Scholars Journal of Medical Case Reports **ISSN 2347-6559 (Online)**
Sch J Med Case Rep 2016; 4(4):206-208 **ISSN 2347-9507 (Print)**

(An International Publisher for Academic and Scientific Resources) DOI: 10.36347/sjmcr.2016.v04i04.008

Hypertriglyceridemia-Induced Acute Pancreatitis: Case Report

Nevzat Gözel[1], Erhan Onalan[2], Ramazan Ulu[3], Fatih Demircan[4], Faruk Kilinc[5], Ahmet Karatas[6], Orhan Kürşat Poyrazoğlu[7], Kader Uğur[8], Emir Dönder[1]

[1]Firat Medical Faculty Department of Internal Medicine, Elazig, Turkey
[2] Ercis State Hospital Department of Internal Medicine, Van, Turkey
[3]Firat Medical Faculty Department of Nephrology, Elazig, Turkey
[4]Private Etic Life Medical Center Department of Internal Medicine, Istanbul, Turkey
[5] Elaziğ State Hospital Department of Endocrinology, Elaziğ, Turkey
[6]Firat Medical Faculty Department of Rheumatology, Elazig, Turkey
[7]Firat Medical Faculty Department of Gastroenterology, Elazig, Turkey
[8]Firat Medical Faculty Department of Endocrinology, Elazig, Turkey

***Corresponding author**
Nevzat Gözel
Email: drngozel@hotmail.com

Abstract: Acute pancreatitis (AP) in a sudden pancreatic inflammation characterized by increased pancreatic enzymes in serum as well as abdominal pain. It is most commonly due to gallbladder stone and alcohol consumption. One of the important reasons of AP that are not common includes hypertriglyceridemia. In this case report, we present a patient diagnosed with hypertriglyceridemia-induced AP.
Keywords: pancreatitis, hypertriglysceridemia, amylase

INTRODUCTION

Acute pancreatitis (AP) is a common disease of the digestive system with high morbidity and mortality rates. Its incidence is around 50 per 100,000 varying between societies [1]. Etiologic factors for the disease may either include hyperlipidemia, bacterial, viral and parasitic agents, interventional procedures such as ERCP, drug use and in particular gallbladder stones and alcohol use or it may be idiopathic. The rate for AP related to drug use has been reported to be around 6% [2].

PATHOGENESIS

The region where the most intensive distribution of triglycerides occurs within the pancreas and surrounding connective tissues is the interstitial region having the most lipase activity. When attached to albumins, free fatty acids do not have any toxic effect to cells. However, excessive amounts of free fatty acids emerged from the effect of pancreatic lipase in the presence of hypertriglyceridemia lead to exceeded capacity of albumin binding. As a result, circulation of released fatty acids increases the permeability of pancreatic acinar cells and capillary basal membranes resulting to emerged cytotoxic effects and induced pancreatitis. The above mentioned series of events was revealed by means of animal studies, and it has proved that, contrary to common belief in past, free fatty acids do not form micro-thrombus leading to obstructed small capillaries and ischemia [3, 4, 5].

CASE REPORT

Our case was a 49 year-old woman patient who diagnosed with type II diabetes mellitus 3 years ago and thus receiving mixtureinsulin twice a day. Her complaints included severe stubbing-like abdominal pain lasting for 4 days from epigastria region extending towards her back in a band-like fashion which alleviated when bending forward, and which aggravated after eating, along with nausea and vomiting. She suffered a continuous nausea with 3 or 4 times a day of vomiting. One day, the patient applied to the emergence department of our hospital due to intensified complaints.

Physical examination revealed that vital findings were stable with a mild tenderness in epigastria region. The remaining systemic examinations were evaluated as normal.

In laboratory assessment, the results were as follows: elevated white blood cell count of 14,900 in complete blood count; higher biochemical results including random blood glucose of 249 mg/dL (Normal: 75-115 mg/dl), amylase of 283 U/L (Normal: 28-100 U/L), lipase of 184 U/L (Normal: 7-60 U/L), triglyceride of 2,249 mg/dL (Normal: 40-180 mg/dL). The results of AST, ALT, alkaline phosphatase, gamma-glutamyl transferase, bilirubins, urea, creatine and electrolytes were measured as normal. Imaging

studies revealed deterioration in pancreatic borders and a minimal collection of loculated fluids around the pancreatic tail in the upper abdominal CT. Contamination in mesenteric structures surrounding the pancreas and prominence in gerotafacia at left were determined(Pıcture 1,2).

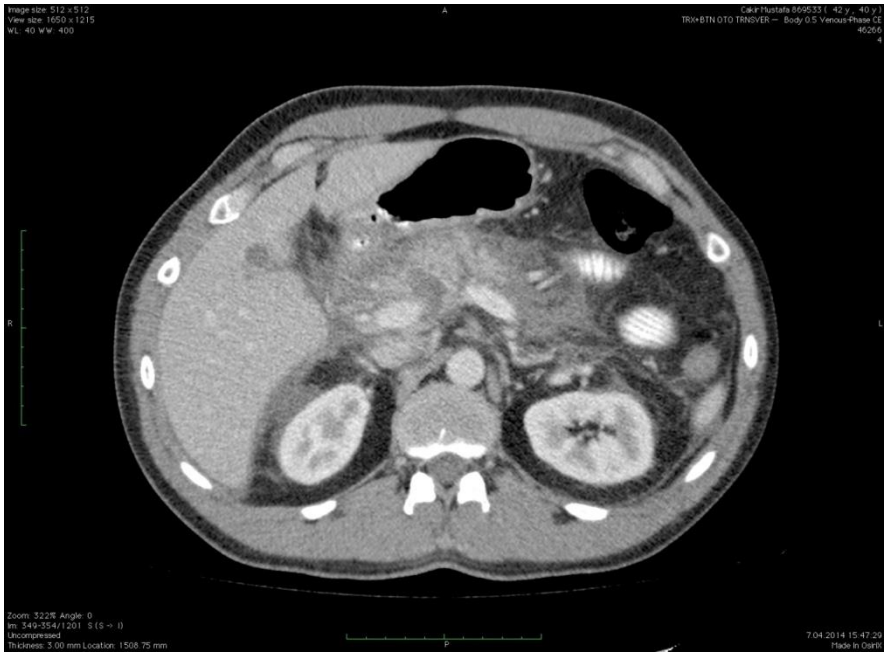

Fig. 1: CT showing upper abdominal

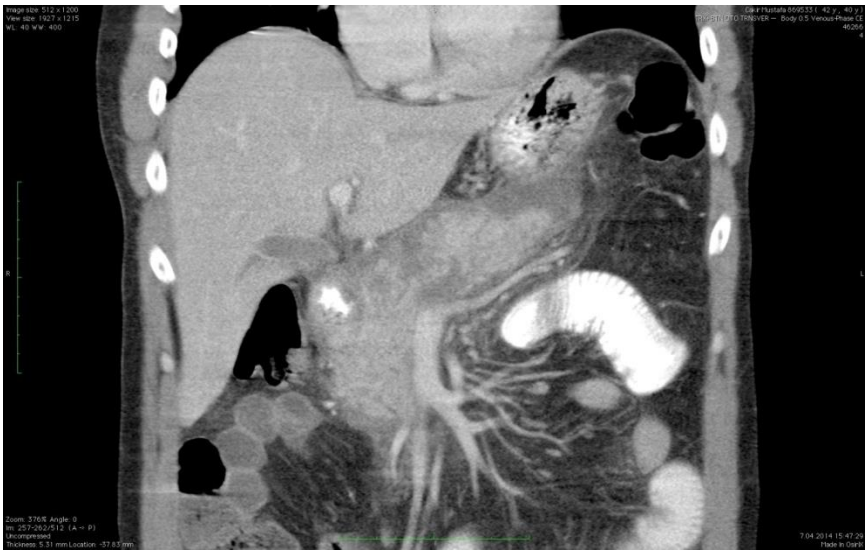

Fig. 2: CT showing Contamination in mesenteric structures surrounding the pancreas

The present imaging findings were interpreted as in favor of acute pancreatitis. The patient was hospitalized to the gastroenterology clinic of our hospital with diagnoses of hypertriglyceridemia and acute pancreatitis. Obliteration at peripancreatic adipose tissue and partial heterogeneity in pancreatic parenchyma were observed from T2 images in MR cholangiography at the clinic. The present findings could confirm the diagnosis of acute pancreatitis.

The patient's oral intake was stopped. The treatment comprised 5000 units of heparin 2x1 intravenous (i.v), 500 cc of 30% dextrose 2x1 i.v. with 50 cc being administered per hour (25 units of crystallized insulin was placed in each 500 cc volume), 500 cc of 5% dextrose 2x1 i.v. with 50 cc being administered per hour, 5 units of crystallized insulin in 100 cc of physiological saline solution with 20 cc being administered per hour (so as to administer 1 unit of crystallized insulin per hour).This treatment continued for 5 days, with a follow-up of blood sugar per hour. The patient's blood sugar levels were measured between 120 and 180 mg/dL. The patient presented with fever on the second day of treatment and therefore blood, urea and throat cultures were empirically collected, followed by administering antibiotics

(Ceftriaxone disodium hemiheptahydrate 1 g 2x1 i.v.). No growth was observed in the culture samples. Antibiotic treatment was terminated after the 10th day. The patient cleared up with complete clinical findings improved, and her triglyceride levels were measured as 415 mg/dL on the 5th day of treatment.

While Apache II score and mortality rate were 13 and 16.5% before the treatment, these values were calculated as 9 and 9.9% after the treatment, respectively. The patient received Gemphibrozile 600 mg 1x1 in the form of tablet along with recommending her previously used insulin regime, and she was discharged to visit the gastroenterology outpatient clinic again for check-up after 10 days. At that time, her triglyceride levels were measured as 208 mg/dL.

DISCUSSION

Acute pancreatitis (AP) is a common disease of the digestive system with high morbidity and mortality rates. Its incidence is around 50 per 100,000 varying between societies [1]. Etiologic factors for the disease may either include hyperlipidemia, bacterial, viral and parasitic agents, interventional procedures such as ERCP, drug use and in particular gallbladder stones and alcohol use or it may be idiopathic. The rate for AP related to drug use has been reported to be around 6% [2]. The region where the most intensive distribution of triglycerides occurs within the pancreas and surrounding connective tissues is the interstitial region having the most lipase activity. When attached to albumins, free fatty acids do not have any toxic effect to cells. However, excessive amounts of free fatty acids emerged from the effect of pancreatic lipase in the presence of hypertriglyceridemia lead to exceeded capacity of albumin binding. As a result, circulation of released fatty acids increases the permeability of pancreatic acinar cells and capillary basal membranes resulting to emerged cytotoxic effects and induced pancreatitis. The above mentioned series of events was revealed by means of animal studies, and it has proved that, contrary to common belief in past, free fatty acids do not form micro-thrombus leading to obstructed small capillaries and ischemia [3, 4, 5].

CONCLUSION

In conclusion, hyperlipidemia-induced acute pancreatitis is the third most common etiology after gallbladder stone and alcoholism. Presence of hyperlipidemia should be studied if the first two factors could not be determined in the etiology of acute pancreatitis.

REFERENCES

1. Takeda K, Takada T, Kawarada Y, Hirata K, Mayumi T, Yoshida M, Sekimoto M, Hirota M, Kimura Y, Isaji S, Koizumi M; JPN Guidelines for the management of acute pancreatitis: medical management of acute pancreatitis. Journal of hepato-biliary-pancreatic surgery, 2006; 13(1):42-7.
2. Vidarsdottir H, Möller PH, Vidarsdottir H, Thorarinsdottir H, Björnsson ES; Acutepancreatitis: a prospective study on incidence, etiology, andoutcome. Eur J Gastroenterol Hepatol., 2013;25(9):1068-1075.
3. Saharia P, Margolis S, Zuidema GD, Cameron JL; Acute pancreatitis with hyperlipemia: studies with an isolated perfused canine pancreas. Surgery, 1977; 82(1):60-7. 4.
4. Cameron JL, CriserC, Margois S, De Meester TR, Zuidenıa CD; Acute pancreatitis with hyperlipemia. Surgery, 1971;70:53-61.
5. Unal H; Acute pancreatit. Çağdaş Surgery Journal, 1988; 2: 68-87.