CASE REPORT
OBSERVATION DE CAS

Necrotizing pancreatitis resulting in abdominal compartment syndrome: a case report from a remote northern hospital and literature review

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INTRODUCTION

Most primary care providers and family medicine residents are aware of acute pancreatitis and a general medical approach. Less commonly seen, however, is its progression to necrotizing pancreatitis, which can further develop into abdominal compartment syndrome (ACS). Abdominal compartment syndrome carries a 50% risk of death.[1]

We describe the case of a middle-aged woman who presented to a remote northern hospital emergency department. She was admitted for presumed acute pancreatitis, but her condition continued to deteriorate, resulting in a clinical diagnosis of necrotizing pancreatitis with ACS. The case highlights the unique challenges of remote medicine, especially the lack of timely access to laboratory investigations, computed tomography (CT) and transportation to definitive care.

We conducted a PubMed and MEDLINE search of the English-language literature spanning 2011–2016 using the MeSH terms "pancreatitis," "ACS" and "intraabdominal hypertension." Based on titles and abstracts, 7 relevant research publications[1–7] were retained and reviewed for this case report.

CASE

A 39-year-old woman presented to the emergency department at the Weeneebayko General Hospital in Moose Factory, Ontario, with abdominal pain. She had a history of renal carcinoma with partial nephrectomy, fatty liver and diet-controlled diabetes mellitus. She described sudden-onset epigastric discomfort that started on waking, with multiple episodes of vomiting. She denied any bowel or bladder symptoms, and her last bowel movement was earlier that morning. She reported having had 3 sips of vodka cooler the night before; she reported rarely using alcohol otherwise. She denied any other substance use. She took no medications regularly. She denied any history of previous pancreatitis, elevated lipid levels or gallstones. She was in a monogamous sexual relationship. Review of systems gave negative results. Her vital signs were stable, and she was afebrile. The patient had diffuse abdominal discomfort on deep palpation of the abdomen, but there were no peritoneal signs.

Laboratory investigations revealed a leukocyte count of 15.8 × 10^9/L, hemoglobin concentration of 158 g/L and platelet count of 403 × 10^9/L. Electrolyte values were within normal limits, and the creatinine level was 36 μmol/L, with a normal estimated glomerular filtration rate. Liver function tests gave normal results aside from the γ-glutamyl transferase level, which was 82 U/L, double the upper limit of normal. The amylase level was elevated (279 U/L [normally 30–110 U/L]). Testing for serum

β-human chorionic gonadotropin and urinalysis gave negative results. Determination of lipase, bilirubin and hemoglobin A_{1c} levels and a lipid profile were ordered, but the results were not available before admission. A Ranson criteria score was not calculated at this time. The patient was admitted with a diagnosis of acute pancreatitis. She was not permitted to eat or drink, and intravenous therapy was started with crystalloid, morphine and dimenhydrinate.

The first morning of the hospital stay, the patient was tachycardic (120–130 beats/min) and febrile (maximum temperature 38.4°C). She was in significant distress from abdominal discomfort and bloating. She had peritoneal signs, including rebound tenderness, guarding, new distension and decreased bowel sounds. It was felt that her symptoms were not in keeping with "typical" pancreatitis. The only imaging available on site was radiography, and 3-view abdominal x-ray films were unremarkable. The locum general surgeon felt this was in keeping with acute pancreatitis and recommended CT of the abdomen.

Urgent medical evacuation was ordered for abdominal CT at the nearest hospital, which was 300 km away. This required helicopter and airplane flights with a nurse. Given the patient's presentation, intravenous antibiotic therapy was added for a potential intraabdominal abscess. That evening, the preliminary CT report included findings of "acute severe hepatitis," ascites and bowel angioedema. The pancreas showed "homogeneous enhancement, and there was no pancreatic or biliary duct dilatation."

Given that the patient did not require intensive care unit (ICU) admission at this time and was still considered a hospitalist patient from Moose Factory, previously accepted transfer protocol dictated that she be sent back to the inpatient unit overseen by family physician hospitalists in Moose Factory. Patients with acute pancreatitis, hepatitis or similar acute conditions requiring admission are returned to the unit owing to a high volume of patients, and a lack of beds and accepting physicians at the referral facility. Because of inclement weather, the patient was held over at a remote nursing station on the way back.

The second morning of the hospital stay, the patient had progressive abdominal distension that made her look 5 months' pregnant. She had ongoing pain, tachycardia and fever. Laboratory results remained normal except for an elevated lactate dehydrogenase level (1800 U/L [normally < 618 U/L]). She had no risk factors for hepatitis. Antibiotic treatment was stopped as there was no infectious etiology. Specimens for serologic testing for hepatitis were sent to a larger centre for processing. The patient's oxygen saturation level was 92% on room air, which improved to 95% on 2 L. Chest radiography showed bilateral opacification indicative of pneumonia, and therapy was started with ceftriaxone and azithromycin. Paracentesis was attempted, but bedside ultrasound examination showed a maximum fluid pocket of only 2 cm^2, with distended, edematous bowel. This did not support a diagnosis of ascites, and the patient's presentation did not fit with hepatitis, as reported on CT. She was accepted by Internal Medicine at a tertiary centre, but no beds were available. As the situation was critical, the provincial emergency referral service was contacted. An ICU physician from the same tertiary centre felt that the patient was too stable for the ICU and that we should continue to wait for the Internal Medicine bed. A nasogastric tube was inserted on the physician's recommendation.

The third morning of the hospital stay, the patient's condition worsened. She had ongoing tachycardia, fever and severe abdominal pain. Her distended abdomen now made her appear 9 months' pregnant. There was new anasarca and a slowly expanding flank hematoma (Grey Turner sign), suggestive of retroperitoneal bleeding. Her hemoglobin concentration was now 104 g/L. The Internal Medicine bed became available, but transportation was not available for the next 12 hours. The provincial emergency referral service was again contacted, and an ICU bed was found at another hospital. Transport arrived a few hours later. The admission laboratory results became available and showed a triglyceride level of 34.2 mmol/L (normally < 2.2 mmol/L) and a hemoglobin A_{1c} level of 9.2%. A diagnosis of necrotizing pancreatitis with ACS was made based on worsening symptoms, fever, tachycardia, respiratory distress and progressive anemia due to retroperitoneal bleeding.

Once at the tertiary centre, the patient had repeat abdominal CT, which showed pancreatitis with peritoneal and retroperitoneal fluid and bowel inflammatory changes. Review of the initial CT scan showed early signs of pancreatic necrosis and surrounding inflammation. She was treated with intravenous insulin therapy, oral fenofibrate therapy, ongoing intravenous administration of fluids and antibiotics for pneumonia. Her condition stabilized,

and she was repatriated back to our centre 8 days later. Serologic testing for hepatitis gave negative results.

DISCUSSION

Acute pancreatitis is the most common gastrointestinal complaint resulting in hospital admission.[1] The classic symptom of pancreatitis is acute epigastric pain radiating to the back with associated nausea and vomiting. There is often low-grade fever, tachycardia and hypotension. The abdomen is tender but usually less than expected for the subjective level of pain.[8] This fits with the initial presentation of our patient. Most patients recover within 3–4 days, but there can be serious complications.[1]

The Ranson criteria are used to predict death in patients with acute pancreatitis.[2] The first set of criteria applies to the patient on admission (leukocyte count > 16 × 10^9/L, age > 55 yr, glucose level > 10 mmol/L, aspartate aminotransferase level > 250 U/L and lactate dehydrogenase level > 350 IU/L). The second set of criteria is applied 48 hours after admission (decrease in hematocrit > 10%, increase in blood urea nitrogen level > 1.79 mmol/L, calcium level < 2 mmol/L, arterial partial pressure of oxygen < 60 mm Hg, base deficit > 4 mEq/L and requiring more than 6 L of fluid since admission). A score of 0–2 indicates a mortality rate of 2%, 3–4 a rate of 15%, 5–6 a rate of 40%, and 7–8 a rate of 100%. Our patient's score on admission is calculated to be 1 solely because of an elevated lactate dehydrogenase level on presentation. At 48 hours, the score was 2 (because of an elevated lactate dehydrogenase level and decrease in hematocrit of > 10% from admission), although some laboratory investigations needed to satisfy the criteria (e.g., arterial partial pressure of oxygen and blood urea nitrogen level) were not ordered, so the score is incomplete.

Intraabdominal hypertension may develop in up to 60% of patients with severe acute pancreatitis and may worsen to ACS in 27%.[3] Intraabdominal hypertension is defined as pathologic elevation in intraabdominal pressure of greater than 12 mm Hg.[4] Abdominal compartment syndrome is present when there is new organ dysfunction with intraabdominal pressure greater than 20 mm Hg.[4] Primary ACS is caused by intraabdominal disease, as in the current case of pancreatitis. Secondary ACS includes etiologies such as burns and postoperative complications.[5]

The cardiovascular, respiratory and renal systems are most commonly affected by ACS. From a cardiovascular perspective, increased intraabdominal pressure impairs venous return to the heart and increases left ventricular afterload. Overall, this reduces cardiac output and lowers organ perfusion pressure. This can predispose the pancreas to necrosis, which was seen in the current case of severe acute pancreatitis. In addition, high intraabdominal pressure elevates the diaphragm, reducing functional residual capacity and thoracic wall compliance. This functional restriction to ventilation can cause bibasilar atelectasis and pneumonia, as was seen on the chest x-ray films in our case. Renal compromise presents as oliguria. Elevated intraabdominal pressure decreases arterial perfusion and venous outflow from the kidneys. The renin–angiotensin system is activated, which causes fluid retention that may worsen the edema. When ACS occurs in patients with severe acute pancreatitis, the gastrointestinal system is often severely affected. High intraabdominal pressure compresses arterial perfusion and portal flow, which results in bowel edema and paralytic ileus,[5] both of which our patient had on physical examination and CT.

In pancreatitis, intraabdominal pressure starts to rise owing to severe inflammation beginning in the retroperitoneal and peripancreatic spaces. Surrounding tissue becomes edematous, and ascites begin to form. Abdominal wall edema reduces compliance, which further increases intraabdominal pressure. Retroperitoneal hemorrhage, which was suspected in our case given the presence of the Grey Turner sign, can further increase intraabdominal pressure.

The mainstay of management of severe acute pancreatitis is fluid resuscitation, which has been reported to be an independent contributor to the development of ACS.[6] Although analgesia, anxiolytics and gastrointestinal decompression are not fully endorsed by the World Society of Abdominal Compartment Syndrome (WSACS), some studies have shown these treatments to be helpful.[3–5]

To diagnose ACS, clinicians must keep this disorder in mind in patients with severe acute pancreatitis. Clinically, the abdomen may be tensely dilated, and there may be evidence of end-organ failure, which is uncommon in routine pancreatitis.[1] Mifkovic and colleagues[5] and the WSACS[4] recommend that intraabdominal pressure be measured routinely in patients with severe

acute pancreatitis to avoid missing the diagnosis of ACS. The WSACS developed a standardized protocol for measuring intraabdominal pressure that uses transvesical pressure as a surrogate instead of direct intraabdominal measurements. More details of the protocol can be found through the WSACS.[4]

The WSACS consensus guidelines outline treatment options that are supported by recent studies. Any patient in whom ACS is suspected requires transfer to an ICU for further care, but initial noninvasive treatment can start anywhere, including rural hospitals. Increasing intraabdominal pressure can be prevented by decreasing fluids after the first 48 hours of resuscitation, avoiding positive fluid balance if possible, and considering hypertonic or colloid fluids.[3–6] As for minimally invasive techniques, percutaneous drainage of intraabdominal fluid lowers intraabdominal pressure.[4,5,7] Finally, definitive management requires surgical decompression via laparotomy, which significantly reduces mortality rates in ACS.[4] In fact, hemodynamic and respiratory improvement can be immediate.[7]

Our case highlights systemic barriers to timely care. The patient was flown multiple times by an emergency air transfer service and required emergency CT at another hospital, and her case involved telephone consultations by multiple specialists and the provincial emergency referral service, yet her condition continued to deteriorate owing to a lack of resources both in the outlying hospital and in the hospitals to which she was to be transferred. Relatively simple initiatives such as funding imaging facilities (e.g., CT, or at minimum, ultrasonography) in rural hospitals and consideration of priority acceptance of rural patients with acute conditions by tertiary centres may dramatically improve patient care and outcomes. Of course, the addition of acute care beds in larger centres would facilitate faster transfers, as our patient was accepted but could not be transferred because of a lack of bed availability. Waiting hours for transport when an accepting physician and bed have been found confounds the situation faced by patients and physicians in rural and remote hospitals.

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