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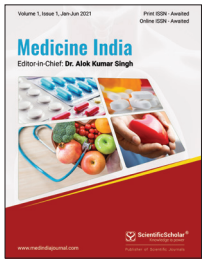

Case Report

Splenic abscess presenting as diabetic ketoacidosis: A rare cause of DKA in Type 2 DM patient

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ABSTRACT

Diabetic ketoacidosis is a relatively common and potentially life-threatening complication of insulin deficiency. The prevalence of DKA is relatively common in patients with Type 1 diabetes mellitus in comparison with Type 2 diabetes mellitus. The prevalence of DKA is seen in 0–128 cases per thousand population in Type 1 diabetes mellitus while Type 2 DM has a prevalence of 18% of the total population. Abdominal pain is the most common and specific symptom of DKA which is reported at around 46%. The two most common infections precipitating DKA are community-acquired pneumonia and UTI which correspond to around 30–50% of the total burden. Meanwhile, any infection or sepsis can precipitate DKA, the splenic abscess is rare, and there have been reported only a few cases. In patients with poor glycemic control, absence of urinary tract infection, and community-acquired pneumonia, deep organ abscesses should be screened for. Here, we present a case report of patient with non-resolving DKA who presented with splenic abscess.

Keywords: Diabetic ketoacidosis, Splenic abscess, Type 2 diabetes mellitus

INTRODUCTION

DKA results from relative or absolute insulin deficiency combined with counterregulatory hormone excess (glucagon, catecholamines, cortisol, and growth hormone). Both insulin deficiency and glucagon excess, in particular, are necessary for DKA to develop. The decreased ratio of insulin to glucagon promotes gluconeogenesis, glycogenolysis, and ketone body formation in the liver, as well as increases in substrate delivery from fat and muscle (free fatty acids, amino acids) to the liver. Ketosis results from a marked increase in free fatty acid release from adipocytes, with a resulting shift toward ketone body synthesis in the liver. Reduced insulin levels, in combination with elevations in catecholamines and growth hormone, also increase lipolysis and the release of free fatty acids.

DKA is characterized by hyperglycemia (serum glucose >13.9 mmol/L [250 mg/dL], ketosis, and metabolic acidosis [serum bicarbonate <15–18 mmol/L with increased anion gap]) along with a number of secondary metabolic derangements.

The timely diagnosis of DKA is crucial and allows for prompt initiation of therapy.

CASE REPORT

A 62-year-old male, retired businessman by occupation, resident of North India, a reformed smoker, with a history of Type 2 diabetes mellitus, for 8 years, on oral hypoglycemic agents irregularly with poor

glycemic control history, and history of hypothyroidism detected 2 years back for which he was taking thyroxine supplementation for 6 months, now presented with the complaints of insidious onset abdominal pain of 1-week duration, to the outpatient department, diabetes, and metabolism, General Medicine AIIMS, Rishikesh, on October 22, 2021. The pain was insidious in onset, moderate intensity, dull aching type, occasionally radiating back, relieved by taking over-the-counter pain medications, and it was aggravated by respiratory movements. There was no burning micturition, colicky flank pain, and hematuria and it was unrelated to the meal. There were no associated nausea, vomiting, and yellowish and discoloration of eyes/urine/body parts. There was no history of fever. There was no history of diarrhea, blood/mucous in stools, and clay-colored stools. There was no history of altered bowel habits initially, but in the past 3 days, the patient had constipation. Patient was admitted, in view of possible DKA. His Arterial blood gas analysis, blood sugar values, urine ketone analysis all led to the diagnosis of DKA. Manaement was started immediately with IV fluids and Insulin infusion.

SPECIAL INVESTIGATIONS

CECT thorax and abdomen (October 26, 2021) [Figure 1]

- Splenic abscess – 3.7 × 6.3 ×6.2 cm, ruptured
- Small right kidney
- Active infective etiology in the lung parenchyma.

USG abdomen (October 24, 2021)

- Cholelithiasis – Grade II fatty liver
- Right Kidney – 6.4 × 3 cm, raised cortical echogenicity and lost CMD
- Left kidney – 9.5 × 4 cm, raised cortical echogenicity, and partially lost CMD
- Spleen – 9 CM, normal size, shape, and echotexture.
- 2D ECHO – No RWMA/hypokinesia/LVEF 60%/no evidence of clot or vegetation, no valvular lesion
- 3 SITE blood C&S (October 27, 2021) – sterile, urine c&s – October 23, 2021 (sterile)
- Blood culture and AST (October 30, 2021) – *Klebsiella pneumoniae*
- Blood culture and AST (October 22, 2021) – No organism
- Blood culture from drained pus of splenic abscess – *Klebsiella pneumoniae* sensitive to meropenem.

DISCUSSION

DKA is defined as the presence of metabolic acidosis (HCO_3 <15), hyperglycemia (RBS >250 mg/dl), and the presence of ketones (positive in urine or serum).[1] Polyuria, polydipsia, weight loss, vomiting, and abdominal pain usually are present in patients with DKA.[2] Abdominal pain can be closely associated with acidosis and resolves with treatment. Physical examination findings such as hypotension, tachycardia, poor skin turgor, and weakness support the clinical diagnosis of dehydration in DKA. Mental status changes may occur in DKA and are likely related to degree of acidosis and/or hyperosmolarity. A search for symptoms of precipitating causes such as infection, vascular events, or existing drug abuse should be initiated in the emergency room. Patients with hyperglycemic crises can be hypothermic because of peripheral vasodilation and decreased utilization of metabolic substrates. Insulin deficiency, increased insulin counter-regulatory hormones (cortisol, glucagon, growth hormone, and catecholamines), and peripheral insulin resistance lead to hyperglycemia, dehydration, ketosis, and electrolyte imbalance which underlie the pathophysiology of DKA. Hyperglycemia of DKA evolves through accelerated gluconeogenesis, glycogenolysis, and decreased glucose utilization – all due to absolute insulin deficiency.[1,2] Of note, diabetes patients who developed DKA while treated with SGLT-2 inhibitors can present without hyperglycemia, that is, with euglycemic DKA. Due to increased lipolysis and decreased lipogenesis, abundant free fatty acids are converted to ketone bodies: β-hydroxybutyrate, acetoacetate, and acetone. Hyperglycemia-induced osmotic diuresis, if not accompanied by sufficient oral fluid intake, leads to dehydration, hyperosmolarity, electrolyte loss, and a subsequent decrease in glomerular filtration. With the decline in renal function, glycosuria diminishes

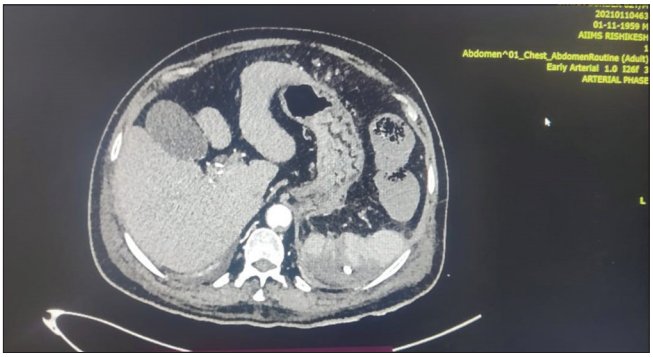

Figure 1: Image of CECT abdomen showing splenic abscess.

Figure 2: Image of splenic abscess drainage.

Table 1: Table of routine investigations done.

Investigations	October 22, 2021	October 26, 2021	October 30, 2021	October 31, 2021	November 1, 2021	November 2, 2021	November 4, 2021
Hb		11.86	11.35		10.2	13.27	12.5
TLC		24.16	17.01		15.29	15.93	12.64
Platelets		300	357.4		254	30	2.9.8
N/L/M/E		82/5.7/8.97/0.94	84/5.5/7.01/1.85		72/12/10.9/3.1	58/16/23	79.05/13.6/4.372/1.9
PT-INR						10.5/0.96	
Bilirubin (T)						1.40	
Bilirubin (D)						0.77	
SGPT						409	
SGOT						620	
ALP						378	
GGT						326	
S. protein						6.2	
S. albumin						3.29	
Blood urea	86	41		52	45	37	25
S. creatinine	2.01	1.64		1.55	1.45	1.4	1.24
S. Na+	130	125		131	130	130	133
S. K+	3.7	4.35		4.4	4.07	3.42	3
S. Cl-	94	98		101	101	101	96
S. calcium	7.6	7.4		7.4	7.8	7.8	7.5
S. uric acid	9.5	3.3		6.6	5.5	5.1	5.1
S. phosphorus	3.9	2.9		3.1	3	3.9	2.3
S. Vitamin D						11.8	
Viral markers		NR				NR	
Urinary sodium/U. potassium		560/56.5					
24 h U. protein		2405					
HbA1C	14.9						

Hb: Heamoglobin, TLC: Total leukocyte count, AG: Anion Gap; RBS: Random blood sugar N/L/M/E; Neutrophil/Lymphocyte/Monocyte/Eosinophil; ALP: Alkaline phosphatase; GGT: Gamma glutamyl transferrase

Table 2: Table of serial ABG monitoring done.

Date	October 23, 2021	October 23, 2021	October 24, 2021	October 24, 2021	October 25, 2021	October 26, 2021	October 31, 2021	November 1, 2022
PH	7.404	7.40	7.36	7.38	7.39	7.42	7.36	7.4
HCO_3-	13.6	12.8	12.4	13.1	14	15.5	21.7	19.6
PCO_2	21.6	20	21.5	23	25	23.7	39.3	32
Lactate	0.9	1.1	1.3	1.1	1	0.9	1.4	1
AG	14	14	13	13	12	9	6	7
RBS	484	378	301	270	250	288	245	188

AG: Anion Gap; RBS: Random blood sugar

and hyperglycemia/hyperosmolality worsens. With impaired insulin action and hyperosmolality, utilization of potassium by skeletal muscle is markedly diminished leading to intracellular potassium depletion. Furthermore, potassium is lost through osmotic diuresis causing profound total body potassium deficiency. Therefore, DKA patients can present with broad range of serum potassium concentrations. Nevertheless, a "normal" plasma potassium concentration may indicate that potassium stores in the body are severely diminished and the institution of insulin therapy and correction of hyperglycemia will lead to future hypokalemia.[1,2]

Splenic abscess is rare in diabetic patients and is associated with a high mortality rate.[3] The incidence of splenic abscesses has ranged from 0.14 to 0.7% in various autopsy series.[1] Type 2 diabetes causes increased risks of splenic abscess, and timely and effective treatment can lower the mortality rate. Splenic

abscesses are caused by wide range of organisms and have evolved over time, with risk of uncontrolled hyperglycemia in diabetics' patient.[4-6] Group B streptococcus, Gram-negative organisms are the most common type of organisms causing splenic abscess. Among them, Klebsiella Pneumonia has been detected to be one of the commonest organism repsonsible for hepatosplenic abscess in diabetic patients with uncontrolled blood sugars in various studies.[7,8]

It is to be noted that risk of deep organ abscess increases with uncontrolled hyperglycemia thus we report here a case report of a large splenic abscess as a precipitating infection of patient presented with DKA.

Our patient presented with high anion gap metabolic acidosis with urine ketones positive of 3+ and RBS showing high value in bedside RBS monitoring glucometer, even after starting immediate management of DKA, patient DKA took more than 48 h to resolve as elimination of source which was deep organ (splenic abscess) in this case took 48 h for antibiotics and drainage to help for elimination and precipitating factor for DKA in this case.

TREATMENT AND OUTCOME

The initial treatment was started in line of DKA management, with IV fluids, insulin infusion, and serial ABG monitoring. In view of septicemia and immunocompromised status (HBA1C-14.9), empirical wide-spectrum antibiotic piperacillin-tazobactam and azithromycin (for atypical coverage) were started. After detecting splenic abscess in CT [Figure 1], immediate ultrasound-guided pigtail insertion was done by IR team, splenic abscess drained [Figure 2] and antibiotics were empirically upgraded to meropenem, metronidazole, and linezolid. Gradually patient's sepsis resolved, which also leads to settling of DKA. Moreover, the patient was shifted to basal bolus regimen of 12-12-12 U HIR and Lantus 14 U. His sugar values were in control, abdominal pain resolved, tachypnea resolved. Serial monitoring of routine blood investigations [Table 1], and ABG [Table 2] showed improving trend. Pigtail was removed after review USG showed no significant residual collection. The patient was discharged on the 16th day of hospital course with strict follow-up advice in the diabetic OPD.

CONCLUSION

The patient with poor glycemic control and with immunocompromised status presenting with DKA should always be screened for deep organ abscesses.

TAKE-HOME MESSAGE

Deep organ abscesses might be the precipitating infection for DKA and screening of deep organ abscesses should be done along with community-acquired pneumonia and urinary tract infection which is two of the most common source of infection. We should rely on CT scans, for diagnosis, more than USG. In resource settings like India, a high level of suspicion is required in OPD in patients with uncontrolled sugars, and a risk of DKA assessment should be done.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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