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Case Report

Chronic Calcific Pancreatitis: A Case Report from Chennai, India

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ABSTRACT

Diabetes Mellitus (DM) encompasses a spectrum of metabolic disorders, with Type 3c DM, also known as pancreatogenic diabetes mellitus, arising secondary to pancreatic disease. This abstract provides an overview of the diagnostic challenges, clinical features, pathophysiology, and management of Type 3c DM, emphasizing the importance of accurate diagnosis and tailored treatment strategies. Despite its prevalence, Type 3c DM remains significantly underdiagnosed due to overlapping symptoms with type I and Type II DM, complicating the differentiation process. Key diagnostic criteria include evidence of exocrine pancreatic insufficiency, pathological pancreatic imaging, absence of Type I DM-associated autoimmune markers, impaired beta cell function, and reduced incretin secretion. Imaging modalities such as endoscopy, MRI, and ultrasound aid in distinguishing Type 3c DM from other types. Management strategies focus on correcting both exocrine and endocrine pancreatic insufficiency, with insulin therapy serving as a cornerstone. Metformin emerges as a promising first-line therapy, while newer incretin-based agents are contraindicated due to potential pancreatic damage. Patients with Type 3c DM are prone to malabsorption, necessitating pancreatic enzyme and vitamin supplements. Dietary modifications, including a high-fiber, low-fat diet, are recommended to alleviate symptoms and prevent complications. Early detection and tailored management are crucial in reducing the morbidity and mortality associated with Type 3c DM, highlighting the importance of accurate history-taking, appropriate diagnostic tests, and recognition of various risk factors. Standardizing diagnostic criteria and increasing awareness among healthcare providers are essential steps toward improving the diagnosis and management of this often-overlooked form of diabetes mellitus.

Keywords: Type 3c diabetes, Diabetes mellitus, Chronic pancreatitis, Pancreatogenic diabetes mellitus, Exocrine pancreatic insufficiency.

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INTRODUCTION

Diabetes Mellitus (DM) is a metabolic disease characterized by persistent and recurrent hyperglycemia resulting from impaired insulin synthesis and insulin resistance, affecting more than 500 million people worldwide (Vonderau and Desai, 2022). While type I and Type II diabetes are well-known types of the disease, type3c remains significantly underdiagnosed despite being more prevalent than Type I (Riaz, 2021). Based on standard classification criteria Type I DM is characterized by the presence of autoantibodies and insulin requirements which are usually early in onset while Type II diabetes is characterized by the absence of autoantibodies and insulin requirements and the presence of insulin resistance (Hardt *et al*., 2008). Type 3c diabetes mellitus, also termed as pancreatogenic diabetes mellitus, refers to diabetes mellitus secondary to a condition or disease of the exocrine pancreas (Hart *et al*., 2021). Among all the diabetics, approximately 2% of all diabetic patients are diagnosed with Type 3c DM (Riaz, 2021). The pathophysiology of pancreatogenic DM is multifactorial, various factors contributing to the mechanism include loss of mass of pancreatic cells, autoimmunity, local or systemic inflammatory response, genetic mutation in CFTR proteins, deficiency in fat-soluble vitamins such as A, D, E, and K, disruption in the insulin—incretin axis (Śliwińska-Mossoń *et al*., 2023).

In Type 3c diabetes, mechanisms such as inflammation, fibrosis, and hardening of endocrine tissue of the pancreas lead to the destruction of pancreatic acinar cells and islets of Langerhans (Śliwińska-Mossoń *et al*., 2023; Vonderau and Desai, 2022). This causes decreased insulin synthesis, reduced glucagon secretion by alpha cells and decreased pancreatic polypeptide secretion from the PP cells causing hypoglycemia. Whereas hyperglycemia occurs by increased hepatic insulin resistance and decreased hepatic glucose production (Duggan

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et al., 2020). Patients with Type 3c diabetes often have a history of pancreatitis, and can also develop it due to various genetic and nongenetic causes such as chronic pancreatitis (76%), hemochromatosis (8%), pancreatic cancer (9%), cystic fibrosis (4%), and previous pancreatic surgery (3%) (Hardt, Brendel, Kloer, and Bretzel, 2008). Chronic pancreatitis refers to irreversible physiological alterations to the pancreas manifesting as persistent pain, dysglycemia, pancreatic insufficiency and a higher risk of pancreatic cancer. The prevalence of Type 3c diabetes in chronic pancreatitis accounts for about 70%. Causes of chronic pancreatitis include alcoholic pancreatitis, hereditary pancreatitis, autoimmune pancreatitis, traumatic pancreatitis and pancreas divisum (Vonderau and Desai, 2022). The risk of DM in chronic pancreatitis increases with an increase in the duration and severity of the diseases (Śliwińska-Mossoń *et al.*, 2023). Acute recurrent pancreatitis causes repeated parenchymal trauma leading to chronic pancreatitis, while severe episodes of acute pancreatitis can directly lead to the development of Type 3c diabetes. Pancreatic insufficiency remains an early manifestation of Type 3c diabetes while other major symptoms of Type 3c diabetes include diarrhea, abdominal pain, bloating, change inappetite, nausea, vomiting, and general fatigue. Apart from pancreatic disease, patients with Type 3c DM may present similarly to those with type I and Type II diabetes complicating the diagnosis and leading to misdiagnosis and mistreatment (Riaz, 2021; Vonderau and Desai, 2022).

CASE REPORT

A 53-year-old male patient came with the complaints of epigastric pain (3/10) for the past 2 days. The patient also had a history of high-colored urine for the past 2 days and had been experiencing weight loss of about 15 kg in the past 10 years. The patient was initially treated in an outside hospital where ultrasonography abdomen was done which suggested pancreatic intraductal calculi with the largest measuring 8 mm and atrophic pancreas and the patient's amylase and lipase levels were found to be elevated. He is a known case of type II diabetes mellitus, Chronic pancreatitis, and dyslipidemia and has been taking T. Metformin, 500 mg BD, T.Pancreatin 300 mg OD, and T.Atorvas 40 mg HS respectively for the conditions. He underwent Cystoscopy + URSL (ureteroscopic Lithotripsy) 20 years back and a DJ (Double J) Stent (Table 1).

Computed Tomography abdomen suggested chronic calcific pancreatitis with multiple intraductal calculi and mild central intrahepatic biliary radicle dilatation with mildly dilated common bile duct. From the above-mentioned data, the patient was diagnosed to have acute on chronic calcific pancreatitis, benign biliary stricture of common bile duct, and Type 3c diabetes which was initially misdiagnosed as type II diabetes mellitus. Endoscopic retrograde cholangiopancreatography was done and a stent was placed. The patient was prescribed pancreatin, pantoprazole, paracetamol, ondansetron, N-Acetyl cysteine, atorvastatin, tramadol, and an antibiotic sulbactam and cefoperazone combination. For the patient's hyperglycemic state, insulin aspart, regular insulin, insulin glargine, and metformin were prescribed (Table 2).

Once the patient's condition improved and he was in stable condition, he was discharged with antibiotic cefpodoxime and

Table 1: Laboratory investigations.

Parameter	Observed value	Normal value
Hematology		
Hemoglobin	14.7 g/100 mL	14–18 g/100 mL
RBC	5.3×10^6 cells/mm^3	$4.5–6 \times 10^6$ cells/mm^3
PCV	46%	38–54%
MCV	87 fL	80–100 fL
TLC	10,820↑	$4–10 \times 10^3$ cells/mm^3
Neutrophils	85.5%↑	40–60%
Lymphocytes	9.9%↓	20–40%
Eosinophils	0.3%	0–5%
Monocytes	4.1	4–8%
Basophils	0.2%	0–1%
Platelet	261,000 cells/mm^3	1.5–4 L cells/mm^3
Renal function test		
Serum creatinine	0.76 mg/100 mL	0.8–1.3 mg/100 mL
Liver function test		
AST	234 U/L↑	0–40 U/L
ALT	362 U/L↑	0–30 U/L
ALP	241 U/L↑	50–160 U/L
Total bilirubin	6.9 mg/100 mL↑	0.1–1.0 mg/100 mL
Direct bilirubin	3.73 mg/100 mL↑	0.2–0.6 mg/100 mL
Albumin	4.3 g/100 mL	3.5–5.5 g/100 mL
Globulin	2.61 mg/100 mL	1.5–3.5 g/100 mL
Albumin: globulin ratio	1.7↑	1: 1
Prothrombin time	12.4 s	11–14 s
INR	1.02	0.9–1.2
Biochemistry		
CBG	312 mg/100 mL↑	70–140 mg/100 mL
Others		
CRP	5.3 mg/100 mL↑	0.3–1 mg/100 mL
Amylase	118 U/L↑	30–110 U/L
Lipase	250 U/L↑	0–160 U/L

Abbreviations: CBG: Capillary blood glucose.

Table 2: Diabetic chart.

Time	CBG	Drug	Dose
Day 1			
2:00 a.m.	226 mg/100 mL	monitoring	
6:00 a.m.	236 mg/100 mL	Inj. Novorapid	4U
12:00 p.m.	261 mg/100 mL	monitoring	
6:00 p.m.	309 mg/100 mL	T.Metformin	500 mg
Day 2			
6:00 a.m.	287 mg/100 mL	Inj. Actrapid	8U
12:00 p.m.	358 mg/100 mL	Inj. Actrapid	8U
6:00 p.m.	215 mg/100 mL	monitoring	
Day 3			
6:00 a.m.	185 mg/100 mL	Inj. Actrapid	4U
1:00 p.m.	313 mg/100 mL	Inj. Actrapid	10U
6:00 p.m.	260 mg/100 mL	Inj. Actrapid	10U
11:00 p.m.	336 mg/100 mL	Inj. Actrapid	10U
Day 4			
6:00 a.m.	177 mg/100 mL	Inj. Actrapid	8U
12:00 p.m.	245 mg/100 mL	Inj. Actrapid	8U
6:00 p.m.	276 mg/100 mL	Inj. Novorapid	8U
11:00 p.m.	336 mg/100 mL	Inj. Lantus	8U
Day 5			
6:00 a.m.	214 mg/100 mL	Inj. Novorapid	8U
12:00 p.m.	336 mg/100 mL	Inj. Novorapid	15U

Abbreviations: CBG: Capillary blood glucose.

Table 3: Diagnostic criteria.

Major criteria (all must be fulfilled)
1 Evidence of exocrine pancreatic insufficiency.
(FE1 <200 _g/g or incorrect direct function testing).
2 Pathological pancreatic imaging.
(Endoscopic ultrasound, magnetic resonance imaging, and computed tomography).
3 Absence of type I diabetes mellitus-associated autoimmune markers.
Minor criteria
1 Impaired beta cell function (e.g., HOMA-B, C-peptide/glucose-ratio).
2 No excessive insulin resistance (e.g., HOMA-IR).
3 Impaired incretin secretion (e.g., GLP-1, pancreatic polypeptide).
4 Low serum levels of lipid soluble vitamins (A, D, E, and K).

Abbreviations: FE1: Feces elastase 1.

clavulanic acid, pantoprazole, pancreatin, silodosin, multivitamin, tramadol, and acetaminophen combination. Diabetic discharge medications include metformin, 500 mg (1-0-1), insulin aspart (16U-14U-12U), and insulin glargine (0-0-16U). The review was scheduled at the hepatology Outpatient Department (OPD), urology OPD, and internal medicine OPD.

DISCUSSION

Chronic Calcific Pancreatitis (CCP) is an inflammatory disease that affects the pancreas, causing calcification and scarring within the gland. Individuals with chronic pancreatitis should be screened for blood glucose levels and HbA1c levels to assess the diabetes. If diabetes is confirmed, further an autoimmune workup should be done to exclude late onset Type I DM. CCP is one of the common cause for Type 3c DM, which is often misdiagnosed or mislabeled. In this case, initially the patient was incorrectly diagnosed with Type II diabetes (Bahl *et al.*, 2023). The patient showed weight loss of about 15 kg in the last 10 years which is a sign of Type IIIc diabetes, and the patient had chronic calcific pancreatitis which is a major risk factor for Type IIIc diabetes, and from the patient's ultrasonography report, the patient was later correctly diagnosed with Type IIIc diabetes. Hence, Computed Tomography and MRI scans are recommended diagnostic tools for early detection of CCP.

The diagnosis of Type 3c DM presents with greater challenges due to its potential overlapping and ambiguity in distinguishing it from other DM, however, it has a distinct clinical course, including poor glycemic control and a notable higher insulin need (Woodmansey *et al.*, 2017). Longstanding cases of Type I DM and Type II DM also exhibit concurrent exocrine pancreatic insufficiency, further complicating diagnosis (Śliwińska-Mossoń *et al.*, 2023). Additionally, individuals with diabetes face an elevated risk for acute and/or chronic pancreatitis, with Type II DM notably associated with a 1.86–2.89 times higher chance of developing acute pancreatitis based on an extensive retrospective analysis. Furthermore, a bidirectional relationship exists between acute pancreatitis and diabetes mellitus, where patients with acute pancreatitis can also develop type I and Type II DM irrespective of exocrine pancreatic dysfunction (Śliwińska-Mossoń *et al.*, 2023).

Uc*et al.*, highlighted standardizing diagnostic criteria to differentiate Type 3c diabetes from Type I and Type II as a critical research requirement (Duggan *et al.*, 2020).It has been found that about 40% of the inpatients with diabetes of the exocrine pancreas are misdiagnosed as Type II DM. Currently, there are no available valid diagnostic criteria for Type 3c DM (Woodmansey *et al.*, 2017). Diagnostic parameters for Type 3c include basic laboratory tests that are performed in Type II DM:

1. Clinical symptoms of hyperglycemia.
2. Blood glucose, ≥200 mg/100 mL.

3. Fasting glucose ≥126 mg/100 mL.
4. Hba1c >6.5% (śliwińska-mossoń *et al.*, 2023).

It is still up for debate, whether or not all the DM linked to pancreatic disease should be classified as Type 3c DM (Śliwińska-Mossoń *et al.*, 2023). Ewald and Bretzel proposed standard diagnostic criteria, however these were criticized for being difficult to apply clinically (Śliwińska-Mossoń *et al.*, 2023). The proposed criteria are represented in the Table 3.

CONCLUSION

Considering the differential criteria, type 3c presents with distinct characteristics compared to Type II DM, including a deficiency of Pancreatic Polypeptide (PP), whereas in the case of Type II DM high levels of nutrients stimulated pancreatic polypeptide is seen (Riaz, 2021). Various pathological imaging parameters including endoscopy, MRI, and ultrasound can be used to differentiate Type 3c DM from Type I. additionally, the absence of certain autoimmune markers associated with Type 1 DM serves as a differentiating factor between Type I DM and Type 3c DM. minor criteria that can further contribute to the differentiation of type I and Type 3c DM include assessment of β-cell function, insulin resistance, abnormalities in incretin secretion, and lower levels of fat-soluble vitamins (A, D, E, K) (Riaz, 2021). Patients with DM following chronic pancreatitis are subjected to retinopathy screening which revealed an increase in risk for microvascular changes in Type 3c than in type I and Type II due to inadequate glycemic control (Śliwińska-Mossoń *et al.*, 2023). In a study, Type 3c DM patients had normal mean BMI and fewer macrovascular complications compared to Type I DM and Type II DM (Lee *et al.*, 2022).

The treatment approach for Type 3c DM includes correction of both exocrine and endocrine pancreatic insufficiency (Śliwińska-Mossoń *et al.*, 2023). Insulin therapy remains a cornerstone for the management of pancreatogenic pancreatitis, however, there is no established standard insulin regimen. caution is required while dosing insulin and monitoring due to the potential risk for hypoglycemia exists from impaired glucagon function (Śliwińska-Mossoń *et al.*, 2023). There is always a basic need to maintain glucose levels a little greater than the normal level to avoid hyperglycemia (Riaz, 2021).

Metformin emerges as a prominent first-line therapy for Type 3C DM, offering various benefits such as a reduction in daily insulin requirements and exerting reduced risk for the development of pancreatic ductal adenocarcinoma. Additionally, it may increase GLP-1 levels and expression of pancreatic GLP-1 and GLP receptor genes and potentially prolong the survival rate of DM patients without pancreatic cancer metastasis (Woodmansey *et al.*, 2017). Other pharmacological agents such as sulfonylureas, thiazolidinediones, DPP-4 inhibitors, GLP-1 analogues and α- glycosidase inhibitors can also be used for the treatment of endocrine dysfunction (Śliwińska-Mossoń *et al.*, 2023). Due to the potential risk for pancreatic damage, newer incretin-based agents such as glucagon-like peptide receptor agonists and dipeptidyl peptidase-4 inhibitors are contraindicated in type 3c patients (Woodmansey *et al.*, 2017).

Type 3c patients are prone to malabsorption secondary to pancreatic exocrine dysfunction than in type I and Type II. hence, pancreatic enzyme and vitamin D supplements are provided to prevent malnutrition and osteoporosis (Woodmansey *et al.*, 2017). In addition, the presence of malabsorption, alcohol consumption, and poor diet complicates the management of the disease (Duggan *et al.*, 2020). Indeed, dietary management also plays an essential role in reducing the symptoms and complications associated with chronic pancreatitis, patients are advised to adhere to a diet rich in fiber and low in fat to alleviate symptoms of steatorrhea and prevent hyperglycemia (Riaz, 2021).

Type 3c DM, arising from the pancreatic disease, is often misdiagnosed or overlooked, contributing to poor patient outcomes. Patients with chronic pancreatitis face an elevated risk of developing diabetes, particularly with development of pancreatic ductal calcification and exocrine insufficiency, indicating the progression of disease. Accurate history taking, appropriate diagnostic tests and recognizing various risk factors are essential identification for Type 3c DM, which aids in timely detection and management, potentially reducing complications and mortality.

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ABBREVIATIONS

DM: Diabetes mellitus; **URSL:** (Ureteroscopic Lithotripsy) - Computed Tomography; **CBD:** Common Bile Duct; **ERCP:** Cholangiopancreatography; **OPD:** Outpatient department; **CCP:** Chronic calcific pancreatitis; **DDP:** Dipeptidyl peptidase 4; **GLP:** Glucagon-like peptide.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

PATIENT CONSENT

The patient has been informed about the publishing and assured that the information will only be used for scientific and research purposes and identity of patient will not be disclosed.

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