Severe Acute Pancreatitis Complicated with Peripheral Nerve Damage: A Case Report and Literature Review

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1. Abstract

Background
Severe acute pancreatitis (SAP) accounts for approximately 20% of acute pancreatitis cases. Due to the combination of persistent organ dysfunction, 10-20% of patients die from persistent organ failure and infection-related shock induced by systemic inflammatory response syndrome [1]. Systemic complications of acute pancreatitis can involve major organs such as the circulatory, respiratory, renal, hepatic, and central nervous systems. Although there is extensive research on systemic complications of severe acute pancreatitis, cases of peripheral nerve damage are relatively underreported and understudied. Peripheral nerve damage can lead to pain, sensory abnormalities, and motor dysfunction, affecting patient recovery and quality of life. This article aims to report a clinical case of severe acute pancreatitis complicated by peripheral nerve damage and to review the related literature, providing reference and insight for future research to promote further understanding of this complication.

1.1. Case Report: We present a case of a 33-year-old male with SAP complicated by peripheral nerve damage. The patient exhibited symptoms of abdominal distension, nausea, and vomiting. Subsequent hospitalization revealed acute respiratory failure, renal failure, and sepsis. During recovery, the patient experienced numbness and weakness in the limbs, diagnosed as multiple peripheral nerve damage through electromyography.

1.2. Conclusion: This case highlights the importance of recognizing peripheral nerve damage as a potential complication in SAP patients and suggests the need for further research in this area.

2. Introduction
Severe acute pancreatitis (SAP) is a critical condition that can lead to multiple organ dysfunction and significant complications. While systemic complications involving major organs are well-documented, peripheral nerve damage in SAP patients is rare and underreported. This report aims to present a unique case of SAP complicated by peripheral nerve damage, providing insights into diagnosis, management, and outcomes.

3. Case Report
The patient is a 33-year-old male admitted for “intermittent nausea and vomiting accompanied by loss of appetite for 5 days.” Five days before admission, after consuming spicy food, he developed significant abdominal distension, nausea, and vomiting, which progressively worsened. He was diagnosed with “acute pancreatitis (severe hyperlipidaemia type)” at a local hospital and was admitted to the ICU due to acute respiratory failure, renal failure, and sepsis. He underwent tracheal intubation, mechanical ventilation, continuous renal replacement therapy, and one session of plasma exchange, along with jejunal nutrition tube insertion for enteral nutrition. After 3 days of treatment, he was transferred to our hospital. The patient had no history of pancreatitis or neurological disorders and had been in good general health. Blood glucose moni-
toring half a month before the onset showed significant elevation, with a glycated haemoglobin level of 12.04%. On admission, due to sedation, abdominal tenderness and limb muscle tone examination were not feasible, and no pathological signs were elicited. He received organ function support, anticoagulation, and lipid-lowering treatments, with initial blood glucose at 23.5 mmol/L. During hospitalization, an insulin micro-pump maintained an average blood glucose level of approximately 12.0 mmol/L. After cessation of sedative drugs, the patient regained consciousness with a GCS score of 15. Five days into treatment, respiratory and renal functions improved, and he was extubated and transferred to the general ward. About 10 days post-onset, the patient reported difficulty lifting his left limb, numbness in the ring and little fingers, and weakness in gripping. Physical examination showed left upper limb muscle strength at grade 2, right upper limb at grade 3+, left lower limb at grade 3, and right lower limb at grade 4+ with a positive left Hoffmann sign and no other pathological signs. A cranial CT scan showed no abnormalities, and electromyography indicated multiple peripheral nerve damage, primarily axonal, with significant involvement of the left upper limb. Multidisciplinary consultation diagnosed peripheral nerve damage of unknown cause, and treatment with methylcobalamin and vitamin B1 injections, as well as oral multivitamins, was initiated. The patient improved and was discharged. Follow-up after six months showed the patient had no specific discomfort, with muscle strength in the left upper limb at grade 4+, right upper limb at grade 5, left lower limb at grade 4+, and right lower limb at grade 5.

4. Discussion
Acute pancreatitis is an acute abdomen characterized by pancreatic enzyme activation causing digestive effects on the pancreas itself and surrounding organs, leading to local inflammatory responses and possibly organ dysfunction [2]. Severe acute pancreatitis can lead to numerous systemic and local complications, including SIRS, multi-organ dysfunction syndrome (MODS), sepsis, and abdominal compartment syndrome, as well as local complications like pancreatic parenchymal and peripancreatic tissue necrosis, gastrointestinal bleeding, and adjacent intestinal necrosis [3]. Neurological complications of acute pancreatitis are relatively rare, such as pancreatic encephalopathy (PE), which presents with neuropsychiatric symptoms and signs such as delirium, disorientation, hallucinations, and even coma [4]. In 1988, Gross et al. [5]. Reported four cases of polyneuropathy complicating pancreatitis and pancreatic pseudocysts, treated with parenteral nutrition and various medications, all with severe sepsis. They questioned whether these neurological complications were caused by critical illness polyneuropathy or had a new connection to pancreatic disease. Vallat et al. in 1989 [6], summarized these reports and suggested that peripheral neuropathy might be caused by acute pancreatitis disease. The reported patient, a young male with multi-organ dysfunction, had clinical and auxiliary examination find-

ings consistent with the diagnosis of severe acute pancreatitis [7]. During the course of the disease, he developed limb numbness and weakness, especially in the distal parts of the upper limbs and the inner two fingers. Electromyography indicated significant involvement of motor and sensory nerves, primarily axonal damage, suggesting multiple peripheral nerve damage. However, the patient had no underlying neurological disease, and the cause remained unclear. Long-term treatment with neurotrophic drugs gradually alleviated his symptoms. The peripheral nervous system, responsible for transmitting nerve signals and controlling sensory and motor functions, is an essential part of the human nervous system. Peripheral neuropathy includes a wide range of diseases affecting the peripheral nervous system in different patterns, with distal sensory polyneuropathy (DSP) being the most common. DSP includes a group of diseases characterized by length-dependent peripheral nerve damage, leading to distal sensory loss and pain, and, in severe cases, gait instability and fall risk, with potential foot ulcers and even the need for amputation. DSP is one of the most common neurological disorders, with increasing prevalence due to an aging population and rising diabetes and obesity rates. Diabetes is the most common cause of DSP, accounting for 50% of cases. More than half of diabetic patients are affected by DSP, and due to pain, gait instability, and associated depression, DSP is a major cause of reduced quality of life. The second most common cause is idiopathic or cryptogenic polyneuropathy, accounting for about 40% of cases, often associated with prediabetes and metabolic syndrome [8, 9]. Experimental studies suggest that the pathogenesis of diabetic DSP is multifactorial, but the exact causes remain unclear. One prevailing theory is that oxidative stress and inflammatory stress may damage nerve cells in metabolic dysfunction [10]. Recent research confirms that exocrine dysfunction in acute pancreatitis can lead to endocrine insufficiency and eventually diabetes [11, 12]. Therefore, poor blood glucose control in this patient might have resulted in DSP. Intensive care unit-acquired weakness (ICUAW) can be caused by critical illness polyneuropathy (CIP), critical illness myopathy (CIM), or a combination of both [13]. CIP is a neurological complication in critically ill patients, characterized by distal axonal sensorimotor polyneuropathy affecting limbs and respiratory muscles, usually symmetrically; mild cases show more pronounced distal muscle weakness, while severe cases mainly involve the lower limbs. Guidelines [14] suggest using the Medical Research Council (MRC) sum score for bedside muscle strength assessment, with a total score <48 diagnosing CIP. Additional electrophysiological testing is required only if clinical presentation is atypical or there are focal deficits. CIP and CIM are difficult to distinguish and can occur singly or together. CIP and CIM cause muscle weakness and difficulty in weaning from mechanical ventilation, often complications of sepsis and MODS [15, 16]. Previous literature identifies sepsis, SIRS, and MODS as key factors [13], and severe acute pancreatitis often leads to these
comlications. CIP’s pathological manifestation is axonal degeneration [17], with its mechanism involving microvascular changes in the endoneurium due to sepsis [18]. Hyperglycemia is also an independent risk factor for this condition [19]. ICUAW currently lacks effective treatment, but hyperglycemia prevention, delaying parenteral nutrition until after the first ICU week, and minimizing sedation can partially prevent ICU-acquired weakness [20]. Guillain-Barré syndrome (GBS) is an immune-mediated polyneuropathy that often follows an infectious disease [21], presenting with acute onset of neurological symptoms and progressive limb weakness lasting up to 4 weeks before reaching a plateau. The exact pathogenesis is unknown, with infections being the most common trigger, alongside rare causes such as surgery, vaccination, transplantation, and trauma [22]. GBS typically presents with symmetrical limb and bulbar muscle weakness, and severe cases involve respiratory muscle weakness, reduced or absent tendon reflexes, and can include sensory abnormalities and autonomic dysfunction, with a self-limiting course [23-25]. Electrophysiological testing often shows peripheral nerve demyelination. Once diagnosed, high-dose intravenous immunoglobulin (IVIG) and plasma exchange are recognized treatments to shorten the course and reduce the severity of GBS [26]. GBS might be a rare complication of severe acute pancreatitis, but the mechanism is unclear and lacks related literature.

This patient had abnormal blood glucose, hyperlipidaemia, and SIRS and MODS caused by severe acute pancreatitis, potentially inducing ICUAW and other neuropathies like GBS. Despite comprehensive neurological examinations, the exact cause of the patient’s neurological damage remained unclear. Nutrotrophic treatment with methylcobalamin and vitamin B1, alongside multivitamins, showed effectiveness, as symptoms and muscle strength gradually returned to normal, suggesting a viable treatment approach. Additionally, controlling systemic complications significantly impacts the improvement of peripheral neuropathy.

5. Conclusion

In conclusion, severe acute pancreatitis (SAP) can lead to significant systemic and local complications, including rare but impactful peripheral nerve damage. This case highlights the necessity of considering peripheral nerve damage in the differential diagnosis of SAP complications. Early recognition and appropriate management of such neurological complications are crucial for improving patient outcomes. The successful recovery of the patient in this case through nutrotrophic treatment emphasizes the potential effectiveness of this approach. Further research is essential to deepen our understanding of the pathophysiology of SAP-related peripheral nerve damage and to develop optimized treatment strategies.

6. Funding Sources


References

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