

Type 3C Diabetes Mellitus Secondary to Chronic Pancreatitis: Case Report

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Abstract

Introduction: Type 3C diabetes mellitus (T3cDM), also known as pancreatogenic diabetes or diabetes of the exocrine pancreas, remains an underdiagnosed clinical entity. Acute pancreatitis is an inflammatory disease of the exocrine pancreas that accounts for over 300,000 hospitalizations annually, with a global incidence of 34 cases per 100,000 people and geographic variation. **Objective:** To present the case of a patient with pancreatogenic diabetes mellitus. **Case Report:** A 54-year-old male with a known history of insulin-requiring type 2 diabetes mellitus presented with marked hypoglycemia and a right gluteal abscess. Laboratory findings showed leukocytosis, anemia, and elevated acute-phase reactants. Imaging studies confirmed chronic pancreatitis and decreased C-peptide levels. **Conclusions:** This condition is often underdiagnosed and is associated with damage to pancreatic cells, leading to abnormal insulin release and an initial hypoglycemic state, which is frequently the presenting symptom, as seen in this case. A significant number of patients diagnosed with type 2 diabetes may present with poorly localized abdominal pain or signs of malabsorption. Therefore, it is important to inquire about alcohol and tobacco use, as both are risk factors for pancreatic injury. When clinical suspicion arises, imaging studies and pancreatic function tests are essential to evaluate pancreatic damage and dysfunction.

Keywords

Diabetes mellitus, pancreas, pancreatic diseases, pancreatitis.

INTRODUCTION

Type 3c diabetes mellitus (T3cDM), also known as *pancreatogenic diabetes*, remains an underdiagnosed pathological entity⁽¹⁾. Etiologically, it develops in contexts of chronic pancreatitis, pancreatic cancer, cystic fibrosis, hemochromatosis, and prior pancreatic surgery⁽²⁾. Its pathophysiology is not fully understood but likely involves multiple factors including: islet cell mass loss, acute pancreatitis-induced autoimmunity, shared etiological risk factors, local/systemic inflammatory responses, and insulin-incretin axis dysfunction⁽³⁻⁵⁾. T3cDM affects all pancreatic islet cells, exhi-

biting both insulin resistance and production deficiency while also disrupting glucagon, pancreatic polypeptide, incretin, and adipokine secretion - making it a unique clinical entity⁽⁶⁾.

Acute pancreatitis - an exocrine pancreatic inflammatory disease causing over 300,000 hospitalizations annually - has a global incidence of 34 cases per 100,000 people with geographic variation, and carries a 40% cumulative diabetes mellitus (DM) incidence rate⁽⁷⁾. Diagnostic criteria include major and minor components: major criteria require DM diagnosis plus pancreatic pathology on imaging (CT, endoscopic ultrasound, or MRI), while minor criteria

include pancreatic polypeptide/incretin secretion defects, low basal C-peptide, or fat-soluble vitamin deficiencies^(8,9). This article presents a case of pancreatogenic DM.

CASE REPORT

A 54-year-old male with a heavy alcohol consumption history (weekly binge drinking) and insulin-dependent type 2 DM presented after self-administering 30 IU insulin glargin, with a home glucose reading of 28 mg/dL accompanied by neuroglycopenic symptoms. He reported a 15-day history of right gluteal abscess without discharge.

On admission, vital signs showed BP 95/67 mmHg, HR 54 bpm, RR 22 rpm, and temperature 36.0°C, with dia-phoresis and mucocutaneous pallor. Initial labs revealed: WBC: 14,570/mm³, neutrophils: 77.4%, hemoglobin: 9.8 g/dL, hematocrit: 29.2%, platelets: 472,000/mm³, serum glucose: 30 mg/dL, sodium: 128 mmol/L, chloride: 108 mmol/L, potassium: 3.5 mmol/L, C-reactive protein (CRP): 24 mg/dL, prothrombin time (PT): 13.3, international normalized ratio (INR): 1.08, partial thrombo-plastin time (PTT): 35.1 sec, and creatinine: 0.74 mg/dL. Soft tissue ultrasound showed right gluteal inflammatory process without abscess.

By day 3, with worsening clinical course and new abdominal pain, contrast abdominal CT revealed chronic pancreatitis signs with anasarca (**Figure 1**), moderate free pleural effusion, moderate ascites, and diminished basal (0.1 ng/dL; reference range [Ref.]: 0.48-5.0 ng/mL) and postprandial (0.2 ng/dL; Ref.: 4.0-6.0 ng/mL) C-peptide levels. Despite treatment, the patient expired on day 4.



Figure 1. Axial CT scan showing atrophic pancreas with parenchymal microcalcifications suggestive of chronic pancreatitis. Image property of the authors.

The patient developed chronic pancreatitis likely secondary to chronic alcohol abuse, causing total pancreatic dysfunction that triggered admission hypoglycemia and subsequent clinical deterioration leading to fatal outcomes.

DISCUSSION

The diagnosis of pancreatogenic diabetes or type 3c diabetes mellitus (T3cDM) presents significant challenges due to the lack of standardized diagnostic tests and frequent misdiagnosis as type 2 diabetes. The American Diabetes Association (ADA) classifies this condition under “other specific types of diabetes,” including diabetes secondary to exocrine pancreatic disease⁽¹⁰⁾. Our patient had been diagnosed with type 2 diabetes for 10 years and required high insulin doses during treatment. Approximately 78.3% of T3cDM cases are initially misdiagnosed as type 2 diabetes⁽⁵⁾. Several risk factors influence diabetes development in chronic pancreatitis patients, including smoking, alcohol consumption, and pancreatic fat accumulation, all identified as significant contributors⁽¹¹⁾. Our patient had a history of early-onset alcohol use and occasional smoking.

The pathophysiology of type 3 diabetes characteristically involves destruction of pancreatic islet cells or acinar cells, leading to glucose dysregulation that manifests as hypoglycemic complications and increased mortality. Exocrine pancreatic dysfunction impairs nutrient absorption and incretin/glucagon secretion, with eventual alpha cell destruction. This glucagon deficiency likely explains our patient’s severe hypoglycemic episodes, while his BMI of 17.6 kg/m² indicated malnutrition⁽¹²⁾. Islet cell destruction and cytokine production promote exocrine pancreatic insufficiency, causing pancreatic polypeptide (PP) cell loss and hepatic insulin resistance. The absence of PP response to mixed-nutrient ingestion helps differentiate T3cDM from type 2 diabetes pathogenesis⁽¹³⁾. Our patient’s PP level of 0.1 ng/dL (normal range: 0.5-2.0) demonstrated complete exocrine pancreatic failure, with deficiency associated with reduced hepatic insulin receptors⁽¹⁴⁾.

Premature beta cell destruction from inflammation leads to diabetes development in these patients. In our case, contrast-enhanced abdominal CT revealed chronic pancreatitis. Calcific pancreatitis represents one of the most common causes of secondary diabetes⁽¹⁵⁾, though it may also follow acute pancreatitis.

Diagnostic criteria for our case included: exocrine pancreatic insufficiency, pathological pancreatic imaging, and absence of type 1 diabetes autoantibodies (unfortunately not assessed due to patient mortality)⁽¹⁶⁾. Early diagnostic imaging like ultrasound is recommended for suspected chronic pancreatitis cases⁽¹⁷⁾.

Management requires oral pancreatic enzyme replacement and fat-soluble vitamin (A, D, E, K) supplementation alongside appropriate insulin therapy. Comprehensive preventive treatment addresses malnutrition and promotes healthy lifestyle modifications^(18,19).

CONCLUSION

This underdiagnosed pathological entity results from pancreatic cell damage, causing abnormal insulin release and initial hypoglycemia - often the presenting symptom, as demonstrated in our case. Many patients diagnosed with type 2 diabetes may report nonspecific abdominal

pain or show malabsorption signs. Clinicians should thoroughly assess alcohol and tobacco use, established pancreatic injury risk factors, and pursue pancreatic imaging/function tests when T3cDM is suspected to evaluate pancreatic damage.

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Conflicts of Interest

The authors declare no conflicts of interest.

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