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Case report

Diabetic ketoacidosis as the presenting manifestation of pancreatic adenocarcinoma with cystic features

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ABSTRACT

The common presenting symptoms of pancreatic cancer are abdominal pain, weight loss, and jaundice. Pancreatic adenocarcinoma presenting with diabetic ketoacidosis is a very rare emergent clinical condition. However, pancreatic ductal cystadenocarcinoma presenting with diabetic ketoacidosis was not reported. We describe a 60-year-old man with pancreatic cystadenocarcinoma presenting with diabetic ketoacidosis as the initial manifestation. It must be kept in mind that in diabetic ketoacidosis cases, the precipitating factor may be pancreatic ductal cystadenocarcinoma.

1. Introduction

The relationship between pancreatic cancer and diabetes is controversial.[1] Patients with pancreatic cancer might have abdominal pain, weight loss, jaundice, back pain, vomiting, indigestion, and pruritus.[2] Pancreatic adenocarcinoma presenting with diabetic ketoacidosis (DKA) is very rare and to the best of our knowledge, only three cases have previously been reported.[3–5] However, pancreatic cystadenocarcinoma presenting with DKA is not known. Here, we present a case of pancreatic cystadenocarcinoma whose presenting symptom was DKA.

2. Case presentation

A 60-year-old male patient presented to the emergency department of our hospital with the complaints of weakness, nausea and loss of appetite for 3 days. There was about 10 kg weight loss during the last 2 months. He had type 2 diabetes for 5 years and had been taking metformin 2000 mg/day for 3 years. Patient had high blood sugar readings at home for the last two months, but he didn't seek medical attention for this. He smoked 5 cigarettes daily for thirty years. On physical examination, he was conscious, arterial blood pressure was 130/85 mmHg, pulse rate was 110/min, and the body temperature was 36.6 °C. His tongue was dry, skin turgor and tonus were decreased. The patient's body mass index was 32.3 kg/m^2. The initial laboratory results were as follows: Serum glucose 473 mg/dL, urea 69 mg/dL, creatinine 2 mg/dL, sodium 132 mEq/L, potassium 4.3 mEq/L, chloride 97 mEq/L, alanine aminotransferase 29 U/L, aspartate aminotransferase 34 U/L, total bilirubin 0.3 mg/dL, direct bilirubin 0.1 mg/dL, and HbA1c 12.5%. Ketones and glucose were both 3 + and density (specific gravity 1028) was increased on urine analysis. Arterial blood gas analysis showed increased anion gap metabolic acidosis (pH 7.28, PaCO2 25.7 mmHg, $HCO3^-$ 14.4 mEq/L, PaO2 88.2 mmHg, SaO2 95.8%, lactate 1.5 mg/dL and anion gap 26.5 mEq/L). He was admitted to our clinic and intravenous fluids, electrolytes, and regular insulin infusion were started. Urea and creatinine values returned to normal range and the clinical picture of DKA improved. Intensive SC insulin treatment was begun. Level of CA19-9 was 202 U/ml (reference range 0–37). On the abdominal ultrasonography, a hypoechogenic lesion was observed in the pancreatic head, which had internal septations and produced sonic posterior wall echoes. On the abdominal CT, a cystic necrotic mass of 54 × 42 mm dimensions was observed in the head of pancreas; it surrounded the gastroduodenal artery and invaded the portal and splenic veins (Fig. 1). No metastasis was detected. However, general surgery decided that the patient was inoperable

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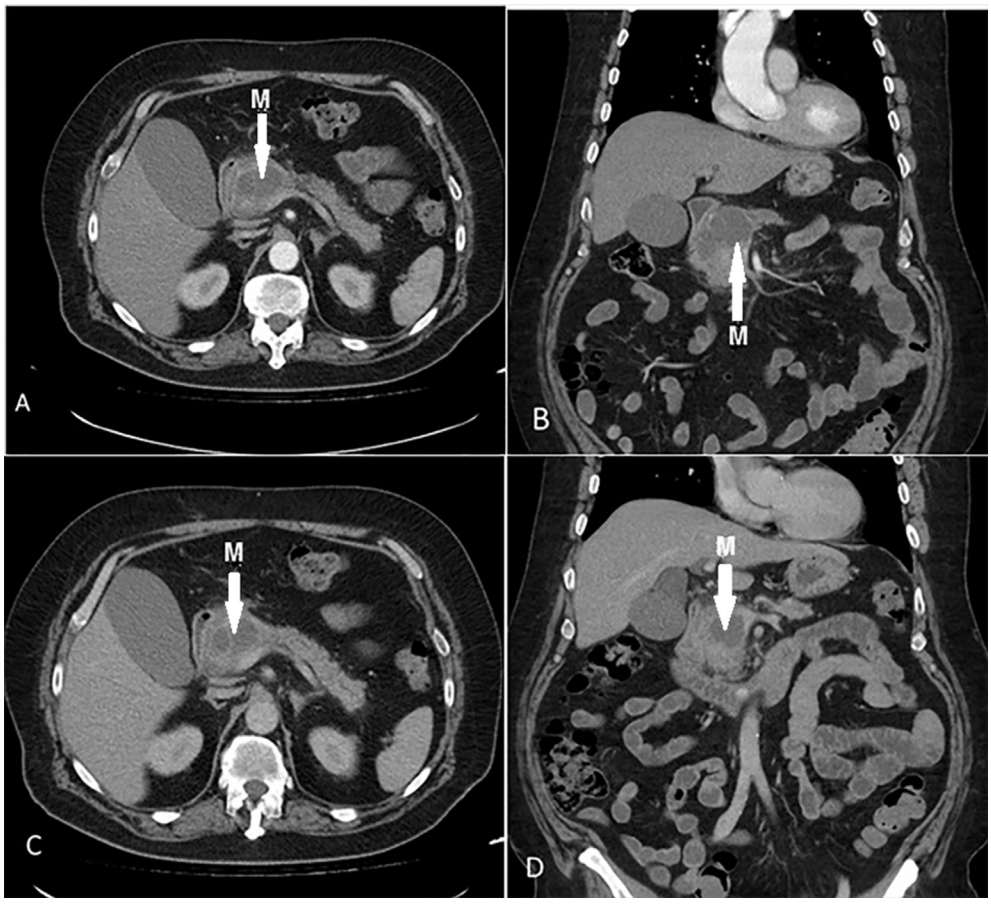

Fig. 1. Abdominal contrast-enhanced CT scan showing the solid and cystic mass in the head of the pancreas. Axial (A) and coronal (B) images in the arterial phase. Axial (C) and coronal (D) images in the portal phase. The lesion invaded the portal vein.

due to the vascular invasion. Icterus developed progressively after the fourth day of hospitalization. While biliary tree was seen to be normal on the admission ultrasonography, repeat USG on the fifth day revealed that intrahepatic biliary ducts and the choledocus were dilated. Obstructive icterus developed secondary to the mass at the head of pancreas. Endoscopic retrograde cholangiopancreatography was performed and a stent was placed for the malignant stenosis at the distal part of the choledocus. In the following days, patient's liver enzymes and bilirubin returned to normal. The histopathological assessment of the mass in the head of the pancreas, from which a biopsy specimen was obtained through percutaneous fine needle aspiration, was found to be consistent with pancreatic ductal adenocarcinoma (PDAC) (Fig. 2). The patient's blood glucose was regulated with basal and mealtime insulin treatment. The patient was discharged and referred to the medical oncology outpatient clinic.

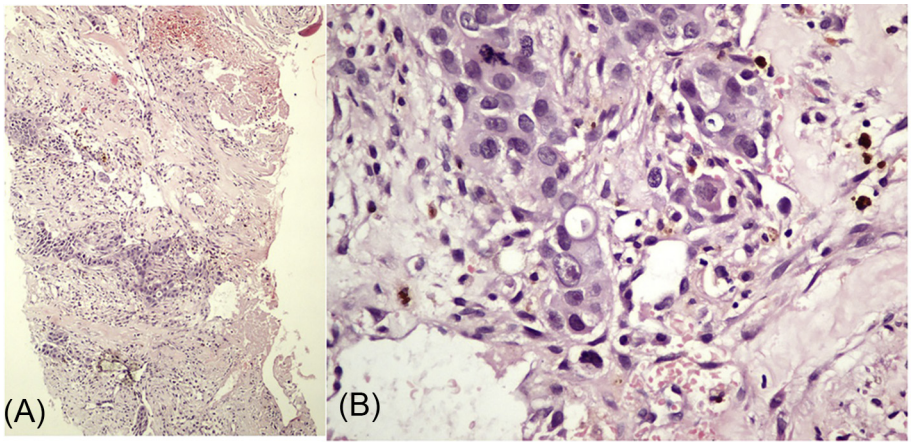

Fig. 2. Histopathology showing the development of malignant tumor as pancreatic ductal adenocarcinoma with consisting pleomorphic cells, infiltrative appearance, and embedded in desmoplastic stroma (Images A and B).

3. Discussion

Pancreatic cancer is more frequent among individuals ≥60 years old, and in the United States, it is ranked in the fourth place among deaths caused by cancer.[6] Over 95% of these cancers originate from exocrine pancreatic cells and the most frequent histological form is PDAC (85%).[2] Although PDAC is solid in general, in 8% of the cases, it may comprise cyst-like features such as cystic degeneration, retention cysts, and attached pseudocysts.[7] PDAC with cystic features as in the present patient is rare. The differential diagnosis of pancreatic benign cystic lesions from PDAC is clinically important. Lv et al reported that the following CT imaging features may be useful for the diagnosis of PDAC: irregular contour, multiple cysts, mural nodes, localized thickening, dilatation of the main pancreatic duct, peripancreatic fat infiltration, and vascular or peripheral tissue involvement.[8] On the CT of the present patient, there were irregular contour, vascular and peripheral tissue involvement, rendering consideration of PDAC.

It has been demonstrated in previous studies that pancreatic cancer and diabetes can both be the cause of one another, and can affect one another as a result.[1] In a meta-analysis, Ben et al had reported that there was a two-fold risk of developing pancreatic cancer in diabetic patients.[9] There is glucose intolerance or frank diabetes in about 80% of patients with pancreatic cancer.[2] The mechanism of development of diabetes related to pancreatic cancer is not completely understood.[10] Mechanical obstruction of the pancreatic duct, hyperinsulinism and insulin resistance, tumor size and genetic variants have been claimed to possibly be effective.[1,10] All of the afore-mentioned mechanisms may have played a role in the development of DKA in our case.

DKA is one of the life-threatening serious acute complications of diabetes. It occurs due to an increase in the counter-regulatory hormones as a result of insulin insufficiency.[11] The main factors that cause DKA include new onset type 1 diabetes, poor compliance with the insulin regimen, severe stress, trauma, infection, cerebrovascular accident, surgery, excessive alcohol intake, pancreatitis, pregnancy, acute myocardial infarction, and drugs that affect the carbohydrate metabolism.[11] As a novel presentation, in this patient, we report DKA as the initial manifestatation of pancreatic cystadenocarcinoma. It must be kept in mind that, although rare PDAC might be the precipitating factor in DKA cases.

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None Declared.

Conflicts of interest

None declared.

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