Case Report

An interesting case of hypertriglyceridemia (HTG) induced pancreatitis

Lokesh Kumar A, Devipriya S, Ramkumar M, Jagadeesan M*

Department of General Medicine, Saveetha Medical College Hospital, India
*Corresponding author email: **drjagadeesan@gmail.com**

International Archives of Integrated Medicine, Vol. 9, Issue 11, November, 2022.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 2-11-2022 **Accepted on:** 9-11-2022
Source of support: Nil **Conflict of interest:** None declared.

How to cite this article: Lokesh Kumar A, Devipriya S, Ramkumar M, Jagadeesan M. An interesting case of hypertriglyceridemia (HTG) induced pancreatitis. IAIM, 2022; 9(11): 29-32.

Abstract

Hypertriglyceridemia induced pancreatitis is the third most common cause of acute pancreatitis after gall stones and alcohol. Most of the patients with Hypertriglyceridemia (HTG) has primary or genetic defect in lipoprotein metabolism. Groove pancreatitis is a rare form of Pancreatitis with most patients being male and history of alcohol consumption. Here we report a case of 33 year old female who is a known diabetic, presented with diabetic ketoacidosis. Her serum lipase was 3133 U/l (<40U/l). Here we report a case of familial dyslipidemia with severe hypertriglyceridemia causing pancreatitis involving the Groove presenting with Diabetic Ketoacidosis.

Key words

Hypertriglyceridemia, Pancreatitis, HTG.

Introduction

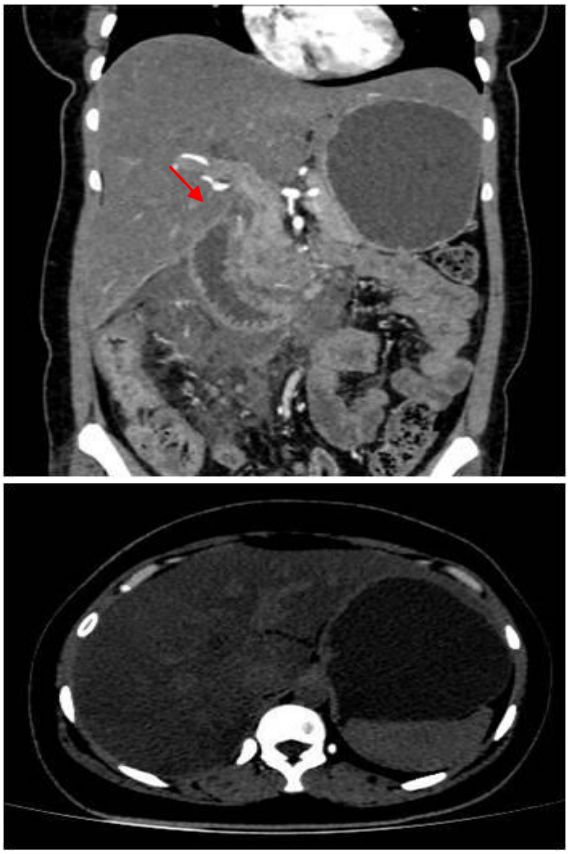
Acute pancreatitis is a painful inflammatory condition of the pancreas. Despite great advances in critical care medicine, the mortality rate of acute pancreatitis has remained at about 20%. Acute pancreatitis secondary to HTG is seen in patients with disorders of lipid metabolism and is highly related to the subsequent development of cardiovascular co-morbidities [1]. Alcohol consumption and gallstones represent the most common etiologies while pancreas divisum, annular pancreas, pancreatic trauma, hypercalcemia, hyperlipidemia, autoimmune, and medications are less common causes of acute pancreatitis [1]. HTG is an uncommon but well-established cause of acute pancreatitis. It is generally believed that a triglyceride level of more than 1,000 mg/dl is needed to precipitate an episode of acute pancreatitis. The etiology should be suspected in obese, diabetic or hyperlipidemic patients. Groove pancreatitis is a segmental chronic pancreatitis that affects the anatomical area between the pancreatic head, the duodenum, and the common bile duct, which is referred to as the groove area. In 1973, Becker [2] first used

the German term 'Rinnenpankreatitis' to describe segmental pancreatitis of the groove area and this was later translated into groove pancreatitis by Stolte, et al. [3] in 1982.

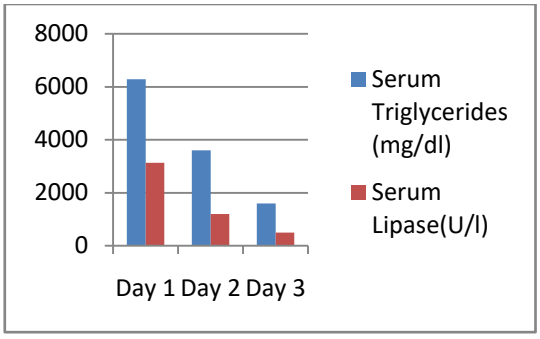
Case report

A 33 year old female who is a known diabetic with poor compliance, non-smoker and non-alcoholic presented with complaints of epigastric pain radiating to the back aggravated on supine position and consumption of food and vomiting for 3 days. She had grade 4 dyspnea. She had similar complaints one year back. Her brother also had dyslipidemia.General examination showed signs of dehydration and had tachypnea. Vitals showed respiratory rate of 28/min. Other vitals were stable.Systemic examination revealed hepatomegaly. Her initial laboratory investigations showed hemoglobin - 13.2 g/dl, total leucocyte count of 14,400 cells/cumm (N-72%, L-20%, M-6%, E-2%, B-0%), Platelets-3,20,000 cells/cumm. Random blood glucose was 513 mg/dl and HBA1C was 11.2%. Urine ketones were positive (3+). Arterial blood gas showed high anion gap metabolic acidosis (pH-7.112, Hco3- 8, pCo2- 27, AG-18). Renal function tests, serum electrolytes, liver function tests, coagulation profile were normal. Cardiac evaluation was normal. Her serum lipase was increased several folds with value of 3133 U/l (<40U/l). Her CECT abdomen (**Photograph - 1**) findings were suggestive of acute pancreatitis and there were extensive peri-pancreatic inflammatory changes in the 2nd and 3rd part of duodenum suggestive of Groove pancreatitis and a enlarged liver 20 cm with diffuse hypoattenuation suggestive of Fatty Liverwith no evidence of pancreaticnecrosis. Lipid profile revealed total cholesterol-1099 mg/dl (160-200 mg/dl), triglycerides- 6280 mg/dl (50-150 mg/dl), HDL-25 mg/dl (30-60 mg/dl), LDL-600 mg/dl (80-175 mg/dl), VLDL-470 mg/dl (2-30 mg/dl).Modified ransons score at admission – 1 point, at 48 hours- 2 Points and BISAP score - 1 point. Initially IV Fluids along with IV insulin were started in view of Diabetic ketoacidosis and analgesics for symptomatic relief. Her symptoms started improving. Repeat serum Lipase at day 3 was 500 and triglycerides were 1600 (**Photograph - 2**). She was discharged with Insulin, statin, fenofibrate and omega 3 fatty acid. She is currently under follow up and her serum lipase, amylase and triglycerides were normal.

Photograph – 1: CECT Abdomen showing moderate peripancreatic fat stranding with reactive inflammatory changes in the 2nd and 3rd part of duodenum suggestive of Groove pancreatitis and enlarged liver 20 cm with diffuse hypoattenuation suggestive of Fatty Liver.



Photograph – 2: Trends of serum triglyceride and lipase levels.



Discussion

Hypertriglyceridemia induced Pancreatitis make up 1 to 35% of all cases of Acute Pancreatitis and 56% of pancreatitis cases during pregnancy. In a study by Nawaz, et al. [4], patients with hypertriglyceridemia induced pancreatitis were younger in age, predominantly male and had a personal history of diabetes or obesity as compared to acute pancreatitis patients with Hypertriglyceridemia.

A recent prospective RCT by He, et al. on 66 hypertriglyceridaemia-induced acute pancreatitis patients received either early high-volume haemofiltration (HVHF) or low molecular weight heparin (LMWH) plus insulin as an emergent triglyceride-lowering therapy. As per the results, HVHF lowered triglyceride levels more quickly than LMWH plus insulin therapy, but no difference was noted in terms of clinical outcomes, local pancreatic complications, need of surgery and cost [6].

A case control study by Oza, et al. involving Thirteen patients with Groove pancreatitis in which 84.6 % were males, all with a history of smoking (mean, 37.54 ± 17.8 pack-years), were identified. Twelve patients (92.3%) had a history of heavy alcohol drinking [7].

Clinicians must consider peripancreatic cancer for a differential diagnosis of groove pancreatitis. Other differential diseases include autoimmune pancreatitis and duodenal hamartoma [8]. Groove pancreatitis should always be considered when a patient has a history of excessive alcohol consumption. But in our case there is no history of alcohol consumption and hypertriglyceridemia was identified as the etiology making it a rare entity.

Conclusion

Acute Pancreatitis secondary to HTG is well established cause but a rare entity. Finding out the etiology and timely intervention can greatly reduce morbidity and mortality in acute pancreatitis.

References

1. Kalidindi L, Linkeshavan M, Kalappan MK, Mohanan J, Rajendran K. An interesting case of acute pancreatits. Int J Adv Med., 2022; 9: 958-60.
2. Becker V. Bauchspeicheldrüse; in Doerr W, Seifert G, Uhlinger E (eds): Spezielle pathologische Anatomie. Berlin, Springer, 1973, vol 4.
3. Stolte M, Weiss W, Volkholz H, Rösch W. A special form of segmental pancreatitis: ‘groove pancreatitis’. Hepatogastroenterology, 1982; 29: 198–208.
4. Nawaz Haq, Koutroumpakis Efstratios, Easler Jeffrey, Slivka Adam, Whitcomb David C, Singh Vijay P, Yadav Dhiraj, Papachristou Georgios I. Elevated Serum Triglycerides are Independently Associated With Persistent Organ Failure in Acute Pancreatitis. American Journal of Gastroenterology, October 2015; 110(10): 1497-1503. doi: 10.1038/ajg.2015.261
5. Warshaw AL, Bellini CA, Lesser PB. Inhibition of serum and urine amylase activity in pancreatitis with hyperlipemia. Ann Surg., 1975; 182(1): 72-75. doi:10.1097/00000658-197507000-00014
6. He WH, Yu M, Zhu Y, Xia L, Liu P, Zeng H, Zhu Y, Lv NH. Emergent Triglyceride-lowering Therapy With Early High-volume Hemofiltration Against Low-Molecular-Weight Heparin Combined With Insulin in Hypertriglyceridemic Pancreatitis: A Prospective Randomized Controlled Trial. J Clin Gastroenterol., 2016 Oct; 50(9): 772-8. doi: 10.1097/MCG.0000000000000552. PMID: 27574886.
7. Oza, Veeral M., Skeans, Jacob M., Muscarella Peter, Walker Jon P., Sklaw Brett C., Cronley Kevin M., El-Dika Samer, Swanson Benjamin, Hinton Alice, Conwell Darwin L., Krishna

Somashekar G. Groove Pancreatitis, a Masquerading Yet Distinct Clinicopathological Entity: Analysis of Risk Factors and Differentiation. Pancreas, August 2015; 44(6): 901-908. doi: 10.1097/MPA.0000000000000351

8. Zamboni G, Capelli P, Scarpa A, Bogina G, Pesci A, Brunello E, Klöppel G. Nonneoplastic mimickers of pancreatic neoplasms. Arch Pathol Lab Med., 2009; 133: 439–453.