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Research Article

Hypertriglyceridemia in Acute Pancreatitis

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Abstract: Hypertriglyceridemia is a rare, but well known cause of acute pancreatitis. Presentation is often similar to other forms of acute pancreatitis, with lipemic serum usually the only distinguishing initial sign. Typically hypertriglyceridemia-induced pancreatitis occurs in a patient with a pre-existing lipid abnormality, along with the presence of a secondary precipitating factor e.g. poorly controlled diabetes, alcohol or medication. Secondary causes of hypertriglyceridemia have to be ruled out. Although the serum triglyceride threshold for considering hypertriglyceridemic pancreatitis is generally considered to be in the range of 1000mg/dl, the severity, clinical course and complication rate do not correlate with lipid levels. The mainstay of therapy is dietary restriction of fatty meal and fibric acid derivatives.

Keywords: Acute Pancreatitis, Fibric acid derivatives, Hypertriglyceridemia, Hyperlipidemia.

INTRODUCTION

Acute pancreatitis is a common condition with various possible etiologies, gallstone and alcohol being the most common. Metabolic, structural and iatrogenic causes account for 20-25% of the cases [1]. Hyperlipidemia in the form of hypertriglyceridemia or chylomicronemia, although less frequent is the well accepted underlying cause of acute pancreatitis in 7% of the cases. The most common after gallstones and alcohol [2]. Typically hypertriglyceridemia induced pancreatitis occur in the patients with a preexisting lipid abnormality along with the presence of secondary precipitating factor (e. g poorly controlled diabetes, alcohol or medication). The triglyceride levels of more than 1000- 2000 mg/dl in patients with type I, III, IV and V hyperlipoproteinemia (Frederickson's classification) is the identifiable risk factor [3]. Genetic factors determine over 60% of the variability in serum lipids [4]. The secondary causes of hypertriglyceridemia have to be ruled out. Most patients can be effectively treated with the existing drug therapy.

MATERIAL AND METHODS

The study was conducted in a private laboratory (Doctors Diagnostic Centre Gandhi Nagar Jammu). A total number of 55 subjects participated in the study out of which 39 were males and 16 were females. All the three parameters i.e. serum triglyceride, serum amylase and serum lipase estimation was performed.

Selection of Patients

The patients were selected based on the following criteria:

- Alcoholic or non-Alcoholic.
- Smoker or Non-Smoker.
- Vegetarian or Non-Vegetarian.

Specimen Collection and preparation for analysis

Blood Collection

Fasting blood samples were collected from the patients coming to the diagnostic centre. 5 ml blood was taken from the patients for the estimation of three biochemical parameters: serum triglycerides, serum amylase and serum lipase. Serum was separated from blood by centrifugation.

METHODOLOGY

Triglycerides

The sample is incubated with lipoprotein lipase enzymatic reaction that converts triglycerides into free glycerol and fatty acid glycerol kinase catalyzes the phosphorylation of glycerol by adenosine 5 triphosphate to glycerol 3 phosphates, glycerol 3 phosphate oxidase oxidizes glycerol 3 phosphate to dishydroxyacetone phosphate and hydrogen peroxide. The catalytic action of peroxidase forms quinoneimine from H_2O_2 aminoanipyrine and 4- chlorophenol. The change in absorbance due to formation of quinoneimine is directly proportional to the total amount of glycerol and its precursors in the sample and is measured using a bichromatic (510, 700 mm) end point technique.

Amylase

A small quantity of serum is incubated at 37^0 C for 7.5 minutes with a solution containing 0.4 amylase of 87 crea**.** The disappearance of blue color that starch gives with iodine solution is the measure of the extent to which the starch has been hydrolyzed to amylase.

Lipase

Lipase is a pancreatic enzyme secreted into the small intestine. It catalyzes the hydrolysis of triglycerides to free fatty acids and glycerol. The liberated free fatty acids at different enzyme concentrations will be titrated with 0.05 N NaOH. Titrate the liberated fatty acids with NaOH noting the time of the titration should not exceed 10 minutes.

RESULTS

Present study was carried out on 55 participants, out of which 39 were males and 16 were females. The participants were grouped in different groups according to the study and gender. (Table no.1) Out of 39 male participants 33 were alcoholics (84.61%) and 6 were non alcoholic (15.38%). Out of 16 females none were alcoholic. Out of 39 males 21 were smokers (53.84%) and 18 were non smokers (46.15%). Out of 16 females 2 were smokers (12.5%) and 14 were non smokers (87.5%).Out of 39 males 29 were non vegetarians (74.35%) and 10 were vegetarians (25.6%). Out of 16 females 10 were non vegetarians (62.5%) and 6 were vegetarians (37.5%). Table no. 2 shows normal and increased levels of serum amylase, serum lipase and serum triglycerides. The mean TG level of participants was elevated among the females, smokers and non-vegetarian patients where as mean levels of serum amylase was more elevated among the females, smokers and non- vegetarian patients. The lipase level of the participants was elevated in females, alcoholic, non-vegetarian and smokers. (Table no. 3)

Table-1: Distribution of Patient in different category--

Category	**Total No. of patients- 55**	**%**
Male	39	70%
Female	16	30%
Alcoholic	33(male-33, female-0)	60%
Non-Alcoholic	22 (male-6, female- 16)	40%
Smoker	23(male-21, female-2)	42%
Non-smoker	32 (male-18, female -14)	58%
Vegetarian	16 (male-10, female-6)	30%
Non- Vegetarian	39 (male-29, female- 10)	70%

Table-2: Normal and increased levels of parameters

Parameters	**No. of patients with normal value**	**No. of patients with higher value**
Serum Amylase	15	40
Serum Lipase	27	28
Serum Triglycerides	17	30

Table-3: Mean Triglycerides, lipase and amylase levels in different category of patients.

Category	No. of Patients	Mean TG (N-50-150 mg/dl)	Mean Amylase N--25-150 IU/L)	Mean lipase (N- 70-200 U/L)
Male	39	369	155.70	273.37
Female	16	**443.24**	**214.83**	**294.46**
Alcoholic	33	369.2	171.61	**299.9**
Non- Alcoholic	22	319.4	177.64	176.57
Smoker	23	**409.3**	**186.1**	**283.2**
Non- Smoker	32	311.5	164.8	**280.5**
Vegetarian	16	363.14	159.28	255.61
Non- Vegetarian	39	**391.7**	**186.494**	**293.71**

DISCUSSION

The clinical presentation of hypertriglyceridemic pancreatitis is similar to other causes of acute pancreatitis, but some evidences suggest that there may be an increased severity and risk of complications. Multiple etiologies of highly elevated triglyceride levels have been implicated, including congenital disorders, metabolic perturbations and certain medications [5].

Chylomicrons are triglycerides rich lipoprotein particles. They are present in circulation when triglyceride are greater than 10 mmol/litre (9000 mg/dl) these are large enough to occlude the pancreatic capillaries, leading to ischemia and subsequent acinar structural alteration, as also a release of pancreatic lipase.

The pro- inflammatory non- esterified free fatty acids generated from the enzymatic degradation of chylomicron- triglycerides may lead to further damage of pancreatic acinar cells and microvasculature. Subsequent amplification of the release of inflammatory mediators and free radicals may ultimately lead to necrosis, edema and inflammation [6, 7].

In our study, all the 55 participants had high levels of serum triglycerides. All of them showed high level of serum amylase and serum lipase. Out of 55 participants, females who comprised 29% of total study group showed highest levels of triglycerides and corresponding highest levels of serum amylase as compared to other study groups.

Similarly smokers having highest TG levels in the group also showed corresponding increased serum amylase levels. A strong positive correlation of triglyceride levels was found with those of serum amylase and serum lipase levels.

Our results were also supported by study performed by Dominguez-Munoz and co-workers who also showed that mild to moderate elevation of serum triglyceride levels are likely to be an epiphenomenon of the pancreatic disease whereas the severe hyperchylomicronemia and hypertriglyceridemia required to trigger acute pancreatitis would require a relevant defect in the lipid catabolism and clearance [8].

Elisaf et al. also concluded in their study that like synthetic estrogens, the tamoxifen- mediated rise in TG's may be either contributory or causative in the development of acute pancreatitis [9].

The clinical presentation and course of hypertriglyceridemic pancreatitis (HTGP) does not differ greatly from the other causes of acute pancreatitis [10]. Lipemic serum frequently associated with an underlying metabolic abnormality or compromising medications, is the single most reliable clue that the pancreatitis is associated with or precipitated by hyperlipidemia [11, 12].

Alcohol itself is an independent cause of pancreatitis. Ethanol compromises fuel and energy metabolism, thereby resulting in decreased serum glucose levels with elevated levels of lipids due to increased production and decreased utilization of energy sources. Alcohol aggravates hypertriglyceridemia and the liberated free fatty acid esters can promote calcium influx which leads to calcium- mediated pancreatic necrosis [13].

Currently there is no clear evidence that hyperlipidemia-induced pancreatitis differs from other types of pancreatitis in terms of frequency of necrosis, complications or outcome [14,15].

Although chylomicrons and triglyceride levels fall rapidly after oral fat intake ceases, efforts to accelerate the removal of precipitating lipoproteins have been considered. Direct removal of chylomicrons in the acute setting can be readily achieved by plasmapheresis and there are numerous documented reports also [16, 17].

Dietary restriction is the cornerstone of therapy. Additional treatment modalities have included insulin and heparin to stimulate the synthesis, release and activation of lipoprotein lipase from capillary endothelial cells to promote triglyceride degradation into free fatty acids for further metabolism or storage [20].

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