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General Medicine

A CASE OF HYPERTRIGLYCERIDEMIA INDUCED ACUTE PANCREATITIS

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ABSTRACT Acute Pancreatitis is characterized by inflammation and destruction of pancreatic tissue. It is an acute medical condition that requires emergent care. Information regarding the prevalence of this disorder in India is scant. The common causes of this disorder are Gall stones and Alcohol consumption. Hypertriglyceridemia is an uncommon cause of Acute Pancreatitis. Here we present a case report of Acute pancreatitis caused by Hypertriglyceridemia. This Case report highlights the necessity of being aware of and evaluating for less common causes of this commonly encountered disorder in certain high risk patients.

KEYWORDS : Hypertriglyceridemia, Acute pancreatitis

INTRODUCTION –

Acute Pancreatitis is a disorder characterized by acute inflammation of pancreas. The most common triggers for this disorder are Gall stones and Alcohol. Less common causes include Hypercalcemia, Hypertriglyceridemia, Drugs and toxins, Trauma and Post surgical. Hypertriglyceridemia is thought to account for 1.3 – 3.8% of cases of Acute Pancreatitis. Usually, the Serum Triglyceride levels exceed 1000mg/dL in such cases. These patients are more prone to develop recurrent attacks of Acute Pancreatitis. These patients may be given Insulin or Heparin or Plasmapheresis as an initial therapy along with the usual Fluid and Analgesic therapy for Acute Pancreatitis. Outpatient therapy includes control of underlying predisposing factor for hypertriglyceridemia such as Diabetes Mellitus, Lipid lowering agents & Weight Loss.

Case Presentation :-

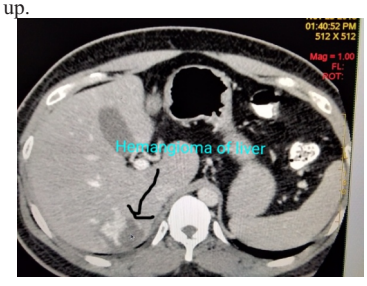
A 31 Year old Male patient named Mahaboob Basha came from Thandrapadu , Kurnool with Chief Complaints of Pain Abdomen since 1 month & Fever since 1 day. Pain was located in Epigastric region and was associated with Burning sensation, worsened on lying down and relieved by sitting upright. Fever was of low grade, intermittent & relieved with antipyretics.T here was no history of vomitings, abdominal distension, Yellowish discoloration of eyes. The patient is a Known case of Diabetes Mellitus for 2 years and was on treatment with Glimiperide 1mg & Metformin 500mg OD for 2 months. He has been using PPIs for pain abdomen for 1 month. He had a history of Pulmonary Tuberculosis at 5 Years of age and was treated with AntiTB Therapy for 1 year. He is married for 5 years, has1 male child & consumes mixed diet. He is an occasional alcoholic and takes 4-5 times/year, 750ml of Beer each time. Family history revealed Type 2 DM in mother & father & uncle.

On examination, the patient was conscious & coherent, BMI – 24.3 kg/m² There was no pallor / Icterus /Cyanosis/Clubbing/ Edema /Lymphadenopathy. No Xanthomas/xanthelasmas. Pulse was 98bpm, regular. BP was 140/110mm Hg. Respirations were normal. His ABPI was 1.Systemic examination revealed tenderness in epigastric region. CVS – S1, S2 heard, RS – Vesicular breath sounds heard, NS examination including Fundus was normal.

Patient was provisionally diagnosed as T2DM with Acute pancreatitis and was worked up for the same. Investigations revealed Hb – 12.9 g/dL WBC count - 4000 /µL PLT Count – 143000/ µL Sr Creatinine – 0.6 mg/dl, Blood Urea - 17 mg/dl , Sr Bilirubin – 0.5 mg/dL , ALT- 29 U/L , AST -23 U/L, ALP – 91 U/L RBS – 302 mg/dL , Sr. Amylase – 430 U/L , Sr. Lipase - 253 U/L ,Serum Triglycerides – 1096 mg/dL

CECT scan of abdomen and pelvis was done which revealed multiple small non-enhancing hypodense areas with bulky head & uncinate process with mesenteric fat stranding around head & uncinate process of pancreas which was suggestive of ACUTE FOCAL PANCREATITIS OF HEAD & UNCINATE PROCESS OF PANCREAS, Also seen were Hemangioma in segment 8 of liver & Cortical cust in upper pole of left kidney.

Patient was finally diagnosed as Uncontrolled T2 DM with Acute pancreatitis secondary to Hypertriglyceridemia and was managed with IV Fluids, Premixed Insulin for glycemic control and was placed on Fibrate therapy for Dyslipidemia. He recovered symptomatically and Blood sugars became stabilized. He was discharged home with advice to follow up.



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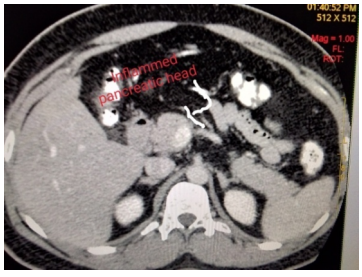

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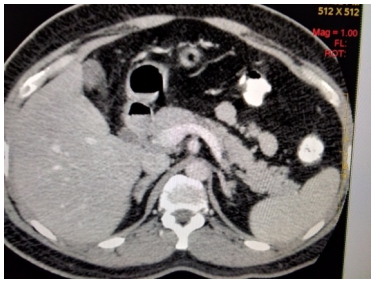

3.

CECT Abdomen Images of the patient

Discussion :-

Hypertriglyceridemia is an important cause of acute pancreatitis. Early clinical recognition is important to provide appropriate therapy and to prevent further episodes. Hypertriglyceridemia is defined by fasting serum triglyceride level of >150 mg/dL (1.7 mmol/L).It is classified as Mild - 150 to 199 mg/dL , Moderate - 200 to 999 mg/dL , Severe - 1000 to 1999 mg/dL,Hypertriglyceridemia-induced pancreatitis causes 1 to 14 % of all cases of Acute Pancreatitis. The risk of developing acute pancreatitis is approximately 5 % with serum triglycerides >1000 mg/dL and 10 to 20 %with triglycerides >2000 mg/dL. Hypertriglyceridemia can be Primary, seen with Types I, IV, V Dyslipidemias or secondary to Diabetes Mellitus, Drugs like Beta blockers, Thiazide diuretics, Protease Inhibitors; Pregnancy, Hypothyroidism & Alcohol. The breakdown of triglycerides into toxic free fatty acids (FFA) by pancreatic lipases is the cause of lipotoxicity to the pancreas. In most cases, the Hypertriglyceridemia is transient and returns to near normal within two to three days. The initial presentation of hypertriglyceridemia-induced pancreatitis is similar to that of acute pancreatitis due to other causes with persistent severe epigastric pain, nausea, and vomiting. Most patients present with symptoms in the fifth decade of life. However, inherited disorders can present with acute pancreatitis in early childhood or adolescence. Physical examination may reveal eruptive xanthomas over the extensor surfaces of the arms, legs, buttocks, and back due to persistent hyperchylomicronemia and hepatosplenomegaly from fatty infiltration. Lipemia retinalis may be seen in patients with triglyceride concentrations exceeding 4000 mg/dL. Management of patients with hypertriglyceridemia-induced pancreatitis includes treatment of acute pancreatitis & reduction of serum triglyceride levels. The main treatment modalities for initial management of hypertriglyceridemia are apheresis & insulin. Insulin reverses the stress-associated release of fatty acids from adipocytes, & promotes intracellular triglyceride generation within adipose tissue, and fatty acid metabolism in insulin sensitive cells, IV insulin may be more effective than subcutaneous insulin in severe cases and is easier to titrate than subcutaneous administration. The efficacy of Therapeutic Plasma Exchange in reducing the severity of hypertriglyceridemia-induced acute pancreatitis has not been established. Therapies with uncertain benefit include heparin & Hemofiltration. Patients with Hypertriglyceridemia induced Pancreatitis require long-term therapy to prevent recurrent acute pancreatitis and to prevent other complications of HTG . This consists of pharmacologic therapy, dietary fat restriction, weight loss in obese patients, aerobic exercise, avoidance of concentrated sugars and medications that raise serum triglyceride levels, and strict glycemic control in diabetics.

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