

Acute Recurrent Pancreatitis Induced By Hypertriglyceridemia

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Introduction :

Hypertriglyceridemia (HTG) is a rare cause of pancreatitis. Hyperlipidemic pancreatitis (HLP) secondary to HTG presents typically as an episode of acute pancreatitis or recurrent acute pancreatitis or rarely as chronic pancreatitis [1]. The typical clinical profile of HLP is a patient with preexisting lipid abnormality along with the presence of secondary factors (such as poorly controlled diabetes mellitus, alcohol abuse, pregnancy, or a medication) that can induce HTG [1]. It is generally accepted that a TG level more than 1000 mg/dL is needed to precipitate an episode of acute pancreatitis [2]. It is postulated that hydrolysis of TG by pancreatic lipase into free fatty acid is toxic to pancreatic endothelium and acinar cells [3].

Plasmapheresis has been claimed to reduce triglyceride level rapidly in HLP [4-11] and is believed to halt the progression of HLP [8-10]. Actually, experiences of plasmapheresis in HLP are limited and only sporadic cases were reported [5-11].

First case: O. H. M. is sixteen years old female, living in Baghdad; she was student, admitted to hospital on 2nd Of August 2014 complaining from severe epigastric pain for several hours duration. The condition started gradually over few hours as an abdominal pain that was mild and tolerable discomfort at the start but soon became severe in

intensity and burning in nature located in epigastric region and radiated to the back, associated with nausea and vomiting.

The pain was more intense when the patient lied supine and she had been often obtained relief by sitting with trunk flexed and knees drawn up. The patient gave history of previous similar attacks, she had been seeking medical advice frequently and OGD was done to here, which revealed antral gastropathy, she was treated with PPI with only mild improvement. She reported no fever, hematemesis, melena, diarrhea or weight loss.

On abdominal examination, there was epigastric tenderness. Abdominal U/S done and was not conclusive, abdominal CT scan with I.V contrast done revealing atrophic irregular outline pancreases showing segmental dilatation of the pancreatic duct measuring 7 mm in the widest part; picture consistent with chronic

Pancreatitis. EUS revealed hypoechoic atrophy of pancreas with dilatation of pancreatic duct of 8 mm, picture of chronic atrophic pancreatitis.

A diagnosis of acute recurrent pancreatitis due to hypertriglyceridemia had been made and medical therapy with I.V fluid, analgesia & lipid lowering agent was started.

Table (1): Laboratory investigations

Parameter	Case 1	Case 2	Case 3
RBS (md/dl)	104	82.5	-
Blood urea (md/dl)	26	25	-
Serum creatinine (md/dl)	0.6	0.55	-
S. calcium (mg/dl)	6.5	12.1	-
S. uric acid (mg/dl)	7.8	-	-
TSB (mg/dl)	0.6	-	-
S ALP (KAU)	7	-	-
S. GPT (ALT) U/L	28	7.8	-
S. GOT (AST) U/L	89	14.7	-
S K mmole/L	6.1	-	-
S Na mmole/L	134.5	-	-
S Cl mmole/L	104.7	-	-
S triglyceride mg/dl	1027	1088.6	1420
S cholesterol mg/dl	213	198.5	288
S LDL mg/dl	-	94.7	92
VLDL mg/dl	99	-	152
HDL mg/dl	41	-	36
S amylase IU/L	251	244	68.2
S lipase IU/L	413	-	48.8

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A decision for ERCP with pancreatic stent insertion had been taken in order to prevent further attacks of acute pancreatitis. On 11th of August, ERCP was done with pancreatic plastic stent 5 Fr*10 cm was inserted. Patient was discharged on Gemfibrozil 600 mg twice daily, Omega-3 1gm twice daily, pancreatic enzymes capsule three times daily & PPI twice daily. In addition, patient advised about dietary fat restriction. Three weeks later, patient was seen for follow up of her condition, she was completely pain-free, Serum Amylase was 61 IU/L, abdominal U/S revealed pancreas is of normal shape and texture, with stent in situ; normal pancreatic duct. At same time, duodenoscopy was done for stent removal. All subsequent readings of TG were ranged 180-220 mg/dl. Second case R. H. M. (old sister of O. H. M.) is twenty years old female, living in Najaf, she is married, housewife admitted on 21st of September 2014 complaining from repeated vomiting & epigastric pain for one day duration. The condition started as a gradual onset of epigastric pain associated with nausea over several hours, then after the pain became more severe radiate to back, worsening with lying flat & relieved by leaning forward associated with vomiting. She had no similar attack previously; she had been diagnosed as a case of familial hypertriglyceridemia six months ago, starting lipid lowering therapy (Gemfibrozil 600 mg twice daily & Omega-3 1gm three times daily). Abdominal U/S was done the pancreas was slightly prominent head possible focal pancreatitis, homogenous texture with no SOL. After reviewing of current available guidelines & literatures about role of plasma lipid pheresis in management of hyperlipidemic phase of acute pancreatitis; and the good results obtained in several centers, a decision made about starting plasma lipid pheresis due to failure of medical therapy in lowering plasma lipids & to prevent recurrent acute pancreatitis. This approach was done for the first time in our center (Baghdad teaching hospital) and even in our country. Patient was discussed about this approach, and we explained the risk of such high level of plasma lipids without intervention by inducing recurrent attacks of acute pancreatitis beside other complications of hyperlipidemia. Patient agreed and first session of plasma lipid pheresis was done on 22nd of September, one week later a second session was done. Serum triglyceride decreased from 1088.6 mg/dl to 593 mg/dl. On the 1st of October the 3rd session was done and serum triglyceride was 211 mg/dl after this session. Patient was pain free, discharged on Gemfibrozil 600 mg twice daily, Omega-3 1gm three times daily & dietary fat restriction. She was seen after 2 weeks for further follow-up, she was feeling well, and her triglyceride level was 198 mg/dl & normal serum lipase & amylase. Thus, therapeutic plasma exchange was

effective in lowering Amylase, triglyceride, improves the attack of hyperlipidemic pancreatitis and aids in preventing recurrent attacks. Third case H. A. Q. (a cousin of previous two cases), two years old male child, admitted on 21st of January 2015 complaining from abdominal pain with repeated vomiting for several days duration. Abdominal U/S was done and reveal pancreas looks slightly enlarged for patient age, hazy contour, uniform hypoechoic texture, no focal SOL, no duct dilatation, just little free fluid collected within epigastric region, few inflammatory L. N. Seen in para pancreatic region.

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