CASE REPORT

Ischemic Acute Necrotizing Pancreatitis in a Marathon Runner

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ABSTRACT

Context Acute pancreatitis due to pancreatic ischemia is a rare condition. **Case report** In this case report we describe a 57-year-old male who developed an acute necrotizing pancreatitis after running a marathon and visiting a sauna the same evening, with an inadequate fluid and food consumption during both events. **Conclusions** Pancreatic ischemia imposed by mechanical and physical stress and dehydration can induce the development of acute pancreatitis. Separately, these factors are rare causes of ischemic acute pancreatitis. But when combined, as in this particular case, the risk of an acute necrotizing pancreatitis cannot be neglected.

INTRODUCTION

Acute pancreatitis due to vigorous exercise is a rare phenomenon and has been scarcely reported in the literature.

In this case report we describe the case of a 57-year-old man who developed acute necrotizing pancreatitis after running a marathon and visiting a sauna, in conjunction with inadequate fluid and food consumption.

CASE REPORT

A 57-year-old man from the United Kingdom was referred to the hospital due to acute epigastric pain, nausea, severe dehydration and a dry mouth, the morning after running a marathon. Although he was a trained marathon runner his fluid and food intake were inadequate. After finishing the marathon he went to a sauna where his fluid intake also was insufficient. It has to be noted that he rehydrated after both events. The patient was not familiar with gallstones or diabetes mellitus, and no history of alcohol or drug abuse was stated.

Physical examination resulted in an expanded, hypertympanic and tender abdomen with lively peristalsis. He had a blood pressure of 165/100 mmHg, a heart rate of 81 beats per minute and a respiratory rate of 24 min^{-1}. No further peculiarities were found during the examination.

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Laboratory examination revealed an elevated glucose 17.5 mmol/L (reference range: 4.0-6.1 mmol/L), amylase 431 U/L (reference range: 0-99 U/L), AST 91 U/L (reference range: 0-36 U/L), LDH 1,080 U/L (reference range: 0-449 U/L), CPK 1,469 U/L (reference range: 0-199 U/L), CRP 244 mg/L (reference range: 0-9 mg/L) and lipase 1,264 U/L (reference range: 0-126 U/L). Total bilirubin was normal. Transabdominal ultrasonography revealed free intra-abdominal fluid containing blood and an amylase concentration of 944 U/L. Contrast enhanced CT scan showed a massive loss of functioning pancreatic tissue (more than 90%). No pancreatic duct dilatation or gallstones were seen. Treatment consisted of nasojejunal feeding, intravenous saline, analgesics and intravenous insulin to regulate blood glucose levels.

His disease course went uncomplicated with the patient showing steady progression in his recovery. Glucose levels remained moderately steady with minor fluctuations over time. A CT scan performed 10 days after admission revealed necrotic pancreatic tissue and peripancreatic fluid collections (Figure 1).

Two weeks after admission a normal diet was sufficient to sustain an adequate calorie intake. Intravenous saline was suspended in the third week.

Our patient was discharged after 30 days of hospital admission and returned to the United Kingdom. No pancreatic enzyme replacement therapy was issued.

Six months after discharge from out clinic, glucose levels were still stable within normal limits using a daily dosage of twelve units of insulin aspart/insulin aspart protamine.

DISCUSSION

In our case report we describe a patient with an acute onset of pancreatitis after strenuous exercise and

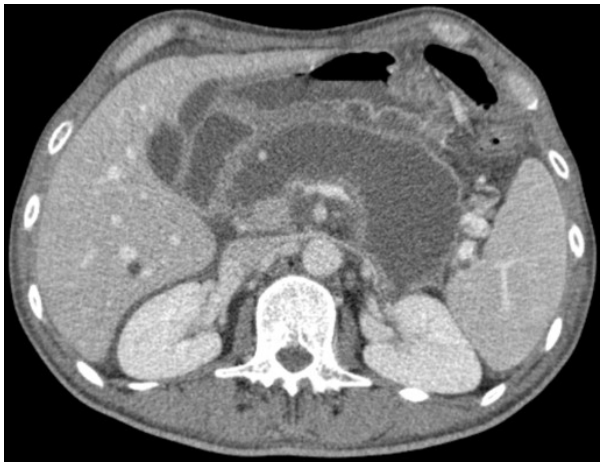

Figure 1. CT-scan 10 days after admission: Necrotic pancreatic tissue and peripancreatic fluid collections.

excessive heat combined with an inadequate fluid and food intake during both events.

Physical stress may be related to ischemic pancreatitis [1]. Systemic stress, in this case strenuous exercise, causes the majority of the blood flow to divert to the muscles in greater need of oxygen. The other parts of the body, including the visceral organs, receive less blood flow, which could lead to ischemia [2]. Furthermore, Takano *et al*. [3] showed in rats that systemic stress causes direct damage to the pancreas and worsens pancreatitis. However, no literature has linked the direct effect of physical stress to acute pancreatitis in humans.

Dehydration possibly played a role in the development of ischemic pancreatitis [4]. The prolonged physical exercise and the extreme heat of a sauna caused our patient to be severely dehydrated, thus leading to hypovolemia and finally ischemia.

The pancreas is known to be susceptible to ischemia and there is general consensus that it plays a pivotal role in the development of acute pancreatitis [5, 6, 7, 8]. Hypoperfusion of the pancreatic tissue results in various pathological mechanisms. Cell necrosis and leukocyte infiltration occur due to hypo-oxygenation and endothelial dysfunction respectively. The endothelial wall also becomes fenestrated and more permeable for active proteases, because of the release of active pancreatic enzymes. Furthermore, O_2 radicals are formed by leukocyte activation, attracting more leukocytes to the pancreas, thus exacerbating the process of acute pancreatitis [8]. Although the patient rehydrated after the marathon and sauna visit, the hypovolemia during the events already damaged the pancreatic tissue. Reperfusion alone does not reverse the necrotic process and even worsens the pancreatitis by inducing the formation of O_2 radicals [7]. With the accompanying diminished blood flow because of the systemic stress, the chances of acute pancreatitis may have been increased.

Mechanical trauma has also been advocated as a possible cause for acute pancreatitis. Due to during long-distance running, repetitive injury of the pancreas may have been possible against the posterior abdominal wall and spine [9].

Physical and mechanical stress were pre-imposing factors, with dehydration playing an overall supporting role in the development of acute pancreatitis.

The aforementioned risk factors separately are rare causes of ischemic acute pancreatitis, but when presented in conjunction with each other, their accumulation may significantly heighten the risk of acute pancreatitis. Therefore, an acute necrotizing pancreatitis should be considered when confronted with similar situations in the clinical setting.

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