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A Case Report on Type 3c Diabetes Mellitus

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HUMAN



Karra Geetha^{*1}, Kotti ramya sree², Udtha laxmi sahithya², Chunchu Smaigdhini², T. Rama Rao³

¹ Associate Professor, Department of Pharmaceutics, CMR College of Pharmacy, Hyderabad, Telangana, India.

² Department of Pharm D, CMR College of Pharmacy, Hyderabad, Telangana, India.

³ Principal, CMR College of Pharmacy, Hyderabad, Telangana, India.

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ABSTRACT

Objective: This case report aims to present a comprehensive analysis of a patient diagnosed with Type 3c diabetes mellitus, focusing on the unique clinical manifestations, diagnostic challenges, and the management approach employed. Methods: We conducted a thorough review of the patient's medical history, diagnostic tests, and treatment interventions. Special attention was given to the identification of etiological factors contributing to the development of Type 3c diabetes mellitus. Results: A 38year male patient was admitted to general medicine, presented with shortness of breath for 2 days, highgrade fever for 2days with chills and rigors, burning sensation in chest on food intake for 2 days, cough with sputum, cold. The patient has a known history of diabetes mellitus and history of pancreatitis and alcoholic. Diagnostic assessments revealed GRBS measured a very high blood glucose level exceeding 600mg/dl, patient laboratory finding: urine ketone test reported 3mmol/L, Arterial blood gas analysis reported as pH-6.9(\downarrow), pCO₂-20.3mmHg (\downarrow), HCO₃-4.1mmol/L and is measured frequently and ultra sound scan of abdomen shows pancreatitis, and upon regular monitoring of glucose readings (GRBS), the values were observed to be >500mg/dl, 290mg/dl, 259mg/dl, 237mg/dl, 202mg/dl. Based on this evaluation the patient was diagnosed as chronic pancreatitis with Type 3c diabetes mellitus presenting with severe acidosis (DKA). The report discusses the intricacies of distinguishing Type 3c DM from other forms and highlights the challenges encountered during the diagnostic process. Conclusion: Pancreatogenesis diabetes, recognized as type 3C diabetes, is a form of diabetes mellitus intricately linked to conditions affecting the exocrine pancreas. This case report underscores the importance of recognizing and appropriately managing Type 3c diabetes mellitus. In patients with Type 3c DM the diagnosis is often mistaken with type 2DM and diagnostic criteria plays a major role. In this case the diagnosis is made using criteria and treatment is given and the glucose levels are closely monitored.

INTRODUCTION:

Diabetes mellitus is characterized by a metabolic regulatory dysfunction that leads to persistent high levels of blood sugar. This condition can arise from a combination of factors, including compromised insulin secretion, increased insulin resistance, or a combination of both. Due to this diverse range of contributing factors, diabetes mellitus is presently categorized into various types, encompassing types 1 through 4[1]. Physicians and the general population tend to have a strong understanding of type 1 and type 2 diabetes mellitus. There are numerous research groups and extensive guidelines available for the diagnosis and treatment of these types. However, diabetes mellitus that arises as a result of pancreatic diseases (type 3c) is often overlooked in everyday medical practice. Surprisingly, recent data on type 3c diabetes (T3cDM) suggests that it might be more prevalent than commonly believed. Studies also suggest that this clinically significant condition may be consistently underdiagnosed and misdiagnosed [2]. Diabetes can also arise as a direct consequence of other medical conditions, including those affecting the exocrine pancreas. In the past, diabetes resulting from exocrine pancreatic diseases was termed pancreatogenesis or pancreatogenesis diabetes mellitus. However, contemporary literature now designates it as type 3c diabetes. The introduction of this term is credited to an annual table released by the American Diabetes Association until 2014. This table categorized diabetes into four general types, with III.C specifically denoting diabetes stemming from exocrine pancreatic diseases. Some authors have interchangeably referred to it as type IIIC diabetes mellitus and type 3c diabetes mellitus [1].

Classification of causes of type 3c diabetes grouped according to their potential mechanisms:

Congenital or acquired complete absence of islets:

- a Pancreatic agenesis
- b Pancreatectomy (total)

Acquired partial absence of functional islets:

- c Chronic pancreatitis*
- d Pancreatectomy (partial)

- e Severe acute pancreatitis
- f Cystic fibrosis
- g Haemochromatosis

Paraneoplastic:

h Pancreatic ductal adenocarcinoma

Other:

i Transient hyperglycemia of acute pancreatitis

Pancreatogenesis diabetes, also known as type 3C diabetes, is categorized as diabetes mellitus linked to conditions affecting the exocrine pancreas. It exhibits a distinct underlying physiological mechanism and is associated with both non-cancerous and cancerous disorders of the exocrine pancreas. [3,4,5]

Around 80% of cases of pancreatogenesis diabetes are a result of underlying chronic pancreatitis (CP) [3,4,5]. CP is a gradual and damaging condition that affects both the exocrine and endocrine functions of the pancreas.

A particular study suggests that in cases of alcoholic chronic pancreatitis (CP), diabetes may emerge in approximately 83% of patients within 25 years from the initial clinical presentation. Moreover, more than half of these diabetic patients may ultimately require insulin-dependent treatment [6]. Risk factors associated with the development of diabetes linked to CP (referred to as CP-DM) encompass early onset of CP, the presence of pancreatic calcification, a history of distal pancreatectomy, concurrent liver cirrhosis, a history of smoking[7], and other established risk factors for type 2 diabetes[8] Interestingly, it has been observed that the incidence of diabetic retinopathy and other complications affecting both microvascular and macrovascular systems in CP-DM is similar to what is observed in both type 1 and type 2 diabetes.[9,10,11].

PATHOPHYSIOLOGY: Endocrine function in chronic pancreatitis

CP-DM develops as a result of islet cell loss, which is accompanied by fibrosis and inflammatory damage to the pancreatic parenchyma. In early CP, islets are resistant to

destruction, but as the game progresses, they lose their resistance. [12,13,14]. CP-DM frequently presents as "brittle diabetes" with poor glucose control due to an altered and complicated endocrine physiology [15]. Although beta cell dysfunction is the primary effect of islet loss, other cell types, including alpha, delta, and pancreatic polypeptide (PP) cells, may also be impacted in CP.

CLINICAL FEATURES AND DIAGNOSIS

Patients are frequently misclassified and T3cDM diagnosis is frequently missed. Several T3cDM patients were incorrectly identified as having type 2 diabetes. Like other kinds of diabetes, the diagnosis of CP-DM is mostly based on clinical presentation and biochemical tests. Pancreatic exocrine insufficiency would be seen in the majority of CP-DM patients [8].If the patient is in a catabolic condition, overt hyperglycaemia may show up as polyuria, polydipsia, polyphagia, and fast weight loss. Due to the prevalence of metabolic dysfunction and clinically asymptomatic mild to moderate hyperglycaemia, frequent and periodic screening for diabetes or glucose intolerance status using fasting glucose and hemoglobin A1C measurements is crucial for the management of CP patients. Testing should be done at least once a year.

Criteria used:

Proposed Major Criteria (all must be met):

- Confirmation of exocrine pancreatic insufficiency through either the monoclonal fecal elastase-1 test or direct function assessments.

- Identification of abnormal pancreatic features through procedures like endoscopic ultrasound, MRI, or CT scans.

- Lack of autoimmune markers associated with type 1 diabetes mellitus.

Minor Criteria:

- Reduced beta cell function (e.g., assessed by HOMA-B, C-peptide/glucose ratio)
- Absence of excessive insulin resistance (e.g., evaluated by HOMA-IR)
- Compromised incretin secretion (e.g., GLP-1, pancreatic polypeptide).

- Reduced serum levels of fat-soluble vitamins (A, D, E, and K). [16,17,18]

CASE PRESENTATION:

A 38year male patient was admitted to general medicine with chief complaints of A 38year male patient was admitted to general medicine On physical examination patient is conscious and coherent and blood pressure measured as 86/80mmHg, pulse rate-92bpm, CVS-S1S2(+),RS-BAE(+),tachypnoea(+) and GRBS measured a very high blood glucose level exceeding 600mg/dl. The patient laboratory finding: urine ketone test reported 3mmol/L, Arterial blood gas analysis reported as pH-6.9(\downarrow), pCO₂-20.3mmHg (\downarrow),HCO₃-4.1mmol/L and is measured frequently and ultra sound scan of abdomen shows pancreatitis, and upon regular monitoring of glucose readings (GRBs),the values were observed to be >500mg/dl, 290mg/dl, 259mg/dl, 202mg/dl. Based on this evaluation the patient was diagnosed as chronic pancreatitis with type 3c diabetes mellitus presenting with severe acidosis (DKA).

The patient has a medical history of type 2 DM and pancreatitis but is non-compliant with prescribed medications. No history of surgical history and allergic history. The patient was managed with insulin of 40 units in 39 cc of normal saline at a rate of 6 cc per hour until glucose levels reached 250 mg/dL. Subsequently, the infusion is tapered to 3 cc per hour with 5% dextrose in normal saline. The prescribed treatment involves intravenous infusion of 5 sodium bicarbonate ampoules in 100 ml normal saline over 20 minutes. Additionally, the patient is prescribed 5 ml potassium chloride syrup and a 500 mg paracetamol tablet. The arterial blood gases and blood glucose levels are frequently monitored and recorded. On treatment, the acidosis is subsided.

DISCUSSION:

Pancreatogenesis diabetes, recognized as type 3C diabetes, is a form of diabetes mellitus intricately linked to conditions affecting the exocrine pancreas. This category of diabetes manifests a unique underlying physiological mechanism and is associated with disorders of the exocrine pancreas, both non-cancerous and cancerous. [3,4,5]

The predominant cause, constituting approximately 80% of cases, is chronic pancreatitis (CP). [3,4,5]

Noteworthy risk factors contributing to the development of diabetes associated with CP (referred to as CP-DM) include early onset of CP, the presence of pancreatic calcification, a history of distal pancreatectomy, concurrent liver cirrhosis, and a smoking history [7].

The pathogenesis of CP-DM is characterized by islet cell loss, accompanied by fibrosis and inflammatory damage to the pancreatic parenchyma. In the early stages of CP, islets display resistance to destruction; however, as the disease progresses, this resistance diminishes. The diagnosis of CP-DM primarily relies on clinical presentation and biochemical tests. Given the prevalence of metabolic dysfunction and clinically asymptomatic mild to moderate hyperglycaemia, it is imperative to conduct frequent and periodic screenings for diabetes or glucose intolerance status. These screenings involve the use of fasting glucose and hemoglobin A1C measurements, providing crucial insights for the effective management of CP patients. [12,13,14]

In this case the patient has history of type 2 diabetes mellitus and pancreatitis and noncompliant with medications and has increased glucose levels above 600mg/dl and diagnosed with type 3c DM with pancreatitis and is prescribed with insulin according to the glucose level, and DKA is monitored by ABG test and prescribed with sodium bicarbonate. This case was diagnosed by ultrasound scan and preliminary tests like urine ketones and arterial blood gases test, complete blood picture, RFT, LFT. The patient was treated with insulin and sodium bicarbonate for 2 weeks and there is progressive in the condition.

CONCLUSION:

In patients with type 3c DM the diagnosis is often mistaken with type 2DM and diagnostic criteria plays a major role. In this case the diagnosis is made using criteria and treatment is given and the glucose levels are closely monitored.

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