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CASE REPORT

Acute Ischemic Pancreatitis Following Cardiac Arrest: A Case Report

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ABSTRACT

Context Ischemia is an established cause of acute pancreatitis; however, acute pancreatitis has never been reported after cardiac arrest. **Case report** We report a case of acute pancreatitis following cardiac arrest with prolonged cardiopulmonary resuscitation in a 58-year-old man, the mechanism of which is likely to be ischemic. The patient developed severe ischemic encephalopathy, leading to death. Possible causes of acute pancreatitis in a context of cardiopulmonary resuscitation are discussed. **Conclusion** In case of abdominal distension following cardiac arrest, diagnoses of mesenteric ischemia and acute ischemic pancreatitis should be considered. Such digestive complications occurring after cardiac arrest probably reflect the severity of the ischemia.

INTRODUCTION

Cardiac arrest is frequently associated with digestive complications. In a study of 130 patients admitted to intensive care unit (ICU) for cardiac arrest and survivors at 48 hours, l'Her *et al*. found that 60% had clinical signs of gut dysfunction, the two most frequent being diarrhea and enteral feeding intolerance [1]. The digestive tract is considered to be the motor of multiple organ failure occurring after cardiac arrest, via increased intestinal permeability and bacterial translocation [2]. Whereas ischemia is known to be a possible cause of acute pancreatitis [3], we report the first case of acute pancreatitis following cardiac arrest, the mechanism of which is likely to be ischemic.

CASE REPORT

A 58-year-old man with a medical history of dilated cardiomyopathy diagnosed in 2001, with a left ventricular ejection fraction of 35%, was admitted to our ICU in January 2010 following cardiac arrest. In the morning, he had gone hunting, and did not have any symptoms. After returning home, while he was seated and without any prodrome, he presented lost consciousness and fell on the floor. There was no sign of breathing and no cardiac activity. Cardiopulmonary resuscitation was immediately started by his son-in-law, a professional fireman. At minute 20 after cardiac arrest, a professional team of firemen arrived and a semi-automatic cardiac defibrillator delivered 4 electric shocks. At minute 50, an emergency medical team arrived; electrocardiogram revealed asystolia, and intravenous adrenaline (epinephrine) was administered. A return to spontaneous cardiac activity was obtained at minute 80. Cardiopulmonary resuscitation was never interrupted during these 80 minutes, and no mydriasis was noted. An electrocardiogram showed abnormalities of the ST segment with suspicion of posterior myocardial infarct. The patient was transferred to our University Hospital. Coronarography showed no sign of arterial obstruction, and the left ventricular ejection fraction was 50%. An intra-aortic balloon pump was begun. The patient was then admitted to our ICU, with a presumed diagnosis of cardiac arrest secondary to ventricular arrhythmia, complicating dilated cardiomyopathy. The Organ Dysfunction and/or Infection (ODIN) score [4] at admission was 4 and the Simplified Acute Physiology Score (SAPS II) score [5] was 76 on the first day of admission, reflecting the poor prognosis of the patient. Hemodynamic status was initially stable under adrenaline infusion and intra-aortic balloon pump. As there was severe hypoxia with signs of aspiration pneumonia, antibiotic therapy directed against gram-negative and anaerobic bacteria was started. Abdominal volume was increased, with tension, but without diarrhea or digestive bleeding (Figure 1). Biologically, the lipase concentration was 866 IU/L (reference range: 0-60 IU/L). The arterial lactate concentration measured after the prolonged cardiopulmonary resuscitation was 10 mmol/L (reference range: 0.3-2.5 mmol/L); it rapidly decreased to 2.5 mmol/L 24 hours after admission to the ICU. During the ICU stay, the arterial lactate concentration

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Abbreviations ICU: intensive care unit

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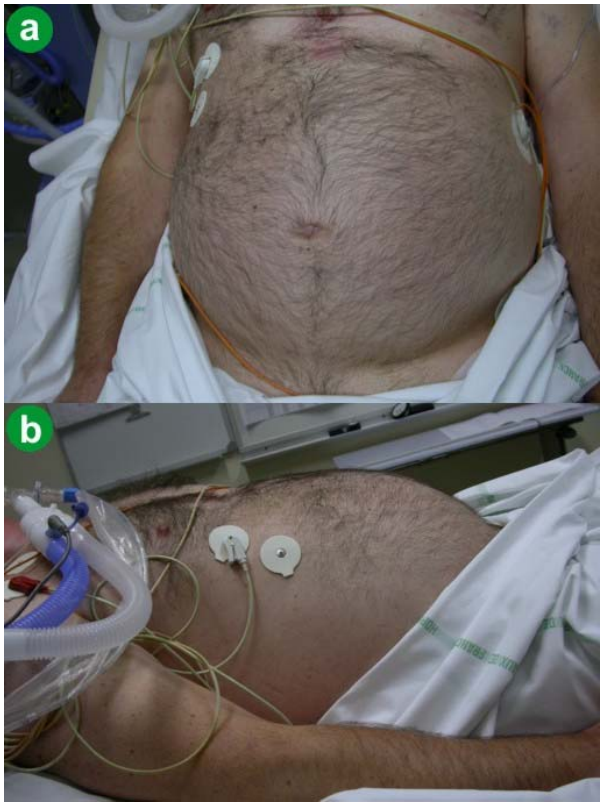

Figure 1. Increased abdominal volume observed in the first hours following cardiac arrest.

remained normal. The patient was oliguric, with acute renal failure and metabolic acidosis; therefore, continuous veno-venous hemodiafiltration was started. Therapeutic hypothermia using an external cooling device to a target temperature of 33°C was carried out during the first 24 hours. In the following days, the intra-aortic balloon pump and adrenaline were stopped, hypoxia rapidly improved, and renal function normalized. However, abdominal examination did not improve, and there was no sign of awakening despite the cessation of sedation. An abdominal CT scan confirmed the diagnosis of acute pancreatitis with a CT severity index of 4 (Figure 2), and a cerebral CT scan revealed diffuse hypodensity with severe ischemic brain edema. After eight days in the ICU, in the absence of awakening, a therapeutic limitation was decided, leading to death.

DISCUSSION

We report a case of acute pancreatitis following cardiac arrest, the mechanism of which was likely to be ischemic.

Acute pancreatitis has exceptionally been described as a precipitating cause of cardiac arrest [6]. In the present case, the patient did not complain of digestive symptoms before the cardiac arrest, and there was no clinical evidence for an alcoholic or biliary primary cause of acute pancreatitis. In the first hours following cardiopulmonary resuscitation, abdominal volume rapidly increased, with an early rise in lipase concentration. Therefore, acute pancreatitis is likely to be a consequence of the cardiac arrest, and not the cause. Various mechanisms of ischemia, such as shock, mesenteric macrovessel occlusion, cardiovascular surgery and thrombotic microangiopathy have been associated with the development of acute pancreatitis [3, 7, 8, 9, 10, 11]. To our knowledge, this is the first report of acute pancreatitis following cardiac arrest. Cardiopulmonary resuscitation was particularly prolonged, 80 minutes being longer than usually recommended [12]. However, the medical team arrived 50 minutes after the onset of cardiopulmonary resuscitation. In addition, the combination of witnessed cardiac arrest, and the absence of both a no-flow period and mydriasis were in favor of proceeding with cardiopulmonary resuscitation. Since cardiac arrest is the end-stage of circulatory failure, it is logical that it can induce ischemic acute pancreatitis.

Other causes of acute pancreatitis should be discussed. First, intra-aortic balloon pump placement has been associated with the development of acute pancreatitis [13]. However, acute abdomen was observed before its introduction. Second, post-traumatic acute pancreatitis could have occurred following prolonged cardio-pulmonary resuscitation. However, no traumatic injury, such as intra-abdominal hemorrhage, was noted on abdominal CT scan. In addition, the patient fell on the floor while he was seated, without direct abdominal injury. Even if trauma is a possible cause of acute pancreatitis, in our opinion, the trauma associated with this fall was not strong enough to be responsible for

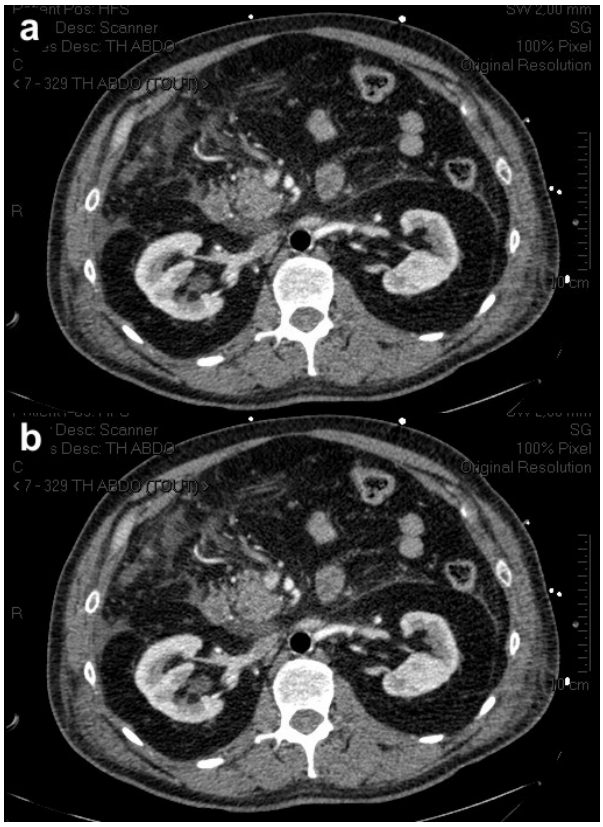

Figure 2. Abdominal CT scan showing acute pancreatitis with a CT severity index of four.

acute pancreatitis. Third, hypothermia is a possible cause of acute pancreatitis [14]. Indeed, at admission to the ICU, his body temperature was 34°C. However, the link between acute pancreatitis and hypothermia is still under debate [15]. Acute pancreatitis could complicate severe hypothermia whereas mild hypothermia (32°C) may have a beneficial effect on acute pancreatitis [16].

Acute abdomen following cardiac arrest is usually associated with the diagnosis of mesenteric ischemia [1, 2]. Gaussorgues *et al*. reported early fetid diarrhea with positive blood culture in 36% (12 out of 33) of patients admitted to the ICU following an out-of-hospital cardiac arrest [2]. Interestingly, in the present case, digestive signs were limited to increased abdominal volume with tension, without diarrhea or digestive bleeding, and the initial blood cultures were negative. The diagnosis of acute pancreatitis was made on the association of abdominal distension with increased lipase concentration, and confirmed by an abdominal CT scan. To date, there is no consensus for recommending a systematic dosage of plasma lipase concentration after cardiac arrest. In our opinion, plasma lipase concentration should be measured after cardiac arrest in patients presenting with digestive abnormalities, such as increased abdominal volume, ileus or feeding intolerance. In case of increased lipase concentration in such a context, an abdominal CT scan should be carried out in order to evaluate the CT severity index. In patients with normal gastric residual volumes, early enteral nutrition should be administered whatever the lipase concentration is.

Acute abdomen following cardiac arrest usually indicates a poor prognosis. Gaussorgues *et al*. found that all bacteriemic patients presenting with fetid diarrhea after cardiac arrest died (12 out of 12) [2]. Multiple organ failure following cardiac arrest could be secondary to small bowel ischemia, leading to bacterial translocation [17]. In the present case, the patient did not die of the initial multiple organ failures. He developed severe ischemic encephalopathy leading to a limitation of active therapies. Plasma lipase concentration measured after cardiopulmonary resuscitation might have a prognostic value. Indeed, Malinoski *et al*. have recently found that elevated serum amylase and lipase values observed among patients with hemorrhagic shock were associated with organ failure and mortality [9]. This suggests that occurrence of acute pancreatitis in a context of shock may well be a prognostic marker, and may reflect the severity of the ischemia. Therefore, both mesenteric ischemia and acute ischemic pancreatitis following cardiac arrest probably complicate prolonged cardiopulmonary resuscitation, and could be a marker of severe ischemia. Future studies should clearly evaluate the prognostic value of plasma lipase concentration after cardiopulmonary resuscitation.

In conclusion, pointing out the information that there is a theoretical risk of acute ischemic pancreatitis after cardiopulmonary resuscitation may help clinicians to think about this complication. In the case of abdominal distension following cardiac arrest, diagnoses of mesenteric ischemia and acute pancreatitis should be considered. Mesenteric ischemia should be envisaged in case of early diarrhea, and acute pancreatitis in case of isolated increased abdominal volume. Both complications after cardiac arrest probably reflect the overall severity of ischemia, and are markers of a poor prognosis.

Conflict of interest None

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