Complicated chronic pancreatitis – A case report of unusual presentation

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Abstract

The authors describe a patient with a five month history of asthenia, anorexia and weight loss. Dysphagia for solid foods appeared together with dyspnea three months after the onset of the disease. The investigation led to a diagnosis of alcohol-related chronic pancreatitis with infected mediastinal pseudocyst, pancreatic fistula, pancreatic pleural effusion and probable splenic artery thrombosis.

Key words: chronic pancreatitis, pancreatic pseudocyst, pancreatic fistula.

INTRODUCTION

Chronic pancreatitis (CP) is an inflammatory disease that results in permanent pancreatic structural lesions that can lead to a decrease in endocrine and exocrine functions of the organ.

Since its earliest description (in 1788 Cawley described a youth who died of emaciation and diabetes and whose autopsy revealed multiple pancreatic calculi), there have been many publications about this entity, but today it still remains an enigmatic disease with uncertain pathogenesis, unpredictable clinical course, and undefined treatment.

The authors describe the clinical case of a patient recently admitted to our hospital with several characteristics of this pathology.

CLINICAL CASE

A male patient, 47 years of age, Black, born and resident in Angola (Luanda), a mining engineer, married.

He arrived at our outpatient service with a history of around 5 months of evolution of insidious establishment and progressive exacerbation of asthenia, anorexia and effort intolerance, with which dyspnea was later associated. The patient was observed at the Hospital Militar de Luanda, where he was diagnosed with right pleural effusion and a pleural tap was performed; he was then informed that he was suffering from a specific pleurisy, and was medicated with Isoniazide + Rifampicin + Pyrazinamide + Ethambutol + Ciprofloxacin. These measures brought about a temporary improvement for around two months, after which the patient complained of fatigue on increasingly little effort, with marked asthenia. There was no fever, thoracic, abdominal or other kinds of pain, nausea or vomiting, orthopnea or paroxysmal nocturnal dyspnea, edemas or nocturia at any time. Symptoms present during the entire period of the current disease were: anorexia and weight loss of about 10 Kg (> 10% of the body weight) and in the last three months, complaints of dysphagia for solid foods, sometimes with a sensation of food obstruction in thoraco-abdominal transition.

The most notable aspects of personal history were: 1) Poliomyelitis at two years of age 2) Pulmonary tuberculosis twenty years previously, diagnosed and treated in Luanda for one year with anti-tuberculosis drugs, with annual clinical and radiological control and no evidence of recurrence of the disease as of the time of the current disease 3) Accentuated alcohol consumption that is difficult to quantify but with definite daily consumption > 100 g alcohol/day for more than thirty years, up to about 1.5 years ago, when the patient abstained totally from alcohol. He has never suffered from abdominal pains, vomiting or alterations in bowel movement. 4) Several paludism episodes, always non-severe, some self-limited and

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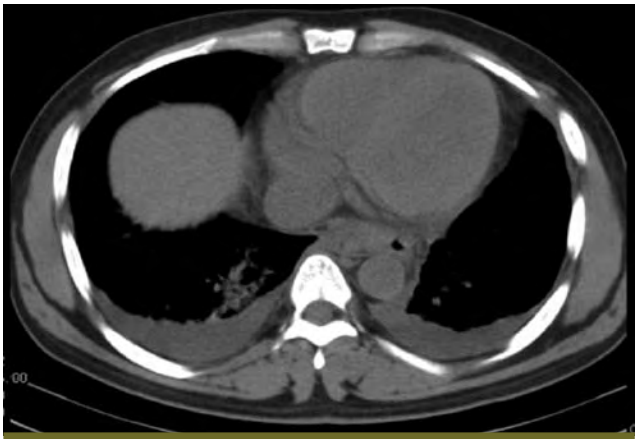
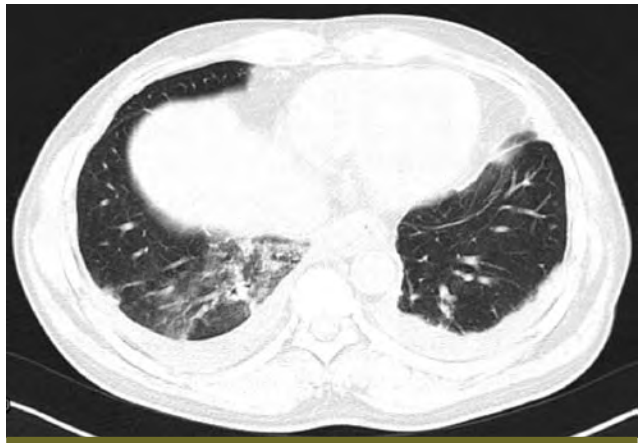


Elongated cluster in right lateroesophageal location with cranio-caudal extension; parietal thickening of the distal thoracic and intra-abdominal esophagus.

FIG. 1



Exudative densification of the pulmonary parenchyma of the right lower lobe, suggested by Imagiology to be inflammatory or hemorrhagic; bilateral pleural effusion.

FIG. 2

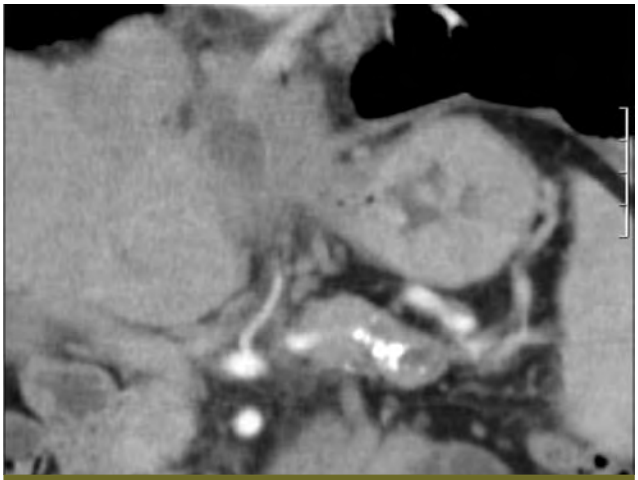
others treated with oral anti-paludism drugs. 5) Aberrant hepatic duct (AHT) was diagnosed one month ago and medicated and controlled with Captopril 25 mg 1x/day + Carvedilol 25 mg 1x/day.

In the objective examination, a positive aspect emphasized was good general and nutritional state, but with evident mucosal paleness; cardiac auscultation revealed a grade III/VI systolic murmur on the left sternal border and mitral area, and pulmonary auscultation showed a bilateral decrease in vesicular in the lung bases and pleural attrition on the right side. There were no palpable enlarged lymph nodes in the superficial ganglionar regions, hepato- or splenomegaly or any other organomegaly or palpable tumor.

Analytically there was normocytic and normochromic anemia (Hb = 6.8g/dl) with normal ferritin, low serum iron and transferrin, ESR = 33 mm 1st hour, CPR normal.

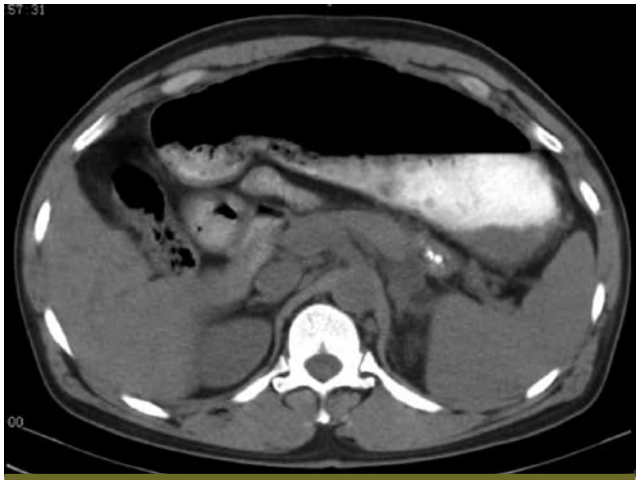
Thoraco-abdominal computed tomography (CT) carried out in the initial radiological evaluation revealed a parietal thickening of the distal esophagus, with an elongated cluster of 2.5 x 4 cm with cranio-caudal extension between this and the diaphragm pillar on the right; the cluster appeared to have an intra-abdominal component through the diaphragmatic hiatus; furthermore exudative densification was observed in the right lung base, with inflammatory or hemorrhagic imagiological suggestion, whose anatomical relation with the paraesophageal cluster was not clear. Bilateral pleural effusion, fibro-cicatricial alterations of the right apex, and several alterations of the chronic pancreatitis limited to the tail (calcifications on the tail partially aligned with the Wirsung duct possibly corresponding to lithiasis) were also described. No other alterations, particularly in the liver or intra-abdominal lymph nodes, were described. *(Figure 1 and 2)*

In view of these findings, the patient underwent upper digestive endoscopy (UDE), which showed gastric varices and findings compatible with severe hypertensive gastropathy. He then underwent an upper echo-endoscopy in which preserved thickness and architecture of the esophageal wall were observed, with the presence, in this location, of a collection of liquid cluster measuring 2.5 x 5 cm, with scalloped wall, some septa in its interior and sedimentary echogenic content, with two associated adenopathies, leading to a hypothesis of abscess; the presence of gastric varices was confirmed (with perigastric collateral circulation) and chronic pancreatitis of the body and tail without focal lesions. This was followed by a transesophageal puncture of the liquid cluster, aspirating 20cc of greenish liquid. Analysis of this liquid showed the absence of cellular elements, amylase levels of 100720 UI/L and total bilirubin of 3.30mg/dL. The test was negative for mycobacteria both by classical direct and culture test and by CPR. Gram staining showed many gram-positive cocci which,



Paraesophageal cluster with craniocaudal extension that reaches from the lower 1/3 of the esophagus to the pancreatic tail.

FIG. 3



Paraesophageal cluster that extends up to the pancreatic tail; alterations of chronic pancreatitis limited to the tail (calcifications on the tail partially aligned with the Wirsung duct, possibly corresponding to lithiasis).

FIG. 4

in the culture test were identified as streptococci of the viridians group.

About 24 hrs after the abovementioned transesophageal puncture, the patient had an episode of lipothymia accompanied by pleuritic thoracalgia at the right base, with clinical signs of poor peripheral perfusion, having objectified hypotension and tachycardia and in the absence hypoglycemia. He later suffered an episode of chills followed by a febrile peak of 38.5ºC.

At this point there was no evidence of esophageal perforation in the thoraco-abdominal CT, and the image continued to show the paraesophageal cluster, now considerably increased and measuring 4 x 10 cm (axial and craniocaudal respectively) and clearly extending from the lower 1/3 of the esophagus to the pancreatic tail, corresponding to a fistula originating from the Wirsung duct *(Fig. 3 and 4)*.

A diagnosis of septic shock was made, and the patient was transferred to the Intensive Care Unit, where he was placed on antibiotic therapy with meropenem + metronidazole. Valvular endocarditis was excluded by echocardiogram.

The patient was operated on day 7 after admission to the ICU, with a body-tail splenopancreatectomy and drainage of the residual cavity and the cystic cavity. In the postoperative period the patient developed difficulty establishing intestinal transit, which did not resolve with conservative therapeutics, and was re-operated on day 14, for lysis of adhesions and adherences in the postoperative period of this second operation, the patient developed pancreatitis of the residual pancreas, which resolved spontaneously, and he was discharged without complaints of abdominal pains, intestinal transit re-established, and the diabetes mellitus controlled by insulin therapy.

DISCUSSION

The pathogenesis of chronic pancreatitis seems to be multifactorial and is probably triggered by different events, such as activation of digestive enzymes inside the pancreatic gland; this activation may be the result of the interaction between the presence of genetic anomalies that predispose to the formation of toxic lesions once exposed to environmental factors, including alcohol.[1] Indeed, chronic alcohol abuse was always indicated as being responsible for the vast majority (60-70%) of chronic pancreatitis cases, but the association between alcohol and chronic pancreatitis is complex. This notion of complexity is based on the fact that only 5-10% of alcoholics develop chronic pancreatitis, and the reason for this predisposition is not known; for example, it is known that a chronic alcoholic who is Black, of African origin, has a greater chance of developing pancreatic disease than a Caucasian, while the opposite is true

of alcoholic liver disease.[1]

Our patient had a longstanding history of regular and high alcohol consumption (>100g/day), which is the factor involved in the etiology of the disease, and there was no imagiological or laboratory evidence of liver disease.

As regards the clinical manifestations of chronic pancreatitis, which are classically pain and endocrinal and/or exocrine pancreatic insufficiency, our patient had never had any episode of pain that would suggest a pancreatic complaint, and there was no evidence of endocrinal or exocrine pancreatic insufficiency. The characteristics of pain in chronic pancreatitis vary from patient to patient, and although it is the most consistent symptom of the disease, it can be absent in some cases (10% to 20%, according to some authors).[2] Exocrine pancreatic dysfunction, which leads to an inability to digest complex foods and absorb those already partially digested, only becomes clinically evident when more than 90% of the glandular function is lost, therefore it is invariably a late manifestation. Endocrine pancreatic insufficiency, in turn, almost always appears with glucose intolerance, while declared diabetes mellitus also generally has a late onset. In fact, although diabetes mellitus is more frequent among patients with chronic pancreatitis and pancreatic calcifications, as was the case of our patient, endocrine dysfunction only became evident after pancreatectomy.[3]

Chronic pancreatitis can be associated with a wide range of complications, the most common of which are the formation of pseudocysts, as well as obstructions of the duodenum and main bile duct; the least common complications include pancreatic ascites or pleural effusion, splenic vein thrombosis with portal hypertension, and the formation of pseudoaneurysms of arteries anatomically close to the pancreas, in particular the splenic artery.

Pancreatic pseudocysts are very frequent (appearing in around 10% of cases of CP) and many are asymptomatic. However, they can give rise to a series of clinical problems, depending on their location, dimension and any complications. This was the case of our patient, who exhibited a large cyst that underwent a sudden and considerable increase (in the first CT it measured 2.5 x 4 cm and in the second CT, 7 days before surgery, 4 x 10 cm), the location of which caused dysphagia for solids. However our patient also had a serious complication of pseudocysts: infection. Indeed, the endoscope puncture enabled the isolation of a streptococcus viridians, which surpassed the dimension of the cyst and the compressive symptoms on the distal esophagus, another argument in favor of not delaying the surgical intervention.[2,4] The pleural effusion that had been drained in Luanda, the results of the cytobiochemical examination being unknown to us, was very likely a pancreatic pleural effusion. It is probable that the formation mechanism of this effusion was the formation of a fistula, possibly resulting from the disruption of a pancreatic duct, and that this was responsible for the formation either of the effusion and possibly of the pseudocyst; there is also the theory that the rupture of the mediastine pseudocyst was inversely responsible for the formation of the pleural effusion.[2] It is worth observing that a course is described in the preoperative CT between the paraesophageal cluster and the pancreatic tail, making the first hypothesis more likely.[5,6] Another less frequent complication is thrombosis of the veins anatomically related to the pancreatic inflammatory process. The splenic vein, which passes along the posterior side of the pancreas, is the most frequently involved.[7] Despite the fact that we did not directly demonstrate thrombosis of the splenic vein by imaging in our patient, we saw evidence of gastric varices and severe hypertensive gastropathy in the HDE and in the echoendoscope, whereas the CT findings suggest venous hypertension located in the splenic vein but not in the portal vein territory.

The clinical case of our patient is notable for the fact that it illustrates a series of complications that are not frequent in themselves, and even less so when occurring together in the same individual, whereas the form of presentation of the base disease, which was alcoholic chronic pancreatitis, was once again defined by the absence of the two manifestations generally considered essential for diagnosis- pain and pancreatic insufficiency. In this patient, the general systemic symptoms of anorexia asthenia and weight loss dominated the picture, together with some more localized symptoms resulting not from the chronic pancreatitis itself, but from its complications, i.e., dyspnea caused by pleural effusion, and dysphagia caused by the compression of the pseudocyst on the distal esophagus.

In terms of differential diagnosis, the one that produced the most controversy was that of neoplasia of the pancreas, since weight loss in the absence of

absorption deficit, and the dominance of the general symptoms constituting the clinical picture of presentation, made this a very likely hypothesis. ■

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