CASE REPORTS

Acute occlusion of the ductus pancreaticus due to abdominal aortic aneurysm: Uncommon cause of silent severe acute pancreatitis - a case report and review of the literature

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ABSTRACT

We report an uncommon case of severe silent acute pancreatitis (SSAP) caused by compression of the Ductus pancreaticus due to an abdominal aortic aneurysm (AAA) of 79 mm × 59 mm external diameter. A 78-year-old patient with known cutaneous progressive T-cell lymphoma and hypertension was referred to our institution in August 2013. During hospitalisation the patient became somnolent and developed elevated infection parameters. Abdominal ultrasonography showed a pulsating abdominal mass and CT examination revealed a stretched pancreas and an underlying partial thrombosed juxtarenal AAA extending distally to the origin of the superior mesenteric artery (SMA) and the aortic bifurcation without signs of visceral malperfusion elsewhere. The Ductus pancreaticus was dilated without involvement of the head. There were no additional radiological findings of occupying character other than the AAA. Because of his advanced age, increasing inflammatory parameters, and cutaneous T-cell lymphoma the patient was at this point neither suitable for open AAA surgery nor endovascular treatment. His clinical condition worsened due to development of a systemic inflammatory response syndrome (SIRS) and resulted in death. The presented case demonstrates that a growing AAA can cause, besides severe complications like perforation or dissection, in some rare cases SSAP as a first complication.

Key Words: Abdominal aortic aneurysm, Pancreatitis, Systemic inflammatory response syndrome

1. INTRODUCTION

Abdominal aortic aneurysm (AAA) is a disease found especially among older patients that in most cases remains asymptomatic until unveiled during radiologic testing or physical examination for other reasons.[1] First symptoms can be caused by growth of the aneurysm or rupture. The symptomatic non-ruptured AAA presents itself through back-, flank-, abdominal- or groin-pains and a pulsatile abdominal mass.[2] Sudden severe pain is typically the consequence of ruptured AAA and can be accompanied by profound hemodynamic instability and eventually limb ischemia due to a thrombus or debris mobilisation from the aneurysm.[1,3,4]

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There are only few documented cases in literature of AAA that initially became symptomatic through complications caused by compression of flanking abdominal organs, in particular, causing acute pancreatitis. In opposition, acute pancreatitis can lead to arterial aneurysms due to enzymatic degradation of the arterial wall, which is a rare, but well known complication.[5–8] However, first manifestation of acute pancreatitis caused by compression of pancreatic ducts due to a growth of arterial aneurysms, particularly AAA, remains a rarity.[8–10]

2. CASE REPORT

A 78-year-old man with known arterial hypertension, chronic obstructory pulmonary disease with cachexia, and vascular dementia was referred to our institution in August 2013 for further diagnostic staging and therapy of a cutaneous T-cell lymphoma, which had been confirmed by skin biopsies at the back and left upper leg (T3N0M0 B0, stadium IIB referring to the cutaneous lymphoma task force of the European organization of research and treatment of cancer[11]). During hospitalisation the patient became somnolent and presented elevated infection parameters (for c-reactive protein 5.9 mg/dl and for leukocytes 13.09 mg/nl). We performed basic diagnostic testing when abdominal ultrasonography demonstrated a pulsating abdominal mass. The patient had no further complaints and showed no symptoms of pain at all. The following CT examination revealed a stretched pancreas and an underlying partial thrombosed juxtarenal abdominal aortic aneurysm extending distally from the origin of the superior mesenteric artery (SMA) to the aortic bifurcation. However, the SMA and the Truncus coeliacus had significant stenosis at their origins, but there were no signs of visceral malperfusion. The Ductus pancreaticus was dilated to 4 mm in the corpus (see Figure 1). There was no involvement of the Ductus segment located in the head of the pancreas, although pancreas head was deviated fronto-lateral caused by the underlying mural thrombus of the AAA (see Figure 2). There were no additional radiological findings of other occupying processes, signs of organ infiltration by the T-cell lymphoma or other causes of obstruction. No additional radiological signs of acute pancreatic inflammation, pancreatic oedema or necrotizing pancreatitis were present. Blood chemistry revealed elevated values of amylase up to 1,741 (13-53) U/ml and lipase to 3,337.6 (5.6-51.3) U/ml (see Table 1). The patient presented without further typical signs of acute pancreatitis and there was no elevation of liver enzymes or total serum bilirubin. The patient had no history of prior abdominal surgery or intestinal disease except gastroesophageal reflux, which has been treated with omeprazole (20 mg/dl). There were no identifiable risk factors indicating the likely development of pancreatic disease and the patient never had been jaundiced. The patient was taking enalapril (10 mg/dl) orally.

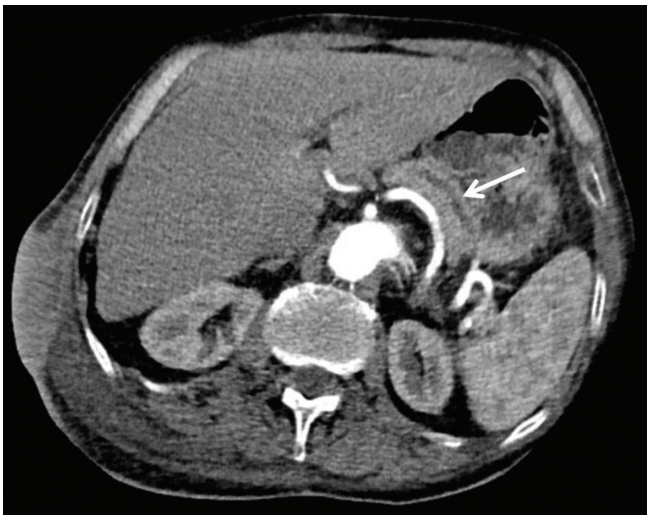

Figure 1. CT image demonstrating a slightly stretched pancreas with dilated pancreatic duct to 4 mm (arrow) and upper portions of the underlying partial thrombosed abdominal aortic aneurysm extending distally from the origin of the superior mesenteric artery (SMA) to the bifurcation with an external diameter of 79 mm × 59 mm

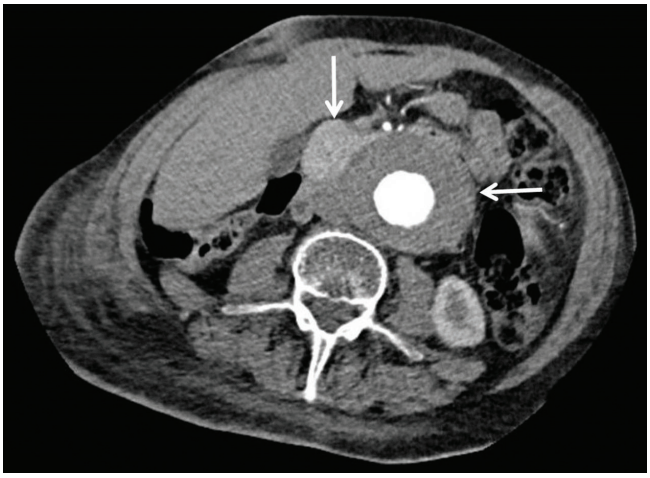

Figure 2. No involvement of the ductus segment located in the head of the pancreas (vertical arrow) was observed, although pancreas head was deviated fronto-lateral caused by the underlying mural thrombus of the AAA (horizontal arrow)

Although elevated values for amylase and lipase were regressive, clinical condition of the patient worsened with a general deterioration of health and the onset of hypotonia. The patient presented no fever at any time but required oxygen

therapy from this point forward. Blood chemistry showed metabolic acidosis with elevated lactate (up to 8 mmol/L). Despite calculated antibiotic treatment with sultamicilin (3 g/dl intravenously) blood values increased for c-reactive protein to 11.2 mg/dl and for leukocytes to 16.75 mg/nl, respectively. The patient was transferred to the intermediate care unit. Volume therapy with lactatet ringers's solution was initially sufficient to compensate the acidosis and stabilized blood pressure. Blood cultures were negative throughout the hospital stay. Due to persisting somnolence, hypotonia and elevated infection parameters the antibiotic medication was changed to meropenem (1 g intravenously). Despite supportive intensive care treatment the condition of the patient worsened and he showed signs of beginning renal failure. An additional CT examination demonstrated a slight increase of the AAA and a constantly dilated Ductus pancreaticus. Again there were no radiological signs of acute pancreatic inflammation, pancreatic oedema or necrotizing pancreatitis. Because of his advanced age, the progressive T-cell lymphoma, and his worsening clinical condition the patient was neither suitable for open AAA surgery nor endovascular treatment at this point. The developed SIRS resulted in multi-organ failure and caused death of the patient 10 days later.

Table 1. Baseline demographic and biochemical characteristics of the patient

Laboratory Studies	September 2013	Reference
White blood cells	13.09 g/nl	3.6-9.2 g/nl
Hemoglobin	11.9 g/dl	11.8-17.5 g/dl
Platelets	184 g/nl	140-320 g/nl
Prothrombin time	81%	70%-130%
INR	1.11	
Calcium	2.38 mmol/L	2.08-2.62 mmol/L
Aspartat aminotransferase	42 U/L	0-50 U/L
Alanine aminotransferase	22 U/L	0-50 U/L
Total bilirubin	0.5 mg/ml	0.3-1.2 mg/dl
Total protein	5.92 g/dl	6.4-8.3 g/dl
γ-Glutamyl-transferase	17 U/L	0-55 U/L
Lactat dehydrogenase	1,284 U/L	100-247 U/L
Amylase	1,741 U/L	13-53 U/L
Lipase	3,337.6 U/L	5.6-51.3 U/L
Cratinine	1.07 mg/dl	0.9-1.3 mg/dl
Blood urea nitrogen	24 mg/dl	6-19.8 mg/dl
C-reactive protein	5.9 mg/dl	0-0.5 mg/dl

Note. Age: 78 years; Gender: Male; Race: Caucasian; Height: 165 cm; Weight: 53 kg; BMI: 19 kg/m^2.

Table 2. Overview of the literature

Author/year	Age/gender	Aneurysm location	Obstruction location	Clinical symptoms	Treatment/Outcome
Dickinson, WL/1891	28/male	Abdominal aorta	Common bile duct	Obstructive jaundice and fatal hemorrhage	None, patient died
Chen, CY/1999	71/male	Abdominal aorta	Common bile duct	Epigastralgia, vomiting, fever	Patient died after three recurrent episodes
Spinelli, GD/1982	71/male	Abdominal aorta	Common bile duct	Jaundice, fever, abdominal pain	Surgery: choledocho-duodenal anastomosis, recovery
Van Gossum, A/1982	76/female	Abdominal aorta	Lower common bile duct	Oral bleeding and disseminated intravascular coagulation	None
Hashmonai, M/1981	73/male	Abdominal aorta	Pancreatic head	Obstructive jaundice and sudden abdominal pain	Surgery: open aortic aneurysm repair
Liebermann, Da/1983	67/male	Abdominal aorta	Common bile duct, pancreas and duodenum	Jaundice, burning epigastric pain	Surgery: open aortic aneurysm repair, postoperative septic shock and death
Dohi, K/1984	69/female	Abdominal aorta	Common bile duct and duodenum	Obstructive jaundice	Surgery: open aortic aneurysm repair, recovery
Van Someren, N/1993	87/female	Abdominal aorta	Common bile duct	Right hypochondrial pain, vomiting	Conservative, recovery
Dorrucci, V/2001	87/female	Abdominal aorta	Common bile duct	Obstructive jaundice, backpain	Surgery: open aortic aneurysm repair, recovery
Smith, AD/2002	84/female	Abdominal aorta	Common bile duct	Vague non-specific abdominal discomfort, abnormal liver test	Conservative, symptoms recurrent
Cowell, D/2010	80/female	Abdominal aorta	Lower bile duct	Abdominal pain, elevated liver enzymes	Conservative, symptoms recurrent
Fukui, T/2012	88/female	Abdominal aorta	Distal common bile duct, pancreatic head and duodenum	Vague non-specific upper abdominal discomfort	Endovascular stent grafting of the aortic aneurysm, complete recovery

3. DISCUSSION

Acute pancreatitis is usually accompanied by severe clinical symptoms. Numerous conditions are known to induce acute pancreatitis. Chronic alcohol abuse and gallstones account for nearly 70% of the cases among industrial populations. In most cases mechanical ampullary or ductal compression is

either caused by gallstones or result of malignant processes, usually of the pancreas itself. To our best knowledge there are only 12 cases according to literature (see Table 2) reporting aneurysms of the abdominal aorta as the cause for acute obstruction of the pancreatic duct or distal common bile duct.[12–23] Also among radiological findings of uncommon types and causes of pancreatitis, aneurysms of the abdominal arteries, especially AAA, remain a rarity.[8]

According to the widely used Atlanta classification, our patient suffered from acute severe pancreatitis by presenting pulmonary insufficiency, hypotension and renal failure. Despite his poor clinical condition, he presented at no point typical clinical symptoms such as severe upper abdominal pain radiating to the back, nausea or vomiting. Cases of symptomatic acute pancreatitis due to compression of the distal common bile duct by a growing AAA, with sudden onset of severe symptoms, have been reported before.[13,15–17,22] However, common bile duct compression by a growing AAA may also lead to episodes of recurrent jaundice[18,19] with only mild and unspecific symptoms.[18,21] Fukui *et al*. reported a case with unspecific abdominal symptoms and compression of the distal common bile duct with distension of the proximal bile and main pancreatic ducts in the body and tail of the pancreas by a growing AAA.[23] The patient was treated successfully by endovascular stent grafting via the femoral artery and recovered completely.

However, due to the absence of typical clinical symptoms in our patient, diagnosis may have been delayed. At the time diagnosis was established, the clinical status of the patient had already worsened to a point where invasive treatment such as open surgical repair or endovascular treatment were no longer feasibly. In general, painless acute pancreatitis is an uncommon and rare finding and has been reported in coherence with sorafenib treatment,[24] after renal transplantation,[25] after assistive device implantation,[26] as a rare complication of Legionnaires' disease[27] and in one other case with panniculitis as the primary solitary manifestation.[28]

Our case is the first to best of our knowledge, where a growing AAA presented via a silent severe acute pancreatitis as the first clinical manifestation. Therefore, we suggest that also uncommon complications caused by a growing AAA should to be taken into consideration, especially in the elderly patient with pancreatitis. This may avoid crossing the line were potential treatment options are not feasibly any longer.

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