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Case Report

A Case of Hypertriglyceridemia Induced Acute Pancreatitis with DKA Resolved with Insulin Therapy

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Abstract

Purpose: To report a case of Hypertriglyceridemia induced acute pancreatitis with Diabetic Ketoacidosis and resolved with insulin therapy in Ethiopia.

Case Presentation: A 60 years old diabetic patient not on medication came to the emergency OPD with a compliant of abdominal pain. Depending on laboratory results and abdominal CT, diagnosis of Hypertriglyceridemia induced pancreatitis with Diabetic Ketoacidosis was made. The present a case of Diabetic ketoacidosis and hypertriglyceridemia induced pancreatitis in a type 2 diabetic patient.

Conclusion: Patient improved with insulin therapy, hydration and electrolyte supplement.

Keywords: *Diabetic Ketoacidosis; Hypertriglyceridemia Induced Acute Pancreatitis*

Background

Gallstones and alcohol abuse are the two most common causes of acute pancreatitis (AP). Hypertriglyceridemia is an uncommon but a well-established etiology of acute pancreatitis, with a reported incidence of 2 - 4%. National cholesterol Education Program ATP III categorizes triglyceride (TGs) level as normal (< 150), borderline high (150 - 199), high (200 - 499) and very high (> 500 mg/dL) [1]. Hypertriglyceridemia is considered a significant risk for pancreatitis when levels are > 1000 mg/dl (@). Diabetic Ketoacidosis (DKA) is a fatal manifestation reported in nearly 25 - 30% of patients with type 1 diabetes and in 4 - 29% of younger type 2 diabetics [3]. A marked elevation of serum triglycerides occurs during episodes of diabetic ketoacidosis (DKA) ($). This case describes a 60 yrs old male who presented with DKA, mild acute pancreatitis and severe hypertriglyceridemia. We discuss the hospital course and management of the case along with literature review.

Case Presentation

A 60 years old African man who is a known diabetic patient for the past 1 year on life style modification with no follow up. He currently presented with severe abdominal pain of 1-day duration associated with nausea and vomiting. Otherwise no history of fever or other

complaints. The patient had no history of alcohol ingestion. No history of gall bladder stone disease. At presentation to emergency OPD blood pressure was 150/80 pulse rate was 112 beats per min, respiratory rate 24 breaths per min and temperature was 36.7°C. Pertinent physical examination findings were signs of dehydration and severe direct tenderness on the left upper quadrant of the abdomen with guarding. He was investigated and pertinent laboratory findings were RBS of 454 mg/dl, urine ketone +3 and HgbA1C 8.4 (Table 1).

Name	Result	Unit	Reference Range	ABN
Hematology				
ESR	10	mm/hr	0 - 20	
Pancreatic enzymes				
Amylase	1848	u/l	< 86	High
Cardiac enzymes/function				
CK MB	2.01	ng/ml	< 5	
Troponin high sensitive				
Troponin I	< 0.1		< 0.1	
Electrolytes				
Potassium	4.99	mmol/l	3.6 - 5.5	
Sodium	148.1	mmol/l	135 - 155	
Chloride	118.2	mmol/l	95 - 110	High
Calcium Ionized	1.17	mmol/l	1.1 - 1.4	
Calcium Total	2.28	mmol/l	2.2 - 2.9	
PH Serum	7.83			
Potassium	3.39	mmol/l	3.6 - 5.5	Low
Sodium	140.6	mmol/l	135 - 155	
Chloride	95.1	mmol/l	95 - 110	
Calcium Ionized	0.74	mmol/l	1.1 - 1.4	Low
Calcium Total	1.44	mmol/l	2.2 - 2.9	Low
PH Serum	7.55			
Liver function test				
Alkaline Phosphatase	100	IU/L	64 - 306	
Serum Albumin	5.3	g/dl	3.5 - 5.5	
Total Protein	8.2	g/dl	6.6 - 8.7	
CBC				
WBC	11500	mm^3	4000 - 10000	High
RBC	5.10	10^12/L	4.00 - 5.50	
HGB	19.5	g/dl	12 - 18	High
HCT	49.0	%	37 - 54	
MCV	96.2	fl	76 - 96	High
RDWc	12.2	%	11.1 - 14	
MCH	38.2	pg	27 - 32	High
MCHC	39.8	g/dl	30 - 35	High

PLT	244	10^9/L	150 - 400	
LYM%	6.6	%	20 - 35	Low
MON%	5.6	%	2 - 8	
GRA%	87.8	%	50 - 72	High
WBC	8620	mm^3	4000 - 10000	
RBC	6.26	10^12/L	4.00 - 5.50	High
HGB	18.7	g/dl	12 - 18	High
HCT	61.3	%	37 - 54	High
MCV	98.0	fl	76 - 96	High
RDWc	11.0	%	11.1 - 14	Low
MCH	29.9	pg	27 - 32	
MCHC	30.5	g/dl	30 - 35	
PLT	223	10^9/L	150 - 400	
LYM%	9.9	%	20 - 35	Low
MON%	8.2	%	2 - 8	High
GRA%	81.9	%	50 - 72	High
Renal Function Test				
Bun/urea	25.3	mg/dl	10 - 50	
Creatinine	1.14	mg/dl	0.7 - 1.4	
Bun/urea	33	mg/dl	10 - 50	
Urine PH	6			
Urine SP.GR	1.020			
Urine Protein	Neg			
Urine Blood	+1			
Urine Glucose	+1			
Ketone	+3			
Urine Bilirubin	Neg			
Urine Urobilinogen	Neg			
Urine Nitrite	Neg			
Urine Leukocyte	Neg			
Lipid profile				
Cholesterol	488	mg/dl	< 220	High
Triglyceride	1440	mg/dl	< 180	High
HDL	31	mg/dl	40 - 110	Low
LDL	55	mg/dl	< 130	
Cholesterol	450	mg/dl	< 220	High
Triglyceride	642	mg/dl	< 180	High
HDL	20	mg/dl	40 - 110	Low
LDL	65	mg/dl	< 130	

Glucose				
Hemoglobin A1C%	8.4	%	4 - 5.7	High
Virology				
Hepatitis C Antibody	Negative			
Hepatitis B Surface Antigen	Negative			

Table 1: *Laboratory results.*

Abdominal ultrasound done on day 1 showed minimal ascites but no other finding. There was no features of gall bladder stone.

He was admitted to the Medical ICU and started on treatment for DKA with an assessment of Poorly controlled type II DM with DKA to rule out acute pancreatitis. Eight hours after insulin infusion was started along with potassium supplement patient was out of DKA but he had persistent severe abdominal pain and serum amylase level, lipid profile and Abdominal CT were done. Abdominal CT showed acute pancreatitis (Figure 1) and serum amylase level was 1845 u/l (21x above the higher normal of the laboratory), but it was impossible to get triglyceride level as the sample was too hyperlipidemic for the machine to read (Figure 2). He was started on management for Hypertriglyceridemia-induced acute pancreatitis with aggressive intravenous hydration, bowel rest, pain control and insulin and dextrose infusion in the ICU with close monitoring of RBS. 36 hrs after admission Serum triglyceride level was 1440 mg/dl. Treatment was continued and symptoms eventually resolved and feeding was started as tolerated.

Triglyceride level on day 4 of admission was 642 mg/dl. He was discharged on the 6th day with serum triglyceride level of 200 mg/dl. He was discharged with insulin, fibrate and a short appointment.

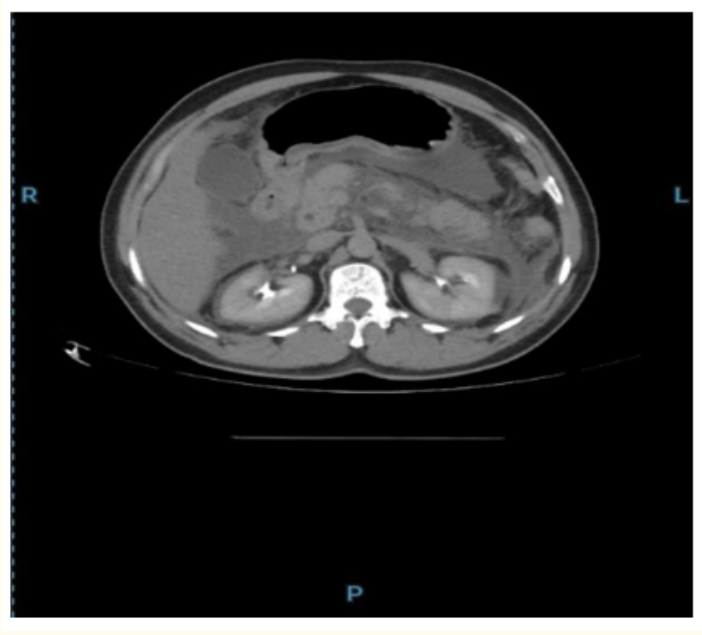

Figure 1: *Abdominal CT of our case showing hypo enhancing pancreas with per pancreatic and peritoneal fluid.*

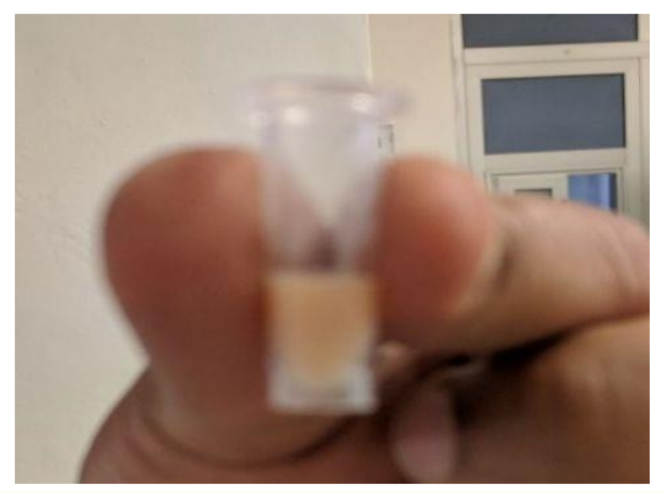

Figure 2: *Hyperlipidemic serum of current patient, 2020.*

Discussion

Acute pancreatitis is a potentially life-threatening acute inflammatory condition of the pancreas with a worldwide rising incidence over the past several decades. Although most cases are mild, there is a subset of patients with severe acute pancreatitis, where mortality may reach upto 30%.

Gallstones and alcohol abuse are the common causes of acute pancreatitis (AP). Hypertriglyceridemia is an uncommon but a well-established etiology of acute pancreatitis, with a reported incidence of 2 - 4% [1]. The exact mechanism involved in HTG-induced pancreatitis is not completely clear. HTG in animals with defect in LPL activity shown to be associated with pancreatitis and histological evidence of hemorrhage and necrosis of the pancreas leading to lobular destruction. Enzymatic degradation of triglyceride-rich lipoproteins generates proinflammatory free fatty acids that can lead to further damage of pancreatic acinar cells and microvasculature [6]. Poorly controlled diabetes mellitus can trigger hypertriglyceridemia. Marked elevation of serum triglycerides occurs during episodes of DKA [4]. There are a few reports of a combination of DKA and hypertriglyceridemia induced pancreatitis. There lies a clinical challenge in diagnosing DKA in the presence of acute pancreatitis and vice-versa. Each of the enzymes amylase and lipase are elevated in patients with DKA, which can be threefold or higher. Moreover, frequently, serum amylase can be normal in patients with acute pancreatitis secondary to hyperlipidemia due to the suppression or inhibition of serum amylase activity in such patients [7]. Therefore the diagnosis of pancreatitis in patients with DKA should be primarily based on the clinical findings and imaging.

Patients with hypertriglyceridemia induced pancreatitis present with typical symptoms of acute pancreatitis associated with high levels of serum triglyceride usually > 1000 mg/dl. Our patient presented with severe left upper quadrant abdominal pain with direct tenderness which lead to the workup on the line of pancreatitis. He has been diagnosed with type 2 DM a year back but refused to be started on medication and was on life style modification which leads to a poorly controlled DM with HgA1C of 8.4 which eventually lead to Hypertriglyceridemia. The DKA in our patient resolved with the initial hydration and insulin infusion with in eight hours. However, the abdominal pain persisted because of the pancreatitis. The eventual resolution of symptoms with continued insulin infusion and drop of serum triglyceride level from 1440 mg/dl to 200 mg/dl strengthens the diagnosis of hypertriglyceridemia induced acute pancreatitis.

Initial diagnostic and therapeutic procedures of acute hypertriglyceridemia induced acute pancreatitis should adhere to the same practice recommendations as established for other causes of acute pancreatitis (including intensive intravenous hydration, analgesic treatment and a fasting state). Earliest possible determination of serum triglyceride levels is crucial, since triglyceride levels are known to rapidly decrease within the first 48h of the onset of acute pancreatitis. Identification of possible secondary causes of hypertriglyceridemia (e.g. drug- associated hypertriglyceridemia, diabetes mellitus and hypothyroidism, among others) is important and requires a thorough medical history and examination in order to identify these causes quickly. Since there are no commonly accepted guide lines on the treatment of HTGP, multiple treatment modalities have been suggested so far. Insulin and/or heparin application, plasmapheresis and oral antihyperlipidemic drug treatment have been evaluated in clinical studies and case reports [5]. We started our patient on intensive hydration and Insulin infusion which helped for DKA, Hypertriglyceridemia and for the pancreatitis. His serum triglyceride level started drooping with the insulin infusion. We didn't consider the other treatment options since he was improving.

Conclusion

In conclusion though there is controversy about the severity and outcome of hypertriglyceridemia induced acute pancreatitis as compared with acute pancreatitis of other etiologies taken together, evidence is rising that hypertriglyceridemia induced acute pancreatitis is associated with a more severe disease and a higher rate of complications [6,8]. These patient do benefit from early diagnosis and management so we have to be highly suspicious and do early lipid profile and imaging in patients with poorly controlled diabetes mellitus presenting with symptoms of pancreatitis. More reports, researches and established guide lines would also help in choosing the modality of treatment.

Ethics and Consent Statement

Written informed consent was obtained from the patient to have the case details and any accompanying images published. Ethical approval for this report was obtained from ICMC Hospital Ethics Committee (ICMC.RESEARCH. REC.13/123/20).

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Disclosure

The authors report no conflicts of interest in this work.

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