International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 32, Issue 5, May 2026 ijppr.humanjournals.com ISSN: 2349-7203

Type 3c (Pancreatogenic) Diabetes Mellitus Secondary to Chronic Pancreatic Disease: A Case Series

Sumangala V*[1], B Sneha [1], E P Akshitha[1], Manasa A[2]and Dr. Syed Mohammed Hussaini[3]

VI Pharm D[1], V Pharm D[2] and Junior Doctor, Sehat Hospital, Ballari[3]
Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104 India.

Received: 27 March 2026 Revised: 22 April 2026 Accepted: 28 April 2026

ABSTRACT

Introduction: Type 3c diabetes mellitus (T3cDM), also known as pancreatogenic diabetes, is a secondary form of diabetes resulting from diseases of the exocrine pancreas. It is frequently underdiagnosed or misclassified as type 2 diabetes mellitus, leading to delayed or inappropriate management. Chronic pancreatitis is the most common underlying cause and is associated with complex disturbances in glucose metabolism, pancreatic enzyme deficiency, and nutritional impairment.**Case Presentation:-** We report a case series of three patients with diabetes secondary to chronic pancreatic disease. The first case involved a 48-year-old male with chronic calcific pancreatitis presenting with uncontrolled hyperglycaemia (random blood glucose 580 mg/dL) and exocrine pancreatic insufficiency. The second case was a 51-year-old female with chronic calcific pancreatitis and cardiovascular comorbidities who presented with abdominal pain, vomiting, and marked glycaemic instability. The third case described a 34-year-old male with a history of pancreatic surgery and long-standing diabetes presenting with diabetic ketoacidosis and poor glycaemic control. All patients demonstrated features consistent with T3cDM, including pancreatic structural abnormalities, elevated pancreatic enzymes, poor glycaemic control, and the need for insulin therapy.**Discussion:-** These cases highlight the diagnostic challenges of T3cDM and emphasize the importance of recognizing pancreatic pathology as an underlying cause of diabetes. Appropriate diagnosis allows for targeted management, including insulin therapy and pancreatic enzyme replacement.**Conclusion**:- Early recognition of type 3c diabetes mellitus is essential to optimize glycaemic control, prevent complications, and improve patient outcomes. Increased clinical awareness can reduce misclassification and support individualized treatment strategies**.**

Keywords:- Chronic pancreatitis; Case series; Exocrine pancreatic insufficiency; Pancreatogenic diabetes; Type 3c diabetes mellitus.

INTRODUCTION:

Diabetes mellitus is a disorder in which the body fails to properly control blood glucose levels, leading to sustained hyperglycaemia.

➢ **Types of diabetes**:

- **Type 1 DM** - This is a form of diabetes characterized by absolute insulin deficiency due to autoimmune destruction of pancreatic β-cells.
- **Type 3c DM** - It is a dysfunction of the pancreas affects endocrine hormone production globally rather than selectively.
- **Type 2 DM** - In T2DM, insulin secretion may be preserved initially, but its biological effectiveness is reduced.[1]

➢ **TYPE 3C DM**

- Type 3c diabetes is a secondary form of diabetes caused by pathological conditions of the pancreas, including inflammatory, genetic, and malignant diseases[2].

- Damage to pancreatic tissue in these disorders disrupts the normal function and survival of insulin-producing β-cells, leading to impaired insulin secretion and the subsequent development of diabetes[3].

- The well-established link between chronic pancreatitis and diabetes has been central to the recognition and classification of this condition as pancreatogenic or type 3c diabetes mellitus.[4].

➢ EPIDEMOLOGY :

Epidemiological data suggest that type 3c diabetes comprises roughly 2% of all diabetes mellitus diagnoses.Approximately one in fifty patients with diabetes mellitus is affected by type 3c diabetes[5].

➢ ETIOLOGY :

1. Type 3c diabetes mellitus develops due to combined exocrine pancreatic damage and β-cell loss, leading to impaired insulin secretion.

2. Reduced insulin receptor expression and dysfunction of GLUT-2 transporters contribute to abnormal glucose regulation in T3cDM.

3. Pancreatic fibrosis and scarring of the islets result in decreased incretin hormone levels.

4. Insufficient pancreatic enzyme production further exacerbates metabolic dysregulation in type 3c diabetes.

➢ SYMPTOMS :

Figure 1. Symptoms of type 3C Diabetes Mellitus[10]

1. Reduced pancreatic enzyme secretion

2. Impaired digestion and nutrient absorption

3. Marked fluctuations between hypoglycemia and hyperglycemia

4. Intermittent fat malabsorption (steatorrhea)

5. Abnormal glucose tolerance[6]

International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 32, Issue 5, May 2026 ijppr.humanjournals.com ISSN: 2349-7203

➢ RISK FACTORS

1. Pancreatic exocrine disease: Substantial risk of low bone mineral density (BMD) depending on the type of exocrine pancreatic disease, with exocrine pancreatic insufficiency (EPI) often preceding or more pronounced.

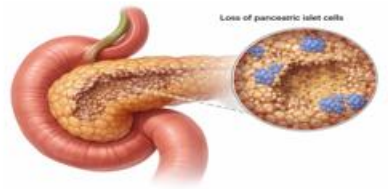
2. Malnutrition and nutrient deficiencies: Common, particularly deficiency of fat-soluble vitamins linked to EPI and poor diet.

3. Overweight despite muscle depletion: Uncommon overall, but observed in some cases with muscle wasting.

4. Brittle diabetes features: Mild to severe hyperglycemia and common severe hypoglycemia, tied to low insulin, glucagon, and pancreatic polypeptide levels[7].

➢ PATHOPHYSIOLOGY

The development of type 3c diabetes mellitus is characterized by a gradual decline in insulin secretion.



This reduction in insulin output may occur due to a loss in the number of pancreatic islet cells.

Alternatively, insulin deficiency can result from β-cell dysfunction caused by pancreatic fibrosis or sclerosis.

Progressive structural injury to pancreatic tissue contributes directly to endocrine insufficiency.

Disruption of the normal functional relationship between exocrine acinar cells and endocrine islets plays a key role in the pathogenesis of T3cDM[8].

Figure 2. Pathophysiology of type 3C DM[11]

➢ DIAGNOSIS

- **Major diagnostic criteria (all required):**

1. Confirmed pancreatic exocrine insufficiency, demonstrated by reduced fecal elastase-1 levels or abnormal direct pancreatic function tests
2. Abnormal pancreatic imaging, showing structural or pathological changes on CT, MRI, or endoscopic ultrasound
3. Absence of autoimmune markers associated with type 1 diabetes mellitus

- **Minor supportive criteria:**

1. Impaired β-cell function, indicated by reduced C-peptide levels or abnormal HOMA-B indices
2. Lack of significant insulin resistance, as assessed by HOMA-IR
3. Defective incretin hormone secretion, including reduced GLP-1 or pancreatic polypeptide levels.
4. Deficiency of fat-soluble vitamins (A, D, E, and K), reflecting malabsorption due to exocrine dysfunction[9].

➢ COMPLICATION

1. Microvascular complications:

✓ These include diabetic retinopathy, nephropathy, neuropathy, and other microangiopathic complications.

2. Misclassification : This can delay appropriate management, leading to progression of eye, nerve, and kidney damage.

3. Acute metabolic complications:

✓ Data on acute complications such as hypoglycaemia and diabetic ketoacidosis are limited.

✓ In patients with chronic pancreatitis, insulin therapy is associated with a high risk of hypoglycaemia, including severe episodes.

4. Influence of additional risk factors :

✓ Factors such as hyperlipidaemia, hypertension, smoking, and obesity may contribute to target organ complications and should be evaluated alongside T3cDM.

5. Macrovascular complications :

✓ Appear to be less common in T3cDM, possibly due to reduced dietary intake and pancreatic exocrine insufficiency.

6. Risk of pancreatic ductal adenocarcinoma (PDAC):

✓ Both diabetes mellitus and chronic pancreatitis independently increase PDAC risk[8].

➢ TREATMENT

NON PHARMACOLOGICAL TREATMENT

1. Regular, balanced meals; avoid skipping meals
2. Controlled carbohydrate intake, prefer high-fibre foods
3. Limit added sugars and sugary drinks

4. Low-fat diet to improve digestion and reduce steatorrhea
5. Adequate protein intake to prevent malnutrition
6. Regular physical activity (moderate exercise)
7. Maintain healthy body weight; avoid rapid weight loss
8. Alcohol avoidance due to hypoglycemia risk
9. Smoking cessation
10. Regular blood glucose monitoring
11. Diabetes education and lifestyle awareness[1]

PHARMACOLOGICAL THERAPY

1. INSULIN THERAPY

Insulin is required in patients with significant insulin deficiency or advanced T3cDM.

It helps achieve adequate glycemic control by replacing deficient endogenous insulin.

Insulin therapy is especially important when oral hypoglycaemic agents fail to maintain glucose control.

2. DPP-IV inhibitors

DPP-IV inhibitors may be considered in selected patients to improve glycemic control.

Their role in T3cDM remains limited due to insufficient clinical evidence.

Further studies are needed to confirm their safety and efficacy in T3cDM.

3. Pancreatic enzyme replacement therapy (PERT)

PERT is essential for managing pancreatic exocrine insufficiency in T3cDM.

It improves digestion, nutrient absorption, and helps stabilize glycemic control.

PERT also supports overall nutritional status and reduces steatorrhea.

4. Vitamin D supplementation

Supplementation is recommended due to low serum levels of fat-soluble vitamins, commonly observed in T3cDM.

It helps improve bone health and overall metabolic stability.

5. Novel drug: Pancreatic polypeptide (PP)

Pancreatic polypeptide has emerged as a promising antidiabetic agent for T3cDM secondary to chronic pancreatitis.

It increases the expression of insulin receptors in the liver, thereby improving insulin sensitivity.

A stabilized micelle formulation of PP has been developed to overcome its short biological half-life.

Experimental studies in pancreatogenic diabetes rodent models have shown significant antidiabetic activity[6].

CASE PRESENTATION 1:

A 48 years old male patient was admitted to the general medicine ward at BMC and RC Bellary (Karnataka) with the chief complaints of abdominal pain which was at right hypochondriac region since 2 years which was on and off but increased since yesterday. The patient was known case of type 2 DM and hypertension since 6 months.The general random blood sugar checked it was high (580mg/dl).

THE LABORATORY INVESTIGATION

- Blood tests were done which revealed that increased neutrophils, decreased red blood cells.
- Random blood sugar was highly increased to 580mg/dl
- Liver function test revealed that increased in globulin, alkaline phosphatase and decrease in A/G ratio and bilirubin unconjugated.
- Renal profile revealed an increased I serum lipase, serum amylase, urine protein creatinine ratio.
- HbA1c test revealed increase sugar
- Ultrasound of the abdomen reported chronic calcific pancreatitis

LABORATORY PARAMETERS:

PARAMETERS	RESULTS			REFERENCE
	D1	D2	D5	
HEMATOLOGY				
Hemoglobin	13.3		13.8	13-18gm%
Total WBC count	9570		9890	4000-11000cells/cumm
Neutrophils	75		74	40-70%
Lymphocytes	20		21	20-40%
Eosophils	02		03	3-6%
Monocytes	03		02	2-10%
Basophils	0		0	0-1
RBC count	4.68		4.97	5.5-6.5m/cumm
Platelet count	4.08		3.77	1.5-4.5l/cumm
MPV	8.5		8.7	7-11fl
PCV	40.6		43.3	45-55%
MCV	86.7		87.1	80-100fl
MCH	28.3		27.8	27-34pg
MCHC	32.7		31.9	31-36%
RDW-CV	13.7		14.4	11.5-14.5%
PDW-CV	14.4		15.1	10-18%
Random blood sugar	580			70-140mg/dl
S.ELECTROLYTE				
Sodium blood	134	138		136-145mEq/L
Potassium blood	4.3	3.7		3.48-5mEq/L
Chloride blood	97	102		96-106mEq/L
LIVER FUNCTION TEST				
Total protein	6.3			6-8.3g/dl
Albumin	3.0			3.2-5.4g/dl
Globulin	3.3			2.5-3g/dl
A/G ratio	0.9			1.2-1.5
Bilirubin total	o.3			0.2-1.2mg/dl
Bilirubin conjugated	0.2			0.1-0.4mg/dl
Bilirubin unconjugated	0.1			0.2-0.7mg/dl

Alanine transaminase	14			0-45IU/L
Aspartate transaminase	11			0-40IU/L
Alkaline phosphatase	167			20-140U/L
RENAL PROFILE				
Serum creatinine	0.8	0.6		0.7-1.4mg/dl
Blood urea	18	15		15-45mg/dl
Serum lipase	224			7-60U/L
Serum amylase	89			19-80U/L
Urine protein creatinine ratio		438		10-150mg/g
HBA1C		14.4		<6.5mmol/l

OTHER INVESTIGATION:

USG ABDOMEN: Chronic calcific pancreatitis

PHARMACOLOGICAL THERAPY:

SL	MEDICATION	FREQUENCY	ROUTE	DOSE	DAYS
1	INJ TRAMADOL	SOS	IV	100mg in 100ml NS	D1-D3
2	INJ PANTOPRAZOLE	1-0-0	IV	40mg	D1-D8
3	INJ INSULIN R	1-1-1	S/C	18-18-16	D1-D8
4	INJ PIPRACILIN TAZOBACTAM	STAT 1-1-1	IV	4.5g	D1
5	IV FLUIDS	@75ml/h	IV	2. NS	D1-D2
6	INJ INSULIN N	1-0-1	S/C		D1-D7
7	INJ CEFTRIAXONE	1-0-1	IV	1gm	D1-D8
8	T PARACETAMOL	1-1-1	PO	500mg	D1-D8
9	INJ BASILOG	1-0-0	S/C	8-0-0	D7-D8
10	INJ PANTOPRAZOL	1-0-0	Infusion in 100ml NS	80mg	D7-D8
11	T PANCREATIN	1-1-1	PO	25000IU	D2-D8

DISCHARGE MEDICATIONS;-

SL	MEDICATION	FREQUENCY	ROUTE	DOSE	DAYS
1	Tab. PANTOPRAZOLE	1-0-0	PO	40mg	D1-D8
2	INJ INSULIN R	1-1-1	S/C	18-18-16	D1-D8
3	INJ INSULIN N	1-0-1	S/C		D1-D7
4	Tab. CEFIXIME	1-0-1	PO	200mg	D1-D8
5	T PARACETAMOL	1-1-1	PO	500mg	D1-D8
6	INJ BASILOG	1-0-0	S/C	8-0-0	D7-D8
7	T PANCREATIN	1-1-1	PO	25000IU	D2-D8

Recommended to review after 2 weeks.

CASE PRESENTATION 2:

A 51 years old female patient admitted to the female emergency ward at BMCRC Bellary (Karnataka) with the chief complaints of vomiting of 2-3episodes which was watery and abdominal pain is present which sudden onset progressive at the periumbilical area. She had a past history old IHD old acute coronary syndrome non ST elevation myocardial infraction since 2 years and also with chronic calcific pancreatitis. On examination BP was found to be 120/80 mmHg, pulse rate was 116 bpm, SpO2 was 96% @RA and GRBS was 583mg/dl. Clinical examination P/A found soft, tender (+) at epigastric area, no orgenomegaly.

LABORATORY INVESTIGATION:

PARAMETERS	RESULTS		REFERENCE
	D1	D2	
HEMATOLOGY			
Hemoglobin	8.4	8.6	12-16gm/dl
Total WBC count	9230	12200	4000-11000cells/cumm
Neutrophils	51	70	40-70%
Lymphocytes	24	25	20-40%
Eosophils	06	02	3-6%
RBC	3.16	3.25	3.5-5.0x10^6/mm^3
MCH	26.7	26.6	27-34pg
MCHC	33.3	33.4	31-36%
Random blood sugar	45		70-140mg/dl
S.ELECTROLYTE			
Sodium blood	137	139	136-145mEq/L
Potassium blood	4.0	3.9	3.48-5mEq/L
Chloride blood	96	104	96-106mEq/L
LIVER FUNCTION TEST			
Albumin			3.2-5.4g/dl
Globulin			2.5-3g/dl
A/G ratio			1.2-1.5
Bilirubin Total	0.2	0.2	0.2-1.2mg/dl
Bilirubin Conjugated	0.1	0.1	0.1-0.4mg/dl
Bilirubin Unconjugated	0.1	0.1	0.2-0.7mg/dl
Alanine Aminotransferase	18	10	0-45IU/L
Aspartate Aminotransferase	26	12	0-40IU/L
Alkaline Phosphatase	187	128	20-140U/L
RENAL PROFILE			
Serum creatinine	1.6	1.3	0.7-1.4mg/dl
Blood urea		15	15-45mg/dl
Serum lipase	224		7-60U/L
Serum amylase	89		19-80U/L
Urine protein creatinine ratio		438	10-150mg/g
THYRIOD TEST			
TSH	0.666		
FT3	1.38		pg/ml
FT4	0.60		ng/dl

OTHERS :

USG ABDOMEN: B/L grade 1 renal parenchyma disease and chronic calcific pancreatitis

ECG: Sinus arrhythmia

Prolonged QT wave

2D ECHO: Concentric LVH

Grade 1 LV diagnostic dysfunction

EF: 60%

PHARMCOLOGICAL THERAPY:

SL	MEDICATION	FREQUENCY	ROUTE	DOSE	DAYS
1	INJ INSULIN R	INFUSION	IV		D1-D2
2	INJ CEFTRIAXONE	1-0-1	IV	1gm	D1-D3
3	INJ PANTOPRAZOLE	1-0-0	IV	40mg	D1-D3
4	INJ ONDANSETRON	1-1-1	IV	4mg	D1-D3
5	T PANCREATIN	1-1-1	PO	25000IU	D1-D3
6	T TRAMADOL	1-1-1	PO	2amp/100ml NS	D1-D3
7	T ASPIRIN	0-1-0	PO	75mg	D1-D3
8	T CLOPIDOGREL	0-1-0	PO	75mg	D1-D3
9	T ATORVASTATIN	0-0-1	PO	40mg	D1-D3
10	SYP SUCRALFATE	1-0-1	PO	5ml	D1-D3
11	INJ INSULIN R	1-0-1	SC	10-10-8	D2-D3
12	T THYRONORM	1-0-0	PO	25mg	D2-D3

DISCHARGE MEDICATION:

SL	MEDICATION	FREQUENCY	ROUTE	DOSE	DAYS
1	INJ INSULIN R	1-0-1	SC	14-0-12	
2	T GLIMEPRIDE1	1-0-0	PO	1mg	15
3	T METAPROLOL	1-0-1	PO	500mg	30
4	T PANCREATIN	1-0-1	PO	25000IU	30
5	T TELMESARTAN	1-0-0	PO	40mg	30
6	T SODIUM BICARBONATE	1-0-0	PO	500mg	15
7	T CETRAZINE	0-0-1	PO	10mg	15
8	T ASPERIN	0-1-0	PO	75mg	30
9	T CLOPEDOGREL	0-1-0	PO	75mg	30
10	T ATORVSTATIN	0-0-1	PO	40mg	
11	T CEFIXIME	1-0-1	PO	200mg	

- Post Splenectomy vaccines are advised.
- Review after 2 weeks.

CASE PRESENTATION 3:-

A 34 years old male admitted to the male medical ward of general medicine in tertiary care hospital, with the chief complaints of breathlessness since 1 day, abdominal pain since 1 day. They'r past history revealed that he was a known case of diabetes mellitus since 10 years on irregular treatment, history of surgery for pancrease since 12 years back. On examination patient BP was 110\70mm of Hg, Pulse rate was 90bpm and SPO_2 was 98%@RA. External examination physician advised for tests like Complete blood count, Liver function test, Renal profile test, Biochemistry , Serum electrolytes, Urine routine test, Urine ketone bodies, serology test for the further diagnosis.

Table 1-LABORATORY PARAMETERS

SL. NO	TESTS	PARAMETERS	RESULTS ON ADMISSION	RESULT DURING DISCHARGE	REFERENCE RANGE
	COMPLETE BLOOD COUNT	Haemoglobin	**11.8**	**11**	12.5-16gm%
		Total WBC Count	**17730**	**8360**	4000-11000 cells/cumm
		Red blood cells	**5.30**	**4.9**	4.5-5.5million/cumm
		Platelets	**2.79**	**1.61**	1.5-4.5lakh/cumm

		Neutrophils	**76**	**70**	40-70%
		Packed cell volume	**44.2**	**24.8**	35-46%
		Lymphocytes	**16**	**24**	20-40%
		Mean platelet volume	**9.4**	**10.0**	7-11 fL
		Mean corpuscular hemoglobin	**27.9**	**27.5**	27-34 pg
		RDW-CV	**13.8**	**14.4**	11.5-14.5 %
	LIVER FUNCTION TESTS	Albumin	**3.7**	**3.8**	3.2-5.4g/dl
		Globulin	**2.8**	**2.5**	2.5-3g/dl
		A/G ratio	**1.3**	**1.5**	1.2-1.5
		Total bilirubin	**0.6**	**1.0**	0.2-1.2mg/dl
		Total protein	**6.5**	**6.3**	5-8.3 g/dl
		Conjugated bilirubin	**0.2**	**0.4**	0.1-0.4mg/dl
		Unconjugated bilirubin	**0.4**	**0.6**	0.2-0.7mg/dl
		Aspartate transaminase	**20**	**19**	0-40 IU/L
		Alkaline phosphate	**114**	**95**	20-140U/L
	RENAL FUNCTION TESTS	Blood urea	**15**	**18**	15-45 mg/dl
		Serum creatinine	**1.1**	**0.9**	0.7-1.4 mg/dl
	SERUM ELECTROLYTES	Sodium	**110**	**125**	136-146mEq/l
		Potassium	**3.2**	**4.1**	3.48-5mEq/l
		Chloride	**102**	**97**	96-106mEq/l
	BIOCHEMISTRY	Random blood sugar	**403**	**80**	70-140 mg/dl
	URINE ROUTINE	Urine albumin	**Traces**	**NIL**	.
		Urine microscopy	**2-3 pus cells**	**NIL**	.
		Urine sugar	**1%**	**NAD**	.

ESR : 16mm

URINE KETONE BODIES : Present

HBA1C TEST :

GLYCATED HAEMOGLOBIN : 15.0

MEAN BLOOD GLUCOSE : 383

SEROLOGY TEST :

HIV 1 & 2 ; NON REACTIVE

HBsAg : Negative

HCV : Negative

CRP (QUANTITATIVE) ; 89

ULTRASOUND SONOGRAPHY ABDOMEN REPORT:- Heterogenous echotexture and calcified foci over pancreas.

PERIPHERAL SMEAR ONLY : Normocytic Hypochromic Blood Picture

Table 2-TREATMENT CHART

SL.NO	NAME OF THE MEDICATIONS	DOSE	ROUTE	FREQUENCY	DURATION
1	Inj. Insulin R	10 unit/ 6ml/hr	IV	STAT/ Infusion	D1-D3
2	Inj. Ceftriaxone	1g	IV	1-0-1	D1-D3
3	Inj. Pantoprazole	40mg	IV	1-0-0	D1-D11
4	Inj. IV Fluids	3pint NS/2pint RL	IV		D1-D11
5	Inj. KCL	2amp in 1 Pint	IV	Over 4 hrs	D1-D2
6	Inj . Sodium bicarbonate	5ampnin 1 pint NS	IV	Over 6 hrs	D1-D2
7	Inj. Thiamine in 100 ml NS	100mg	IV	1-1-1	D2-D10
8	Inj. PIPZO	4.5mg	IV	1-1-1	D2-D8
9	Inj. Insulin R		S/C		D4-D8
10	Inj. Insulin N		S/C		D4-D8
11	Inj. Insulin mixtard		S/C		D9-D11

The patient was treated with above medications.

Table 3-DISCHARGE MEDICATION

SL.NO	NAME OF THE MEDICATION	DOSE	DOSE	FREQUENCY
1	Inj. Insulin R	6-6-8	S/C	
2	T. Ceftriaxone	500mg	PO	1-0-1
3	U. Sodium bicarbonate	500mg	PO	0-1-0
4	T. Pantoprazole	40mg	PO	1-0-1
5	T.Vitamin b complex		PO	1-0-1

Suggested to review after 2 weeks.

Discussion

Type 3c diabetes mellitus (T3cDM) or pancreatogenic diabetes, arises secondary to diseases affecting the exocrine pancreas and remains widely underrecognized in clinical practice. The present case series highlights the varied clinical presentations, diagnostic challenges and therapeutic complexities of T3cDM associated with chronic pancreatic pathology. All three patients demonstrated structural pancreatic abnormalities, poor glycaemic control and features suggestive of both endocrine and exocrine dysfunction, fulfilling the diagnostic criteria for T3cDM.

Chronic calcific pancreatitis was the predominant underlying etiology in this series, consistent with published literature identifying it as the most common cause of T3cDM. Progressive pancreatic inflammation, fibrosis, and calcification lead to destruction of insulin-producing β-cells as well as impairment of glucagon and pancreatic polypeptide secretion. This combined hormonal deficiency explains the marked glycaemic variability observed in these patients, including severe hyperglycaemia and episodes of hypoglycaemia, particularly in those requiring insulin therapy.

Misclassification of T3cDM as type 2 diabetes was evident, especially in patients with long-standing diabetes and metabolic instability. Such misdiagnosis may delay appropriate treatment, including early initiation of insulin and pancreatic enzyme replacement therapy (PERT). In this series, the addition of PERT improved digestive symptoms and contributed to better metabolic control, supporting its essential role in managing exocrine pancreatic insufficiency.

Furthermore, the presence of comorbidities such as cardiovascular disease, renal impairment and nutritional deficiencies underscores the need for a multidisciplinary approach. Early identification of T3cDM through careful clinical assessment, pancreatic imaging and evaluation of exocrine function is critical to prevent acute metabolic complications and long-term microvascular damage.

Conclusion

Type 3c diabetes mellitus is an underdiagnosed but clinically significant form of secondary diabetes associated with pancreatic disease. This case series emphasizes the importance of considering T3cDM in patients with diabetes and underlying pancreatic pathology, particularly chronic pancreatitis or prior pancreatic surgery. Accurate classification enables tailored management strategies including insulin therapy and pancreatic enzyme replacement, which are essential for achieving glycaemic stability and improving nutritional status. Increased awareness among clinicians can reduce misdiagnosis, prevent complications, and improve overall patient outcomes.

ACKNOWLEDGEMENT

CONSENT

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

It is not applicable.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

REFERENCES

1. Nutrition Interest Group of the Pancreatic Society (NIGPS). Type 3c diabetes and healthy living. London: Pancreatic Society; 2019. Review date: May 2021
2. Duggan SN, Conlon KC. Pancreatogenic type 3c diabetes: underestimated, underappreciated and poorly managed. Pract Gastroenterol. 2017 May;41(5):14–23
3. Duggan SN, Johnston PC. Type 3c diabetes and nutritional management. CN. 2024;24(4):22–25
4. Pantea-Stoian A, Diculescu M et al. Pancreatogenic type 3C diabetes. J Mind Med Sci. 2018;5(2):270-277. doi:10.22543/7674.52.P270277.
5. Riaz S. Prevalence and epidemiology of diabetes mellitus type 3C. International Journal of Biomedical and Clinical Research. 2025;3(3). doi:10.59657/2997-6103.brs.25.052
6. Arjun HR, Halagali P, Tausif M, Vaishnavi J, Rakshith UR, Rakshanaa GS. Type 3c diabetes mellitus. Int J Pharm Investig. 2023;13(4):698-702. doi:10.5530/ijpi.13.4.087.
7. Wayne CD, Benbetka C, Besner GE, Narayanan S. Challenges of managing type 3c diabetes in the context of pancreatic resection, cancer and trauma. **J Clin Med.** 2024;13(10):2993. doi:10.3390/jcm13102993
8. Bhattamisra SK, Siang TC, Rong CY, Annan NC, Sean EHY, Xi LW, **et al.** Type-3c diabetes mellitus, diabetes of exocrine pancreas – an update. Curr Diabetes Rev. 2019;15(1):1–10. doi:10.2174/1573399815666190115145702
9. Ewald N, Bretzel RG**.** Diabetes mellitus secondary to pancreatic diseases (Type 3c)—are we neglecting an important disease? Eur J Intern Med. **2013**;24(3):203–206. doi:10.1016/j.ejim.2012.12.017
10. MedlinePlus. Chronic pancreatitis [Internet]. Bethesda (MD): U.S. National Library of Medicine; c2023 [updated 2023 Dec 31; cited 2026 Feb 5]. Available from: https://medlineplus.gov/ency/article/000221.htm
11. Hart PA, Bellin MD, Andersen DK, Bradley D, Cruz-Monserrate Z, Forsmark CE, et al. Type 3c (pancreatogenic) diabetes mellitus secondary to chronic pancreatitis and pancreatic cancer. Lancet Gastroenterol Hepatol. 2016;1(3):226-237. doi:10.1016/S2468-1253(16)30106-6. PMID:28404095.

International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 32, Issue 5, May 2026 **ijppr.humanjournals.com** ISSN: 2349-7203

How to cite this article:
Sumangala V et al. Ijppr.Human, 2026; Vol. 32 (5):230-242.

Conflict of Interest Statement: All authors have nothing else to disclose.

Image **Author -1**	Sumangala V – Corresponding Author VI Pharm D, Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104 India. Ward no : 05 near nursing college, Guggarahatti, Ballari, Karnataka/ Veerasaiva College Campus, Airport Road, Kolagal, Road, Cantonment, Ballari, Karnataka 583104. India
Image **Author -2**	B Sneha VI Pharm D, Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104 India. Near durgamma temple, sirguppa road, Ballari, Karnataka/ Veerasaiva College Campus, Airport Road, Kolagal, Road, Cantonment, Ballari, Karnataka 583104. India
Image **Author -3**	E P Akshitha VI Pharm D, Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104 India. Ballari, Karnataka/ Veerasaiva College Campus, Airport Road, Kolagal, Road, Cantonment, Ballari, Karnataka 583104. India
Image **Author -4**	Manasa A V Pharm D, Togari Veeramallappa Memorial College of Pharmacy, Karnataka, Ballari – 583104 India. Ballari, Karnataka/ Veerasaiva College Campus, Airport Road, Kolagal, Road, Cantonment, Ballari, Karnataka 583104. India
Image **Author -5**	**Dr. Syed Mohammed Hussaini** Junior Doctor, Sehat Hospital, Ballari Ballari, Karnataka.