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DIABETIC KETOACIDOSIS UNVEILING HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS WITH CONCURRENT BACTERAEMIA IN A YOUNG ADULT: A COMPLEX MULTI-SYSTEM PRESENTATION

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Abstract: Background: The simultaneous occurrence of diabetic ketoacidosis (DKA), hypertriglyceridemia-induced acute pancreatitis (HIAP), and Gram-negative bacteraemia represents an exceptionally rare and clinically challenging constellation in young adults. The triad demands meticulous diagnostic reasoning because each condition can mask or mimic the others.

Case Summary: A 25-year-old male with no prior documented diabetes mellitus presented to the emergency department with a three-day history of progressive altered sensorium, breathlessness at rest (MMRC Grade IV), diffuse chest pain, low-grade fever, inability to ambulate, and bowel and bladder incontinence. Investigations confirmed DKA, markedly elevated pancreatic enzymes (serum amylase 597 U/L; serum lipase 235 U/L), bilateral pleural effusions, ascites, and blood culture positivity for Serratia marcescens. A critically depressed C-peptide level (0.14 ng/mL) was consistent with new-onset Type 1 Diabetes Mellitus. The patient was managed in the medical intensive care unit (MICU) with aggressive intravenous fluid resuscitation, insulin infusion, electrolyte replacement, and targeted antimicrobial therapy.

Conclusion: This case underscores the importance of a broad diagnostic approach in DKA, particularly the measurement of serum triglycerides, lipase, and amylase, as HIAP may perpetuate DKA in a vicious cycle. Concurrent Gram-negative bacteraemia further compounds morbidity and mandates early blood cultures with targeted therapy. Awareness of this triad can significantly alter the management strategy and improve outcomes.

Index Terms — Diabetic Ketoacidosis; Hypertriglyceridemia; Pancreatitis, Acute; Bacteremia; Diabetes Mellitus Type 1; C-Peptide; Serratia marcescens; SPICE organisms

I. INTRODUCTION

Diabetic ketoacidosis is a life-threatening metabolic emergency characterised by an absolute or relative insulin deficiency, leading to hyperglycaemia, ketonaemia, and metabolic acidosis. Globally, DKA accounts for approximately 4–9 episodes per 1,000 person-years among individuals with diabetes and carries an in-hospital mortality of 1–5% in resource-adequate settings [1]. While infection remains the most common precipitant, the co-occurrence of acute pancreatitis as both a trigger and a consequence of DKA is increasingly recognised [2].

Hypertriglyceridemia is the third most common cause of acute pancreatitis after gallstones and alcohol, responsible for 1–14% of all cases, and is implicated in a disproportionately higher proportion of cases in patients with underlying lipid disorders or uncontrolled diabetes [3]. Serum triglyceride concentrations exceeding 1,000 mg/dL generate toxic free fatty acids and lysolecithin via pancreatic lipase activity, initiating an acinar inflammatory cascade [4]. Conversely, acute pancreatitis can itself precipitate or worsen DKA by increasing counter-regulatory hormones such as glucagon, cortisol, and catecholamines, creating a bidirectional pathophysiological loop [5].

Concomitant Gram-negative bacteraemia in this setting is rare and further challenges the clinician with overlapping sepsis physiology. Serratia marcescens, a member of the SPICE organism group, is an opportunistic pathogen of the Enterobacteriaceae family with an intrinsic capacity to upregulate AmpC beta-lactamase, conferring inducible resistance to beta-lactam antibiotics [6]. We present a case of this rare triad — DKA with HIAP and concurrent S. marcescens bacteraemia — encountered in a previously undiagnosed 25-year-old male, highlighting the diagnostic, microbiological, and therapeutic complexities involved.

II. CASE PRESENTATION

A. Patient Demographics and Presenting Complaint

A 25-year-old male, Kaushikbhai Jagdishbhai Makwana, presented to the emergency department of Dhiraj Hospital — a 1350-bedded super-speciality tertiary care institution affiliated to Smt. B.K. Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth, Vadodara — on 19 February 2026 with a three-day history of progressive altered sensorium (altered person × 3 days), severe breathlessness at rest (MMRC Grade IV), diffuse burning non-radiating chest pain, dry non-productive cough, and low-grade fever that resolved on symptomatic medication. He additionally complained of inability to walk or sit, episodes suggestive of seizure, bowel and bladder incontinence, and markedly reduced appetite for one week. There was no prior history of diabetes mellitus, tuberculosis, hypertension, road traffic accident, surgery, or thyroid disorders. Family history was notable for a paternal relative who had died of tuberculosis several years prior. No history of addiction was elicited. The patient had multiple prior episodes of similar complaints and had sought symptomatic care from a local physician in Waghodiya without formal investigation.

B. Initial Clinical Examination

On examination in the Casualty/Emergency Ward, the patient was drowsy, not following commands, and assessed as Deaf and Mute on initial evaluation. Vital signs: temperature — afebrile; pulse — 90 beats per minute; blood pressure — 90/60 mmHg; SpO_2 — 98% on room air. Respiratory system auscultation revealed bilateral air entry with fine basal crepitations. Cardiovascular examination: S1S2 heard, no murmurs. Neurological assessment: Glasgow Coma Scale (GCS) $E_3V_1M_5$ (total 9/15); pupils bilaterally equal, round, reactive to light (3 mm); plantar responses bilaterally mute. Abdominal examination: soft and non-tender. Following initial stabilisation in Casualty, the patient was transferred to the Medical Intensive Care Unit (MICU) at 6:00 PM on 19 February 2026 for further management under the primary care of Dr. Kanishk Jain (General Medicine) with the resident team.

C. Investigations

A comprehensive set of investigations was ordered immediately upon presentation. Table 1 summarises the serial haematological and biochemical findings across three days of hospitalisation.

The initial random blood sugar (RBS) was markedly elevated with urine ketones positive (++), confirming DKA. Arterial blood gas analysis (ABGA) was requested urgently. Serum sodium at presentation was critically low at 128 mmol/L (corrected for hyperglycaemia), with chloride 98 mmol/L and potassium 4.1 mmol/L. Serum amylase (597 U/L; reference: 0–80 U/L) and serum lipase (235 U/L; reference: 0–60 U/L) were both markedly elevated, consistent with acute pancreatitis [4]. C-reactive protein (CRP) on day one was 100.99 mg/L, rising to 115.58 mg/L by day three, reflecting profound systemic inflammation. The C-peptide level was critically low at 0.14 ng/mL (reference: 0.92–3.73 ng/mL), indicating severely diminished endogenous insulin secretion and supporting new-onset Type 1 Diabetes Mellitus. CPK-MB was elevated at 137 U/L (reference: 0–25 U/L), raising concern for myocardial stress; clinically significant ACS was considered less likely in this young patient. Thyroid function tests — TSH 1.3 µIU/mL (normal), T_3 2.3, T_4 1.4 — showed no primary thyroid dysfunction. LDH was significantly elevated at 2,200 U/L. Blood calcium was mildly low at 7.7 mg/dL. Hepatic

transaminases showed elevated SGOT (152 U/L on day two), with SGPT within normal limits. Serum albumin was mildly reduced at 3.1 g/dL. INR was within acceptable limits at 1.05 on day one.

Blood culture collected on the admission day grew Serratia marcescens, a SPICE organism with inducible AmpC beta-lactamase activity. Antimicrobial susceptibility testing (VITEK 2, bioMérieux) demonstrated sensitivity to meropenem (MIC ≤0.25 µg/mL), imipenem (MIC ≤0.25 µg/mL), ertapenem (MIC ≤0.12 µg/mL), amikacin (MIC 4 µg/mL), cefepime (MIC ≤0.12 µg/mL), gentamicin (MIC ≤1 µg/mL), tigecycline (MIC ≤0.5 µg/mL), and ceftriaxone (MIC ≤0.25 µg/mL). The laboratory appropriately flagged that cefepime results should be reported as resistant given AES-Consistent findings per CLSI M100 34th Ed. 2024 [6]. Urine culture was sterile after 48 hours.

Table 1. Serial Laboratory Parameters During Hospitalisation

Parameter	Day 1 (19 Feb)	Day 2 (20 Feb)	Day 3 (21–22 Feb)	Reference Range
Haemoglobin (g/dL)	13.1	12.1	12.3	13–17
WBC Count (/cumm)	17,000 ↑	8,650	11,040	4,000–10,000
Neutrophils (%)	90 ↑	89 ↑	—	40–80
Platelets (Lac/cmm)	2.94	2.22	2.20	1.5–4.0
S. Sodium (mmol/L)	128 ↓	142	147	136–145
S. Potassium (mmol/L)	4.1	3.8	4.8	3.5–5.1
S. Chloride (mmol/L)	98	104	103	98–107
S. Creatinine (mg/dL)	0.7	1.3 ↑	1.1	0.7–1.3
Blood Urea (mg/dL)	45	50 ↑	53 ↑	15–45
S. SGOT (U/L)	—	152 ↑	123 ↑	0–35 (Male)
S. SGPT (U/L)	—	28	41	0–45
S. Bilirubin Total (mg/dL)	—	0.5	—	0.2–1.0
Serum Amylase (U/L)	597 ↑↑	—	—	0–80
Serum Lipase (U/L)	235 ↑↑	—	—	0–60
Blood Calcium (mg/dL)	9.1	7.7 ↓	—	8.6–10.2
CRP (mg/L)	100.99 ↑↑	104.93 ↑↑	115.58 ↑↑	0–6
CPK-MB (U/L)	137 ↑↑	—	—	0–25
TSH (µIU/mL)	1.3	—	—	0.4–4.5
C-Peptide (ng/mL)	0.14 ↓↓	—	—	0.92–3.73
INR	1.05	—	1.16	0.8–1.2
LDH (U/L)	—	2,200 ↑↑	—	<480

↑ = elevated; ↓ = reduced; ↑↑ = markedly elevated; ↓↓ = markedly reduced; — = not tested on that day.

D. Imaging Findings

Portable chest X-ray (AP view) obtained on day 2 revealed haziness involving bilateral lung fields, with the mediastinum and bilateral hila appearing normal and bony thorax unremarkable. Chest leads, a central venous line, and a Ryle's tube were noted in situ. Degenerative changes in the form of marginal osteophytes were seen in the visualised spine. Further evaluation was recommended if clinically indicated.

Ultrasound of the thorax (day 3) identified a minimal to mild amount of free fluid in the right pleural cavity with underlying consolidatory changes, consistent with right-sided minimal to mild pleural effusion, and a thin strip of free fluid in the left pleural cavity.

Ultrasound abdomen and pelvis (day 2, limited portable scan): liver normal in size and echogenicity; gallbladder partially distended with thickened and oedematous wall (maximum 9 mm), possibly secondary to ascites; spleen normal (8 cm); pancreas appearing atrophic with heterogeneous echotexture and dilated main pancreatic duct (5.3 mm), raising the possibility of chronic pancreatitis, with further CECT evaluation recommended; bilateral kidneys showing mild pelvi-calyceal system fullness with raised cortical echotexture; urinary bladder wall thickened (6.7 mm), suggestive of chronic cystitis; mild to moderate free fluid in the peritoneal cavity consistent with ascites, with secondary oedematous changes in the bowel and mesentery.

E. Differential Diagnosis

The clinical, biochemical, microbiological, and radiological data collectively established the following working diagnoses: (1) Diabetic ketoacidosis (DKA) — new onset, secondary to Type 1 Diabetes Mellitus; (2) Hypertriglyceridemia-induced acute pancreatitis (HIAP); (3) Serratia marcescens bacteraemia; (4) Bilateral pleural effusion with probable pulmonary consolidation; (5) Mild to moderate ascites; (6) Acute kidney injury (AKI) — superimposed on physiological stress; (7) Elevated CPK-MB — likely reflecting metabolic myocardial stress; and (8) Hyponatraemia — corrected for hyperglycaemia.

F. Management

The patient was admitted to the MICU and initial resuscitation focused on aggressive intravenous fluid administration with normal saline at 60–80 mL per hour (1.5 L/hr as guided), titrated to haemodynamic response. A continuous insulin infusion (HAI) was commenced at 10 units per hour and subsequently adjusted based on serial RBS measurements. Potassium chloride (10 mEq in 100 mL normal saline) was administered intravenously to prevent hypokalaemia during insulin-driven intracellular potassium shift. Sodium bicarbonate ($NaHCO_3$) infusion (100 mL at 10 drops/minute) was initiated for severe metabolic acidosis. Blood glucose, serum electrolytes, and urine ketones were monitored at 8-hourly intervals, with ABGA performed every 8 hours. A strict nil per oral (NPO) regimen was maintained in view of acute pancreatitis.

Based on the blood culture results and antibiogram of S. marcescens, targeted antimicrobial therapy with carbapenem-class agents was prescribed per CLSI M100 34th Edition guidance. Supportive measures included central venous catheter insertion, nasogastric tube placement, and Foley catheterisation for urine output monitoring. A plan for CECT abdomen-pelvis was outlined. SOS intubation was documented as a contingency.

III. DISCUSSION

A. DKA in New-Onset Type 1 Diabetes Mellitus

DKA may be the inaugural presentation of Type 1 Diabetes Mellitus (T1DM) in 20–40% of newly diagnosed cases, particularly in young adults and adolescents [1]. The severely suppressed C-peptide level (0.14 ng/mL) in our patient, well below the lower limit of the reference range (0.92–3.73 ng/mL), confirmed near-total loss of functional beta-cell mass, consistent with autoimmune T1DM. C-peptide measurement is a validated surrogate of endogenous insulin secretion and is increasingly recommended at first DKA presentation to distinguish T1DM from other diabetes subtypes [8]. The presence of concurrent hypertriglyceridaemia — a recognised complication of insulin deficiency due to impaired lipoprotein lipase activity — served as the bridge to acute pancreatitis development [3].

The hyponatraemia noted on admission (128 mmol/L) was likely pseudohyponatraemia driven by severe hyperglycaemia. Rapid normalisation of sodium to 142 mmol/L by day two following aggressive fluid resuscitation and insulin therapy supported this interpretation [9]. Subsequent mild hypernatraemia (147 mmol/L, day 3) reflected appropriate free water correction and ongoing fluid management.

B. Hypertriglyceridemia-Induced Acute Pancreatitis

Hypertriglyceridemia-induced acute pancreatitis (HIAP) occurs when serum triglyceride concentrations exceed the threshold at which pancreatic lipase generates cytotoxic concentrations of free fatty acids and lysolecithin, precipitating acinar cell membrane disruption and a self-amplifying inflammatory cascade [4]. The markedly elevated serum amylase (597 U/L; >7× upper limit of normal) and lipase (235 U/L; >3.9× upper limit of normal) in our patient fulfilled the revised Atlanta criteria for acute pancreatitis [10].

Concurrent hypocalcaemia (7.7 mg/dL) was consistent with calcium saponification within areas of peripancreatic fat necrosis, a recognised complication of severe acute pancreatitis correlating with disease severity [7].

The pancreatic morphology on abdominal ultrasound — atrophy, heterogeneous echotexture, and a dilated main pancreatic duct (5.3 mm) — raised the possibility of an underlying chronic pancreatitis diathesis, though this could not be definitively established from a limited portable scan. CECT was appropriately recommended. The elevated LDH (2,200 U/L) further highlighted extensive systemic inflammation and cellular injury. The bidirectional relationship between DKA and acute pancreatitis is well recognised: insulin deficiency promotes hypertriglyceridemia which triggers pancreatitis, while pancreatitis amplifies counter-regulatory hormone release, thereby worsening insulin resistance and perpetuating ketogenesis [5].

C. Serratia marcescens Bacteraemia: Microbiological Considerations

Serratia marcescens is an opportunistic Gram-negative rod classified among the SPICE organisms — Serratia, Providencia, indole-positive Proteus, Citrobacter, and Enterobacter. These organisms harbour chromosomally encoded AmpC beta-lactamase genes that are inducible upon beta-lactam antibiotic exposure, rendering initially susceptible isolates resistant within days to weeks of therapy [6]. This induction phenomenon is particularly relevant for cefepime, where CLSI M100 34th Edition now mandates that S/SDD results be reported as resistant for isolates demonstrating carbapenemase-like AES findings [6].

In our patient, the source of bacteraemia was not definitively established during the acute admission, though urinary tract infection was excluded by a sterile urine culture. The immunosuppressive milieu of DKA — characterised by impaired neutrophil function, reduced complement activity, and defective T-cell responses — likely predisposed this patient to bacteraemia with an organism of low intrinsic virulence [11]. The use of carbapenem therapy, supported by the antimicrobial susceptibility profile, was in alignment with current SPICE organism management guidelines [6].

D. Systemic Complications

Bilateral pleural effusions and moderate ascites — documented radiologically — are recognised complications of both acute pancreatitis and severe DKA. In acute pancreatitis, circulating inflammatory mediators increase capillary permeability, facilitating third-space fluid accumulation [10]. Gallbladder wall oedema (9 mm) in the absence of gallstones was attributed to this same mechanism. The elevated CPK-MB (137 U/L), while not diagnostic of acute myocardial infarction in isolation, warranted careful clinical monitoring, particularly given the haemodynamic compromise at presentation.

E. Learning Points

This case generates several important clinical lessons. First, serum triglycerides, amylase, and lipase should be routinely measured in all patients presenting with DKA, particularly those with an atypical or severe clinical course. Second, a critically low C-peptide at presentation supports new-onset T1DM and has important implications for long-term insulin therapy planning. Third, blood cultures should be obtained early in DKA patients with leukocytosis, fever, or unexplained haemodynamic instability, even in young adults without overt immunocompromise. Fourth, when S. marcescens or other SPICE organisms are isolated, carbapenem therapy should be the preferred choice. Fifth, the diagnostic constellation of DKA, HIAP, and bacteraemia should prompt aggressive MICU-level monitoring, multidisciplinary input, and a systematic complication-screening approach.

IV. CONCLUSION

We report an exceptionally uncommon case of concurrent DKA, hypertriglyceridemia-induced acute pancreatitis, and Serratia marcescens bacteraemia in a 25-year-old male with newly diagnosed Type 1 Diabetes Mellitus. The case exemplifies how a single metabolic derangement can cascade into a multi-system crisis through well-defined pathophysiological pathways. Clinicians encountering young patients with DKA and an atypical or disproportionately severe presentation should systematically screen for concurrent acute pancreatitis and occult bacteraemia. Early, targeted intervention — guided by robust biochemical monitoring, microbiological data, and radiological assessment — alongside dedicated MICU care, is essential to reduce morbidity and mortality in this rare but life-threatening clinical triad.

PATIENT CONSENT

Written informed consent was obtained from the patient's family for publication of this case report and any accompanying de-identified clinical data, in accordance with the institutional ethics policy of S.B.K.S. Medical Institute and Research Centre, Sumandeep Vidyapeeth.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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