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Severe Pancreatic Involvement In Severe Dengue: Simultaneous Presentation Of Acute Pancreatitis And Diabetic Ketoacidosis: A Case Report.

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ABSTRACT

Background:
Dengue fever is a major global health problem in tropical and subtropical regions. Moreover, severe dengue is associated with significant morbidity and mortality. Pancreatic involvement is an uncommon but clinically important manifestation which is reported in less than 2% of hospitalized dengue cases. Diabetes mellitus is increasingly recognized as a risk factor for severe dengue and dengue-associated diabetic ketoacidosis (DKA) has been infrequently described. The simultaneous occurrence of severe dengue, acute pancreatitis and DKA is rare and poses significant diagnostic and therapeutic challenges because of overlapping clinical features and competing management priorities.

Case Report:
We report a case of 50-year-old woman with long-standing poorly controlled diabetes mellitus who presented with a 5-day history of febrile illness, severe abdominal pain, vomiting and progressive dyspnea. On admission, she was hypotensive, hypoxemic, hyperglycemic and had Kussmaul breathing. Laboratory evaluation revealed high–anion gap metabolic acidosis with ketonuria consistent with DKA. Additionally, haemoconcentration, leukopenia, severe thrombocytopenia, acute kidney injury and markedly elevated serum amylase and lipase levels were also present. Dengue NS1 antigen was positive. Contrast-enhanced computed tomography demonstrated acute interstitial edematous pancreatitis (Balthazar grade E) with minimal ascites and bilateral pleural effusions. A diagnosis of severe dengue complicated by acute pancreatitis, DKA and hypovolemic shock was thus made. The patient was managed in the intensive care unit. Carefully titrated intravenous fluids, insulin infusion, vasopressor support was given along with close monitoring of vitals. She showed progressive clinical improvement and was subsequently discharged in stable condition. Follow-up imaging showed asymptomatic pancreatic pseudocyst which was managed conservatively.

Conclusion:
This case of a patient with poorly controlled diabetes mellitus highlights a rare but life-threatening triad of severe dengue, acute pancreatitis and DKA. A high index of suspicion, early diagnostic workup and individualized multidisciplinary management were important determinants for favorable outcomes.

Keywords:*Acute Pancreatitis, Dengue Fever, Diabetic Ketoacidosis, Expanded Dengue Syndrome, Shock*

INTRODUCTION

Dengue fever is caused by one of four serotypes of the dengue virus (DENV 1–4). It continues to be a significant public health problem in tropical and subtropical regions worldwide. The World Health Organization (WHO) estimates that approximately 390 million dengue infections occur annually out of which approximately 96 million manifest clinically.[1] Severe dengue can lead to life-threatening complications particularly in patients with pre-existing comorbidities. Among the less frequently reported but clinically significant complications in dengue fever is pancreatic involvement. Acute pancreatitis in the context of dengue is rare, with an estimated prevalence of 0.8%–1.2% in hospitalized dengue patients. Acute pancreatitis in cases of dengue is associated with increased morbidity as well as mortality.[2] The pathophysiology is believed to involve a combination of direct viral cytopathic effects, immune-mediated injury as well as ischemic damage secondary to dengue-associated shock.

Diabetes mellitus further complicates the clinical course and management of dengue infection. The pro-inflammatory milieu and endothelial dysfunction seen in cases of diabetes may predispose individuals to severe dengue manifestations including multiorgan dysfunction.

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In addition, baseline hyperglycemia and relative immune dysfunction may increase susceptibility to secondary infections and worsen capillary leak, thereby complicating fluid resuscitation during the critical phase. Stress-induced hormonal surges during severe dengue can further aggravate glycemic control, and pancreatic inflammation may contribute to transient insulin deficiency. These factors collectively raise the risk of acute metabolic decompensation, making close glucose and ketone monitoring clinically important in high-risk patients.

Moreover, diabetic ketoacidosis (DKA) has been infrequently reported as a concurrent presentation with the dengue fever. When both acute pancreatitis and DKA present simultaneously in a case of severe dengue it represents a complex clinical scenario with overlapping symptomatology. Abdominal pain, electrolyte imbalance, metabolic acidosis as well as respiratory distress may be due to any of the three conditions thereby making early recognition and appropriate management a difficult task in these cases.[3]

Reports of pancreatic involvement in dengue particularly those complicated by DKA remain scarce in the literature. Most available data come from isolated case reports or small case series. This limits the understanding of the pathogenesis, clinical spectrum and optimal therapeutic strategies. In the rare instances where pancreatitis and DKA co-occur combined effects of dengue-induced pancreatic inflammation and pre-existing metabolic dysregulation becomes clinically relevant. Acute pancreatitis may precipitate DKA in patients with diabetes whereas severe hyperglycemia and dehydration in DKA may also exacerbate pancreatic injury. This is a classical vicious cycle where one pathology exacerbates the other thereby increasing the severity of both.[4]

Diagnostic evaluation in such cases requires a high index of suspicion particularly if severe abdominal pain, persistent vomiting and metabolic acidosis is present. In these cases, laboratory investigations (serum amylase, lipase, blood glucose and arterial blood gas analysis) needs to be interpreted in the broader clinical context. Imaging studies such as ultrasound and contrast-enhanced computed tomography (CECT) remain important in confirming pancreatic involvement and assessing its severity. However, interpretation of findings may be complicated by the presence of ascites or pleural effusions both which are common in dengue. Furthermore, differentiating dengue-associated plasma leakage from fluid shifts due to pancreatitis or DKA can be diagnostically challenging. Treatment is largely supportive and must address fluid-electrolyte imbalances, insulin therapy and hemodynamic stabilization while avoiding fluid overload in the critical phase of dengue.[5]

Despite the growing incidence of dengue and increasing global prevalence of diabetes mellitus, simultaneous presentation of dengue with acute pancreatitis and DKA has been rarely documented. This case report highlights the unique triad of complications in a single patient and contributes to the limited body of evidence.

CASE REPORT:-

A 50-year-old woman with a maternal family history of diabetes mellitus and hypertension, presented to the Emergency Department. She had a history of sporadic alcohol intake without episodes of intoxication and also reported chronic exposure to biomass smoke. She had long-standing diabetes mellitus with poor adherence to medical therapy. Her surgical history included a cesarean section and bilateral tubal ligation. She reported a 5-day history of headache, retro-orbital pain, myalgias, generalized arthralgia, fever (38.8 °C), severe abdominal pain, polyuria and multiple episodes of nausea and vomiting.

Over the subsequent hours she developed progressive dyspnea and tachypnea thereby prompting referral for comprehensive evaluation. On arrival, she was having tachycardia (120 beats/min), hypotension (79/49 mmHg), hypoxemia (SpO_2 82% on room air) and marked hyperglycemia (390 mg/dL). General Examination revealed dehydration with dry mucous membranes, generalized pallor, tachypnea and Kussmaul breathing. On auscultation decreased bibasilar breath sounds were noted. Severe epigastric tenderness was also present. Capillary refill time was prolonged (4 seconds) suggestive of peripheral hypoperfusion.

Initial arterial blood gas and metabolic evaluation demonstrated high–anion gap metabolic acidosis with appropriate respiratory compensation (pH 6.9, pCO 19 mmHg, pO80 mmHg, glucose 500 mg/dL, lactate 2.2 mmol/L, HCO 8 mmol/L, base excess −19.5). In presence of poorly controlled diabetes and Kussmaul breathing possibility diabetic ketoacidosis (DKA) was suspected. Urinalysis was done which showed marked glucosuria and ketonuria confirming DKA.

Complete blood count revealed haemoconcentration (hemoglobin 17.2 g/dL, hematocrit 55%), leukopenia (3,900/μL) with relative lymphocytosis (46%). Additionally, neutropenia (25%), and severe thrombocytopenia (30,000/μL) was also present. Given the clinical presentation, hematologic profile and epidemiological setting, dengue serology was performed. It showed for NS1 antigen positive with negative IgG and IgM antibodies. Blood chemistry showed acute kidney injury suggestive of KDIGO stage II (creatinine 1.7 mg/dL, urea 54 mg/dL) and hypernatremia (153 mEq/L). Liver function tests showed elevated transaminase levels without criteria for acute hepatitis (AST 271 U/L, ALT 120 U/L). Pancreatic enzymes were markedly raised (lipase 4,670 U/L; amylase 8,000 U/L). Inflammatory markers showed a significantly elevated C-reactive protein (192 mg/L) with a normal procalcitonin (0.12 ng/mL). The lipid profile was unremarkable.

In view of severe abdominal pain and dyspnea a thoracoabdominal computed tomography (CT) scan was obtained. This demonstrated bilateral pleural effusions with right predominance (Figure 1).

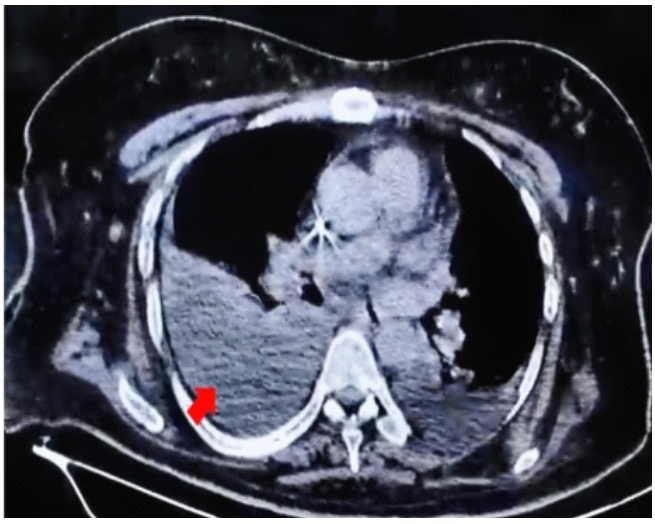

Figure 1:- Non-Contrast CT showing Bilateral pleural effusion with an attenuation of 4 HU and a maximum pleura-to-pleura thickness of 6.2 cm on the right side (red arrow) and 3 cm on the left side, resulting in passive atelectasis.

The abdominal CT findings were consistent with acute interstitial edematous pancreatitis (Balthazar grade E). The pancreas showed diffuse edema (head 3.4 cm, body 3.1 cm, tail 2.7 cm) with marked peripancreatic fat stranding, and areas of hypo-enhancement after contrast administration suggestive of necrosis, consistent with acute interstitial edematous pancreatitis (Balthazar E) (Figure 2).

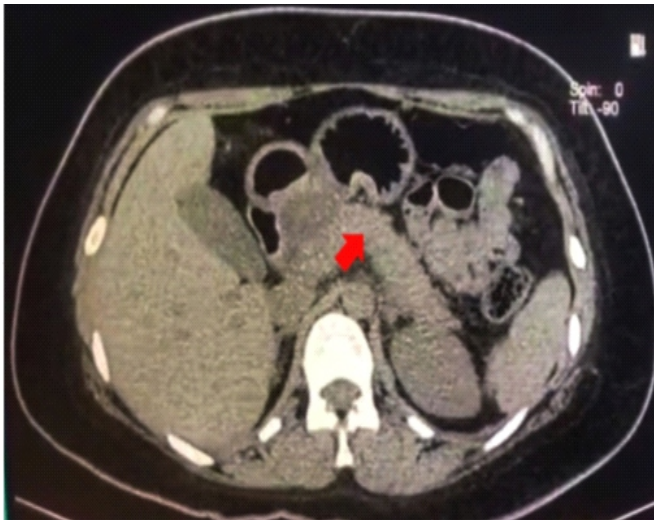

Figure 2 : Pancreatic gland with diffuse edema (red arrow). Marked peripancreatic fat stranding and areas of hypoenhancement after contrast administration are observed, suggestive of necrosis, consistent with necrotising pancreatitis.

Based on the combined clinical, laboratory and imaging findings a diagnosis of severe dengue with acute pancreatitis, severe DKA and non-hemorrhagic hypovolemic shock was established. Treatment was initiated with continuous intravenous insulin infusion, bicarbonate therapy, and aggressive fluid resuscitation with Hartmann's solution. Owing to persistent hypotension with signs of hypoperfusion, norepinephrine drip was started as first-line vasopressor support. She was admitted to the intensive care unit (ICU) for further management as well as close monitoring.

During her 7-day ICU course, she developed increasing oxygen requirements. A diagnostic and therapeutic right-sided thoracentesis was performed removing 800 mL of pleural fluid. The pleural fluid was transudate in nature. Intravenous fluids and insulin infusion were continued which led to resolution of the hyperglycemic crisis within 24 hours. There was progressive recovery of platelet count, correction of haemoconcentration, and eventual discontinuation of vasopressor therapy. Abdominal pain improved substantially after 48 hours, enabling initiation of enteral feeding, which was well tolerated.

With sustained clinical improvement and no ongoing organ failure, she was stepped down from ICU care with continued endocrinology follow-up for poor glycemic control and possible pancreatic dysfunction. As surveillance for late complications of acute pancreatitis a repeat abdominal CT was performed 4 weeks after discharge which demonstrated a pancreatic pseudocyst measuring 4.2 × 2.6 × 2.7 cm and corresponding to an estimated volume of 15 cc adjacent to the pancreatic neck. Despite presence of pancreatic pseudocyst on imaging patient was completely asymptomatic. The remaining pancreatic parenchyma was normal in size and attenuation, with preserved peripancreatic fat (Figure 3).

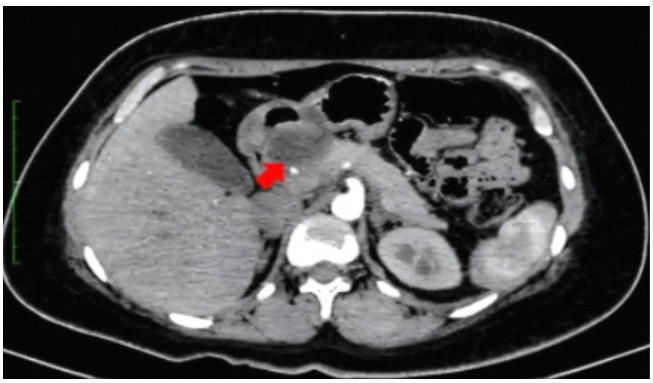

Figure 3: An oval-shaped lesion compatible with a pancreatic pseudocyst (red arrow) is identified in the pancreatic head and proximal body. It is well circumscribed, with a mean attenuation of 22 HU on non-contrast images. After contrast administration, enhancement of the cyst wall was observed.

Given the size (<6 cm), lack of symptoms and absence of infection, bleeding or obstruction, conservative line of management was adopted.

DISCUSSION

In this report, we describe a complex presentation of severe dengue characterized by acute pancreatitis and diabetic ketoacidosis (DKA) in a 50-year-old woman with poorly controlled diabetes mellitus. Acute pancreatitis in dengue is an unusual manifestation of expanded dengue syndrome and its true incidence remains uncertain due to under-reporting as well difficulty in its diagnosis. A recent systematic review by Ricco et al identified 70 cases of dengue-associated acute pancreatitis.[6] This review article concluded that although rare, pancreatic involvement is well documented in the literature and may contribute to significant morbidity particularly when associated with organ dysfunction and systemic complications such as shock and respiratory failure. In our patient, markedly elevated lipase and amylase values in addition to contrast-enhanced CT findings clinched the diagnosis.

The pathophysiology underlying dengue-induced pancreatitis remains debated. Proposed mechanisms include direct viral cytopathic effects, ischemia from plasma leakage and immune-mediated inflammation. In many published case reports severe abdominal pain and elevated pancreatic enzymes prompted imaging confirmation similar to our approach. This emphasises the need to maintain a high index of suspicion for such complications when persistent or severe abdominal symptoms accompany dengue infection. Some reports such as by Alnuaimi MK et al suggested that immune activation and increased vascular permeability in dengue create a milieu conducive to pancreatic injury, independent of gallstone or alcohol etiologies.[7] These findings are important and underscores the need for prompt recognition and supportive care in dengue cases complicated by pancreatitis. Our case adds to this evidence by illustrating how pancreatic inflammation may be a component of the critical phase of dengue, particularly in the context of comorbid metabolic disturbances caused by diabetes mellitus.

Simultaneous presentation of DKA with dengue infection, as seen in this case, is another important phenomenon from the perspective of management. The available literature suggests that dengue infection can precipitate DKA in both known as well as previously undiagnosed diabetic patients. For example, Thadchanamoorthy V et al described a child with dengue hemorrhagic fever who developed DKA in the critical phase of illness and required meticulous fluid and glycemic management.[8]

Similarly Lertdetkajorn et al reported an adult with DKA and dengue hemorrhagic fever thereby emphasizing the clinical complexity of managing fluid balance when both conditions coexist.[9] These reports are similar to our finding that hyperglycemia and ketosis in dengue may reflect a combination of exaggerated inflammatory responses and metabolic stress particularly in individuals with pre-existing diabetes.

The presence of diabetes mellitus is increasingly recognized to be increasing severity of dengue fever. Many observational studies have shown that individuals with diabetes often exhibit heightened inflammatory markers thereby predisposing to complications such as plasma leakage and organ involvement as compared with individuals without diabetes. Hyperglycemia itself is known to enhance dengue virus replication through increased translation activity. These findings suggest a biological basis for more severe disease in poorly controlled diabetes. In this case chronic poor glycemic control likely compounded endothelial dysfunction and significantly increased the severity of plasma leakage and systemic inflammation thereby facilitating both pancreatic injury as well as DKA.[10]

Management of patients with dengue complicated by pancreatitis and DKA remains largely supportive and requires careful balancing of fluid therapy, hemodynamic monitoring and metabolic control. Fluid management in dengue is particularly important and tricky when DKA is also present because aggressive hydration therapy which is necessary for DKA may worsen plasma leakage and risk fluid overload in dengue. Our multidisciplinary approach involving careful fluid resuscitation underscores the need for individualized management strategies. The favorable outcome in this patient underlines the importance of early recognition of these concurrent pathologies such as pancreatitis and DKA in patients of dengue.

CONCLUSION

This case underscores the importance of recognizing pancreas as a target organ in severe dengue. This is more so in patients with poorly controlled diabetes mellitus in whom the development of pancreatitis may precipitate severe hyperglycemic crises such as diabetic ketoacidosis (DKA). The coexistence of severe dengue, DKA and acute pancreatitis represents a rare but potentially fatal clinical scenario that requires a high index of suspicion and a multidisciplinary approach.

Conflict Of Interest : None

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