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Fibrocalculous Pancreatic Diabetes (FCPD) Presenting with Severe Hyponatremia in 58-Year-Old Male: A Case Report

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ABSTRACT

Fibrocalculous Pancreatic Diabetes (FCPD) is a rare, tropical form of secondary diabetes mellitus caused by chronic calcific pancreatitis, predominantly seen in developing countries. It is characterized by pancreatic ductal calculi, exocrine insufficiency, and insulin-requiring diabetes with relative ketosis resistance. We report a case of a 58-year-old non-alcoholic male from West Bengal, India, who presented with severe hyponatremia (serum sodium 117 mEq/L) and was found to have poorly controlled diabetes (HbA1c 8.2%) along with imaging evidence of chronic calcific pancreatitis — dilated main pancreatic duct with multiple calculi and parenchymal calcification on both ultrasonography and CECT abdomen. The patient was also found to have microalbuminuria, normocytic anemia, and echogenic floaters in the urinary bladder. This case highlights the occurrence of FCPD in an older patient and the importance of recognizing this entity in the differential diagnosis of diabetes with abdominal imaging abnormalities in tropical settings.

Keywords: Fibrocalculous Pancreatic Diabetes; FCPD; Chronic Calcific Pancreatitis; Tropical Diabetes; Hyponatremia; Microalbuminuria

INTRODUCTION

Fibrocalculous Pancreatic Diabetes (FCPD) is a unique form of diabetes mellitus classified by the World Health Organization (WHO) as a secondary or 'other specific type' of diabetes, distinct from Type 1 and Type 2 diabetes. It represents the diabetic manifestation of Tropical Chronic Pancreatitis (TCP), a disease predominantly seen in tropical and developing countries, including India, Sri Lanka, Bangladesh, and parts of sub-Saharan Africa. The disease is characterized by chronic fibrosis and calcification of the pancreas leading to both endocrine and exocrine insufficiency.

FCPD classically presents in younger, lean, malnourished individuals with a history of recurrent epigastric pain, steatorrhoea, and insulin-dependent diabetes without ketosis. However, atypical presentations — including older

patients and those with relatively preserved nutritional status — have increasingly been reported. The pathognomonic feature is the presence of pancreatic calculi on imaging, alongside diabetes diagnosed by standard WHO criteria, in a patient from a tropical region, with no identifiable alternative etiology for chronic pancreatitis such as chronic alcohol use or primary hyperparathyroidism.

We present a case of FCPD in a 58-year-old non-alcoholic male from West Bengal, India, who presented with severe hyponatremia as the primary presenting abnormality, with incidental discovery of chronic calcific pancreatitis and poorly controlled diabetes on investigation. This case underscores the atypical age of onset and the importance of comprehensive metabolic and imaging workup in hospitalized diabetic patients.

CASE PRESENTATION

Patient Information

A 58-year-old non-alcoholic male, Mr. Nitai Barik (UHID: KPC/170326/0003, IPID/170326/0014), a resident of West Bengal, India, was admitted to the ICU of KPC Medical College and Hospital, Jadavpur, Kolkata, under the care of Dr. Mayur Bahan Mukherjee in the Department of General Medicine on 17th March 2026.

Chief Complaints and History

The patient presented with symptoms warranting ICU admission. He had a history consistent with diabetes mellitus and reported episodes of abdominal discomfort. He denied chronic alcohol consumption. There was no history suggestive of gallstone disease, primary hyperparathyroidism, or autoimmune pancreatitis. There was no family history of pancreatic disease.

Clinical Examination

On examination, the patient was hemodynamically assessed and admitted to the ICU (Bed ICU-4/1). General examination revealed pallor suggestive of anemia. There were no signs of jaundice, cyanosis, or lymphadenopathy. Abdominal examination findings prompted further imaging workup. No features of acute pancreatitis (peritonism, guarding) were noted on clinical examination at the time of imaging.

INVESTIGATIONS

A. Haematology (20th March 2026 — Complete Haemogram)

Parameter	Result	Reference Range	Interpretation
Haemoglobin	11.3 g/dl	13–17 g/dl	Low (Mild Anaemia)
RBC Count	3.58 mill/cumm	4.5–5.5 mill/cumm	Low
Haematocrit	30.6%	40–50%	Low
MCV	85.5 fl	80–100 fl	Normal (Normocytic)

MCH	31.6 pg	27–32 pg	Normal
MCHC	36.9%	31.5–34.5%	Mildly elevated
TLC	10720/cumm	4000–11000/cumm	Upper normal
Platelet Count	2.4 Lac/cmm	1.5–4.0 Lac/cmm	Normal
Neutrophil	84%	40–80%	Neutrophilia
Lymphocyte	10%	20–40%	Low
ESR	32 mm/hr	1–12 mm/hr	Elevated
RDW	11.7%	11–15%	Normal

B. Biochemistry — Serum Electrolytes & Metabolic Panel

Test	Date	Result	Reference	Interpretation
Sodium	17-Mar-2026	117 mEq/L	135–148 mEq/L	Severe Hyponatremia
Potassium	17-Mar-2026	3.3 mEq/L	3.5–5.5 mEq/L	Mild Hypokalemia
Sodium (repeat)	19-Mar-2026	139 mEq/L	135–148 mEq/L	Corrected to Normal
Potassium (repeat)	19-Mar-2026	3.5 mEq/L	3.5–5.5 mEq/L	Normal
Serum Lipase	19-Mar-2026	11 U/L	10–140 U/L	Lower normal (exocrine insufficiency)
Serum Amylase	19-Mar-2026	34 U/L	28–100 U/L	Lower normal (exocrine insufficiency)
Calcium	19-Mar-2026	9.7 mg/dl	9.6–10.3 mg/dl	Normal
Magnesium	19-Mar-2026	2.2 mg/dl	1.9–2.5 mg/dl	Normal

C. Glycaemic Control — HbA1c (17th March 2026)

HbA1c (Glycosylated Haemoglobin by HPLC): 8.2% — In the diabetic range (>6.4%), confirming poorly controlled diabetes mellitus with chronic hyperglycaemia over the preceding 2–3 months.

D. Urine Investigations

Test	Result	Interpretation
Urine Routine (17-Mar)	Slightly hazy, pH 6.0, Sp. Gravity 1.010, Protein Nil, Glucose Nil, Pus Cells 2–3/hpf	Low specific gravity; no frank infection
Urine Culture (17-Mar)	No growth after 48 hours	Sterile — no urinary tract infection
Microalbumin Creatinine Ratio (17-Mar)	228.50 mg/g creatinine (Microalbumin: 39.6 mg/L)	Microalbuminuria (ref: 30–300 = microalbuminuria) — Diabetic nephropathy
Urine Spot Sodium (17-Mar)	54 mmol/L	Low — consistent with hypovolemia/renal sodium retention

E. Stool Routine Examination (21st March 2026)

Semi-solid, yellowish stool with mucus present. Microscopy showed pus cells 1–2/hpf, RBC 0–1/hpf, vegetable cells 1–2/hpf. No fat globules, muscle cells, or starch noted. No ova, cysts, or parasites detected. Reaction: Acidic. The absence of overt steatorrhoea (fat globules) in the stool examination does not exclude exocrine insufficiency, as mild forms may not manifest fat globules on routine microscopy.

F. Imaging Studies

Ultrasonography — Whole Abdomen (19–20th March 2026):

USG abdomen revealed a heterogeneous atrophic pancreas with a markedly dilated main pancreatic duct measuring 10 mm in diameter containing multiple calculi within the duct lumen. The common bile duct (CBD) was mildly prominent at 7 mm. The liver was normal in size (141 mm) with normal echogenicity. The gallbladder showed no calculi and normal wall thickness. Both kidneys were normal in size and echogenicity with no hydronephrosis or calculus. Echogenic floaters were noted in the urinary bladder lumen. Impression: Heterogeneous atrophic pancreas with dilated main pancreatic duct containing multiple calculi — ? Acute on Chronic Calcific Pancreatitis; mildly prominent CBD; echogenic floaters in urinary bladder.

CECT — Whole Abdomen with Oral and IV Contrast (20th March 2026):

CECT of the whole abdomen confirmed chronic calcific pancreatitis with its sequelae. The main pancreatic duct was dilated measuring 3–5 mm. Multiple calculi within the duct system and parenchymal calcification were seen. Atrophic changes were noted in the pancreatic parenchyma. Additionally, the liver showed fatty infiltration with a small non-enhancing cystic focus of 6 mm in the right lobe. The gallbladder was hyperdistended with a clear lumen and no pericholecystic fluid. The extra-hepatic biliary tree was not dilated. Both kidneys were normal; no calculi detected. No retroperitoneal lymphadenopathy or ascites. Final

Impression: (1) Fatty infiltrate in liver with a small non-enhancing cystic focus in the right lobe; (2) Chronic calcific pancreatitis with its sequelae.

DIAGNOSIS

Based on the clinical presentation, laboratory investigations, and imaging findings, the diagnosis of Fibrocalculous Pancreatic Diabetes (FCPD) was established. The patient fulfilled the following WHO diagnostic criteria for FCPD:

1. Occurrence in a tropical country: The patient is from West Bengal, India — a tropical region with well-documented prevalence of FCPD.
2. Diabetes mellitus by standard WHO criteria: HbA1c of 8.2% confirms poorly controlled diabetes.
3. Evidence of chronic pancreatitis:

a) Pancreatic calculi demonstrated on both USG and CECT abdomen — pathognomonic of FCPD.
b) Abnormal pancreatic morphology: Heterogeneous atrophic pancreas on USG; atrophic parenchyma with parenchymal calcification on CECT.
c) Dilated main pancreatic duct: 10 mm on USG and 3–5 mm on CECT.
d) Low-normal serum amylase (34 U/L) and lipase (11 U/L) — consistent with pancreatic exocrine insufficiency.

4. Absence of other causes of chronic pancreatitis: No history of chronic alcohol use; serum calcium normal (9.7 mg/dl) ruling out hyperparathyroidism; no hepatobiliary obstruction.

Additional findings of microalbuminuria (ACR 228.50 mg/g creatinine) indicate early diabetic nephropathy, a recognized microvascular complication of FCPD.

DISCUSSION

FCPD is a form of pancreatogenic diabetes that is relatively underrecognized, particularly in older patients. It was classified by the WHO as 'malnutrition-related diabetes mellitus' in 1985 and later reclassified as a distinct entity under 'other specific types' of diabetes. The disease is endemic to tropical developing countries, and India, particularly South India and parts of Eastern India including West Bengal, contributes significantly to the global burden of FCPD.

Classically, FCPD presents in young (10–40 years), underweight, malnourished individuals with a history of recurrent abdominal pain and steatorrhoea. However, as demonstrated in this case, FCPD can present atypically in older patients (58 years) without overt malnutrition or steatorrhoea. This late-onset or atypical presentation may lead to initial misdiagnosis as Type 2 diabetes, particularly when a thorough abdominal imaging workup is not performed at the outset. The absence of frank steatorrhoea in this case may be attributed to dietary fat

restriction, mild exocrine insufficiency, or advanced exocrine gland atrophy with minimal residual secretory activity.

The primary presenting finding in this case was severe hyponatremia (serum sodium 117 mEq/L), which was subsequently corrected to 139 mEq/L with appropriate management. The coexistence of a low urine spot sodium (54 mmol/L) suggests hypovolemic or euvolemic hyponatremia — likely related to either SIADH in the context of systemic illness or volume depletion with hypotonic fluid replacement. SIADH as a cause of severe hyponatremia is well documented in chronic pancreatitis and systemic inflammatory states. Hyponatremia in the context of FCPD and chronic pancreatitis has been infrequently highlighted in the literature, making this an educationally important aspect of this case.

Serum amylase and lipase were in the low-normal range (34 U/L and 11 U/L respectively), consistent with significant exocrine pancreatic atrophy in chronic calcific pancreatitis, where the residual pancreatic acinar mass is insufficient to produce markedly elevated enzyme levels during acute exacerbations — a well-recognized phenomenon in 'burnt-out' chronic pancreatitis. The serum calcium was normal at 9.7 mg/dl, effectively ruling out primary hyperparathyroidism as an etiology of pancreatitis, and the serum magnesium was also within the normal range.

The microalbumin-to-creatinine ratio of 228.50 mg/g creatinine places this patient in the microalbuminuria range (30–300 mg/g), indicating early diabetic nephropathy — a recognized microvascular complication of FCPD. Notably, while macrovascular complications are less common in younger FCPD patients, older FCPD patients like the present case may have accumulated microvascular complications due to prolonged hyperglycaemia, as evidenced by the elevated HbA1c of 8.2%.

The hematological findings of normocytic anaemia (Hb 11.3 g/dl, MCV 85.5 fl) with elevated ESR (32 mm/hr) and neutrophilia (84%) suggest an underlying chronic inflammatory or systemic state, consistent with the inflammatory milieu of chronic pancreatitis and its systemic complications.

The stool examination, while not showing frank fat globules or muscle fibres suggestive of severe steatorrhoea, showed the presence of mucus and mild pus cells. Acidic stool reaction is also consistent with carbohydrate malabsorption in the context of exocrine pancreatic insufficiency.

Management of FCPD is multifaceted and involves: (1) glycaemic control — typically with insulin, as patients with FCPD are insulin-requiring and ketosis-resistant due to concomitant glucagon deficiency from alpha-cell destruction; (2) pancreatic enzyme replacement therapy (PERT) for exocrine insufficiency; (3) pain

management; (4) nutritional rehabilitation; and (5) regular surveillance for pancreatic malignancy, which is the most dreaded long-term complication of FCPD/TCP, with an estimated 5–10% lifetime risk.

CONCLUSION

This case highlights the atypical presentation of Fibrocalculous Pancreatic Diabetes in an older patient from West Bengal, India, presenting with severe hyponatremia as the primary metabolic derangement. The constellation of chronic calcific pancreatitis on imaging (pancreatic ductal calculi, dilated main pancreatic duct, atrophic parenchyma), poorly controlled diabetes (HbA1c 8.2%), microalbuminuria indicating early diabetic nephropathy, and absence of alternative etiologies for chronic pancreatitis fulfils the WHO diagnostic criteria for FCPD. Clinicians in tropical settings must maintain awareness of FCPD in any diabetic patient — irrespective of age — who has imaging evidence of chronic calcific pancreatitis, to ensure accurate diagnosis, appropriate endocrine and exocrine management, and timely surveillance for pancreatic malignancy.

DECLARATIONS

Ethics: Written informed consent was obtained from the patient for publication of this case report and accompanying investigation images.

Conflicts of Interest: The authors declare no conflicts of interest.

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