one or more signs of organ dysfunction, hypoperfusion, hypotension, metabolic acidosis, acute alteration in mental status, oliguria or adult respiratory distress syndrome[2].

In our case however there was no altered mental status and weakness was noticed during the course of illness. Another interesting point is the involvement of facial muscles which is not often seen in critical illness neuropathy. There was significant autonomic dysfunction which is common to both to both the entities. CSF studies wouldn't yield much in the early course of the disease as CSF proteins will rise only after 1 week of the onset of the weakness.

Reports on peripheral neuropathy following acute pancreatitis are rare. Gross et al reported four cases of acute or sub acute axonal neuropathy complicating pancreatitis and pseudocyst formation. In three cases, major pancreatic resection was undertaken and in the fourth case, repeated pseudocyst drainage. In addition to patients with pancreatitis they also described polyneuropathy complicating critical illness. These patients did not have any evidence of heavy metal poisoning, porphyria, viral infection orspecific vitamin deficiency. Many of these patients had received antibiotics of the amino glycoside group and other combinations including metronidazole. All these patients suffered sepsis at the time and the possibility of neuropathic bacterial toxins could not be excluded. Nutritional deficiency was considered in these patients as the depressed levels of blood albumin improved after the institution of total parenteralnutrition.[3].

Clinical manifestations of GBS can vary, and an extensive number of other disorders could cause similar features of acute neuromuscular paresis. The diagnosis of GBS can be difficult, particularly in patients with asymmetric weakness, in those with weakness initially only in the arms, in patients with rapidly progressive deterioration in pulmonary function with relative preservation of muscle force in the extremities, and in patients with prominent pain or autonomic dysfunction as the presenting symptom[4].

Diagnosis of typical GBS[5]

Clinical Features required for diagnosis

Progressive weakness in both arms and legs (might start with weakness only in the legs)
Areflexia (or decreased tendon reflexes)

Features that strongly support diagnosis

Progression of symptoms over days to 4 weeks
Relative symmetry of symptoms
Mild sensory symptoms or signs
Cranial nerve involvement, especially bilateral weakness of facial muscles
Autonomic dysfunction
Pain (often present)
High concentration of protein in CSF
Typical electrodiagnostic features

Features that should raise doubt about the diagnosis

Severe pulmonary dysfunction with limited limb weakness at onset
Severe sensory signs with limited weakness at onset
Bladder or bowel dysfunction at onset
Fever at onset
Sharp sensory level
Slow progression with limited weakness without respiratory involvement (consider subacute inflammatory demyelinating polyneuropathy or CIDP)
Marked persistent asymmetry of weakness
Persistent bladder or bowel dysfunction
Increased number of mononuclear cells in CSF (>50×106/L)
Polymorphonuclear cells in CSF

Critical illness polyneuropathy invariably occurs at the peak of critical illness and sepsis, but in Guillain-Barré syndrome there is a brief period of recovery after a relatively minor illness or inoculation. Except for differences in the predisposing causes, as Bolton et al reported, it is difficult to distinguish critical illness polyneuropathy from Guillain-Barré syndrome on purely clinical grounds.

In both, polyneuropathy runs a monophasic course, the onset being relatively acute but with subsequent improvement in most instances. The clinical features also are similar; evidence of muscle weakness in all four limbs, occasional involvement of facial muscles and frequent involvement of the muscles of respiration, the depression or absence of deep tendon reflexes, and some evidence of distal sensory impairment[4].

Critical illness myopathy and/or critical illness neuropathy are frequentand serious complications to intensive care that[6]:

- delay weaning from mechanical ventilation
- increase the length of stay at the intensive care unit
- compromise rehabilitation and may result in a lifelong loss of
- function and in a reduction in quality of life.

It seems reasonable to ensure maximal functional status for survivors ofintensive care unit stays by applying a multimodal therapeutic approach including:

- screening and early diagnosis is possible
- intensive insulin therapy
- minimal sedation
- early physiotherapy
- – electrical muscle stimulation.

15 critically ill polyneuropathy patients were compared with 16 Guillain-Barre syndrome patients observed in a study done by Bolton et al. The analysis showed that the two polyneuropathies are likely to be separate entities that can be distinguished in most instances by the predisposing illness, electrophysiological features and cerebrospinal fluid results.

CONCLUSION

This is an unusual case of acute advanced neuropathy consequent to pancreatitis. The diagnostic dilemaencounterd in these cases warrants attention as acute polyneuropathy is becomes an additional challenge along with the sepsis to treat. Even though early intervention was done, outcome was fatal due to multiple associated medical factors and partly due to intervention.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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