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Case Report

Fatal Wernicke's Encephalopathy Following Severe Acute Pancreatitis: A Rare Case Report

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Abstract

Background: Severe acute pancreatitis is associated with significant morbidity and mortality, often due to systemic complications. Nutritional deficiencies are frequent in this setting, yet neurological complications such as Wernicke's encephalopathy (WE) remain exceedingly rare. We are a neuropsychiatric syndrome caused by thiamine deficiency, classically linked to alcohol misuse but increasingly recognized in non-alcoholic patients with prolonged fasting, vomiting, or parenteral nutrition. Early recognition and treatment are crucial, as delayed diagnosis is associated with poor outcomes. ***Case Presentation:*** We report the case of a 54-year-old woman admitted with severe acute pancreatitis complicated by multiorgan failure. One month later, she developed progressive confusion, ocular abnormalities, and impaired coordination, raising suspicion of Wernicke's encephalopathy. Neuroimaging supported the diagnosis. Despite supportive management and initiation of thiamine supplementation, the patient's condition deteriorated, ultimately leading to death. ***Conclusion:*** This case illustrates the diagnostic challenges and devastating consequences of Wernicke's encephalopathy complicating severe acute pancreatitis. Clinicians should maintain a high index of suspicion for this condition in critically ill patients with risk factors for thiamine deficiency. Early empiric thiamine supplementation is a safe and cost-effective preventive strategy that may significantly improve outcomes.

Keywords: Severe acute pancreatitis; Wernicke's encephalopathy; Thiamine deficiency; Neurological complications.

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INTRODUCTION

Severe acute pancreatitis is a potentially life-threatening condition characterized by systemic inflammation, multiorgan failure, and high mortality rates [1]. Patients with severe pancreatitis are prone to metabolic and nutritional complications due to prolonged fasting, vomiting, increased catabolism, and the frequent use of parenteral nutrition [2]. One of the rare but serious consequences of nutritional deficiency in this context is Wernicke's encephalopathy (WE), a neurological emergency caused by thiamine deficiency. Although WE is classically described in alcohol-dependent patients, it has been increasingly recognized in non-alcoholic individuals with risk factors such as hyperemesis gravidarum, malignancy, bariatric surgery, and prolonged parenteral feeding [3]. Untreated or late-treated cases are associated with irreversible neurological deficits or death. Early recognition and prompt thiamine supplementation are therefore essential. Reports of WE complicating severe pancreatitis remain scarce, and outcomes are often unfavorable due to diagnostic delays [4]. Here, we present the case of a patient with severe acute pancreatitis who developed WE during hospitalization, with a fatal outcome. This case

aims to increase awareness of this rare but preventable complication.

CASE PRESENTATION

A 54-year-old woman, with no significant past medical history, presented to the emergency department with a two-day history of epigastric pain. On examination, she had localized abdominal tenderness in the epigastric region without peritoneal signs. Laboratory investigations revealed an inflammatory syndrome with a C-reactive protein (CRP) level of 108 mg/L, elevated liver enzymes indicating cytolysis, and a markedly elevated lipase of 1090 U/L. An abdominal computed tomography (CT) scan confirmed the diagnosis of severe acute pancreatitis, classified as stage E. The patient was admitted to the intensive care unit (ICU), where she received aggressive fluid resuscitation, correction of electrolyte imbalances, multimodal analgesia, and parenteral nutrition. After stabilization, she was transferred to the gastroenterology department for further management and subsequently discharged after clinical improvement. One month later, the patient was readmitted to the emergency department with acute-onset altered mental status with ocular findings. A brain CT scan showed no abnormalities. However, further evaluation with magnetic resonance imaging (MRI) of the brain revealed findings consistent with Wernicke's encephalopathy (Figure 1, 2). Thiamine supplementation was initiated promptly. Despite treatment, the patient's neurological status continued to deteriorate, and her clinical course was unfavorable, leading to death.

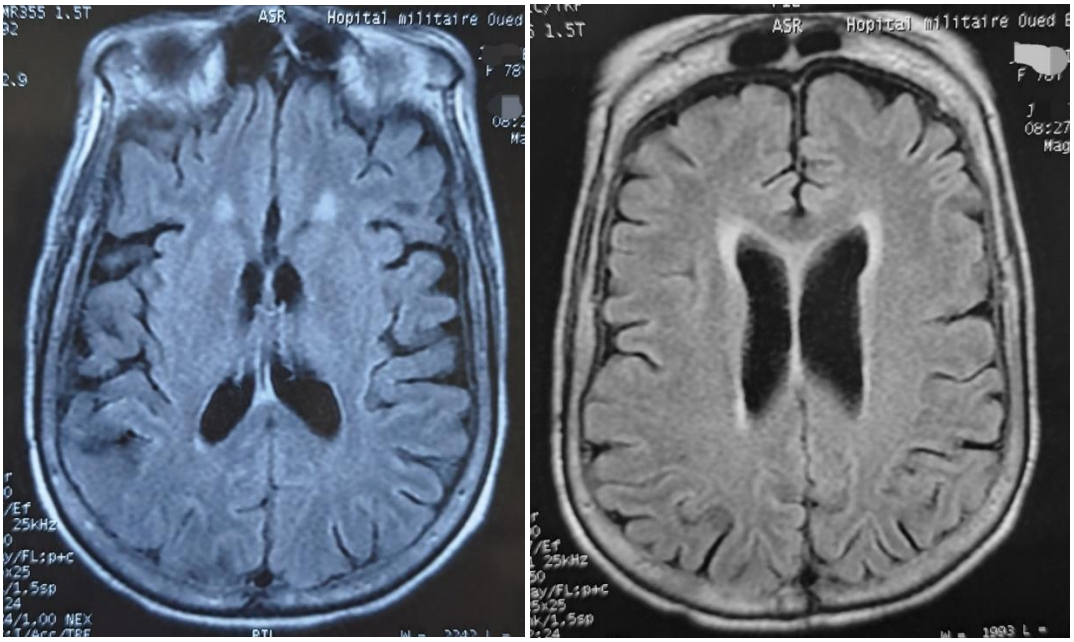

Figure 1 and 2: FLAIR axial shows hyperintensity of the periaqueductal grey

DISCUSSION

Wernicke's encephalopathy (WE) is an acute neuropsychiatric disorder caused by thiamine deficiency, classically associated with chronic alcoholism but increasingly recognized in non-alcoholic settings such as malnutrition, prolonged vomiting, or hyperemesis [3]. Its occurrence during severe acute pancreatitis is particularly rare. Patients with pancreatitis are at risk due to prolonged fasting, increased metabolic demands, and parenteral nutrition without adequate vitamin supplementation [4]. This unusual association highlights the importance of considering nutritional complications in critically ill patients with pancreatitis.

The diagnosis of WE is often difficult in intensive care units, where altered mental status may be attributed to metabolic encephalopathy, sepsis, or hypoxemia. The classical triad of confusion, ophthalmoplegia, and ataxia is observed in only a minority of cases [5]. In our patient, the presence of neuropsychiatric deterioration along with ocular findings suggested WE. Magnetic resonance imaging is considered the most useful diagnostic tool, often showing bilateral symmetric hyperintensities in the thalamus, mammillary bodies, or periaqueductal gray matter [6]. However, a normal MRI does not exclude the diagnosis, and WE remains primarily a clinical diagnosis requiring urgent management.

Thiamine supplementation represents the cornerstone of treatment, with proven efficacy when administered promptly. Delayed recognition and initiation of therapy, as in our case, are strongly associated with irreversible neurological sequelae and increased mortality [3,7]. Preventive administration of thiamine in high-risk patients—particularly those on prolonged fasting, receiving parenteral nutrition, or experiencing recurrent vomiting—should be systematically implemented [8]. This practice is simple, safe, and potentially lifesaving.

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This case underscores several important clinical lessons. First, Wernicke's encephalopathy should be considered a potential but under-recognized complication of severe pancreatitis. Second, clinicians should adopt a low threshold for empiric thiamine supplementation in critically ill patients, especially in the presence of risk factors for deficiency. Finally, reporting this case raises awareness regarding the devastating consequences of delayed recognition, reinforcing the need for vigilance and preventive strategies.

CONCLUSION

This case highlights the importance of considering Wernicke's encephalopathy as a rare but serious complication in patients with severe acute pancreatitis. Malnutrition, prolonged fasting, and parenteral nutrition without vitamin supplementation are key predisposing factors. Given the diagnostic challenges and high risk of morbidity and mortality, clinicians should maintain a high index of suspicion when neurological symptoms develop in such patients. Early empiric thiamine supplementation is a simple, safe, and effective measure that can prevent devastating outcomes.

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