Clinical Case Report

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Fatal cerebral hemorrhage associated with acute pancreatitis

A case report

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Abstract

Rationale: Pancreatic encephalopathy (PE) refers to the abnormalities in mental status that complicate acute pancreatitis (AP). We report the case of a patient who developed AP that was complicated by PE and followed by fatal cerebral hemorrhage.

Patient concerns: A 41-year-old male patient with AP that initially manifested with PE and was subsequently complicated by fatal cerebral hemorrhage.

Diagnoses: A head computed tomography (CT) scan showed a fatal intracerebral hemorrhage located in the occipital lobe, and an abdominal CT scan presented a large amount of peripancreatic fluid collections.

Interventions: The patient received a hematoma evacuation. The volume of the hematoma was approximately 15 mL. A consequent open pancreatic necrosectomy was performed to remove all necrotic tissues and to drain the peripancreatic fluid collections.

Outcomes: The patient could perform his normal daily activities efficiently, and no abnormality was observed in the physical examination 3 months after his discharge.

Lessons: Although uncommon, PE should be properly monitored. Once the neurological symptoms of a patient dramatically worsen within a short time, the possibility of fatal cerebral hemorrhage should be considered.

Abbreviations: AP = acute pancreatitis, CT = computed tomography, PE = pancreatic encephalopathy, SAP = severe acute pancreatitis.

Keywords: acute pancreatitis, cerebral hemorrhage, pancreatic encephalopathy

1. Introduction

Acute pancreatitis (AP) is a common and occasionally fatal gastrointestinal disorder that requires hospitalization. A major complication, following early organ failure, is the secondary infection of pancreatic or peripancreatic necrotic tissues, which leads to sepsis and multiple organ failure. Pancreatic encephalopathy (PE), which generally occurs during the early stage of severe AP (SAP), is an uncommon complication with a high mortality rate.[1] PE refers to abnormalities in mental status, such as spatial disorientation, trance, agitation with delusion, and hallucination, in patients with AP.[2] To our knowledge, this case of AP that initially manifested with PE and was subsequently complicated by fatal cerebral hemorrhage in a healthy 41-year-old male is the first to be reported in the literature.

2. Case report

A 41-year-old male was admitted to the emergency department of our hospital with symptoms of acute epigastric pain, nausea, vomiting, and fever, which started approximately 15 hours before admission and were gradually worsening. The patient did not report any similar symptoms in the past or any other medical history, such as hypertension or diabetes, before admission. His vital signs were as follows: heart rate = 101/min, blood pressure = 132/78 mm Hg, body temperature = 38.3°C, and respiratory rate = 20/min. Distension and tenderness in the upper abdomen were observed during physical examination. Laboratory tests showed a white blood cell count of 20,500/mL and an amylase count of 720 IU/L. Computed tomography (CT) scan performed upon admission indicated intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat.

The aforementioned clinical and laboratory data indicated a diagnosis of AP, and the patient received conservative treatments, including antibiotics, protease inhibitors, and parental nutrition. However, the symptoms of the patient were not relieved, and his arterial blood oxygen tension ranged from 50 to 60 mm Hg, despite a fraction of inspired oxygen of 0.30. Thus, the patient was treated in the intensive care unit 2 days after his admission. Symptoms of trance and hypersomnia began 3 days after admission, and a neurological examination was requested. The

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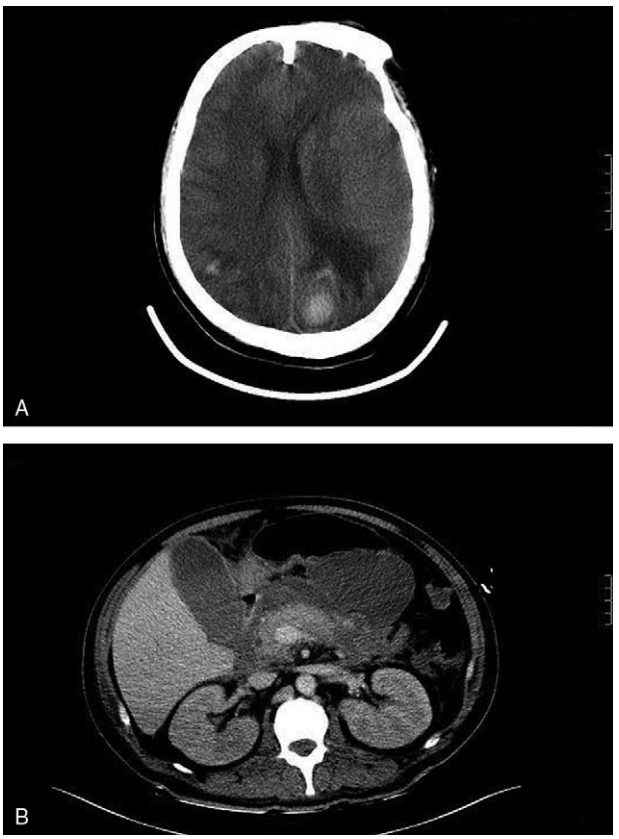

Figure 1. (A) Computed tomography (CT) scan. Intracerebral hemorrhage was located in the occipital lobe. (B) Contrast-enhanced computed tomography (CT) scan. Large amount of peripancreatic fluid collections were found. CT = computed tomography.

patient was conscious during the examination, but he was only partly cooperative.

The body temperature of the patient remained higher than 38.0°C, and a blood culture showed an *Enterobacter aerogenes* infection. The aforementioned manifestations persisted for 2 weeks until the patient suddenly sank into a coma. Neurological examination showed that light reflex on both sides had disappeared, and the patient was incapable of responding to any stimuli. A head CT scan was immediately performed. The result showed a fatal intracerebral hemorrhage that was located in the occipital lobe and that the midline shifted toward the right side (Fig. 1A). Blood pressure and the results of coagulation tests were normal.

Consequently, the patient received a hematoma evacuation. The volume of the hematoma was approximately 15 mL. Four weeks after admission, the patient gradually recovered from the coma and started to exhibit certain automatic activities. Nevertheless, an abdominal CT scan showed a large amount of peripancreatic fluid collections (Fig. 1B). Accordingly, an open pancreatic necrosectomy was performed to remove all necrotic tissues and to drain the peripancreatic fluid collections.

The patient was discharged 2 months after admission in good condition, with normal biochemical parameters and body temperature. An outpatient follow-up examination was performed 3 months later. The patient could perform his normal daily activities efficiently, and no abnormal finding was found in the physical examination. The report was conducted in accordance with the principles of the Declaration of Helsinki. All patients or their legal representatives provided written informed consent. The ethics review board of West China Hospital approved the study.

3. Discussion

Central nervous system symptoms that accompany SAP during its early stages are described as PE.[3] Patients with SAP that is complicated by PE face high risks of death. Cerebrospinal fluid examination in patients with PE shows no abnormal result, and magnetic resonance imaging only presents abnormal white matter signals at various levels.[4]

PE is related to phospholipase A2 (PLA2) activation.[3] PLA2 damages the structure of brain cell membrane and increases vascular permeability, which lead to brain edema. Excessive generation of cytokines during the pathologic course of AP always causes organic injury. The generation of the platelet-activating factor promotes platelet aggregation and release, which induce cerebral capillary thrombosis and hypoxemia, thereby causing metabolic disturbance of the brain cells and worsening cerebral edema. A PLA2 inhibitor and low-molecular-weight heparin can alleviate brain injury in several experiments.[5] Guardia et al[6] presented the case of a patient who developed progressive hypoxemia and multiple system failure during the course of AP; autopsy showed fat embolisms in the lungs, kidneys, and heart, and multiple petechial hemorrhages in the brain. However, this patient did not develop fatal cerebral hemorrhage.

The present case showed a special aspect of PE. To our knowledge, this case is the first clinical pathological report of PE that involved fatal cerebral hemorrhage. As previously described, the neurological symptoms of the patient turned from relatively mild during the early stage to severe within a short time. The patient neither had a history of hypertension nor suffered from disseminated intravascular coagulation during the entire course. We believe that the vascular injury of the central nervous system is the direct reason for the cerebral hemorrhage. However, the mechanism of the severe injury remains unclear.

In conclusion, although PE is a rare condition, considerable attention should be directed toward patients with PE, particularly when their neurological symptoms dramatically worsen within a short time. Radiological tests are necessary, and surgical interventions should be prepared for such patients to prevent fatal outcomes.

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