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Original Research Article

Serum triglyceride level: a predictor of complications and outcomes in acute pancreatitis?

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ABSTRACT

Background: The aims and objectives were to analyse the influence of elevated triglyceride level in acute pancreatitis so the ability to predict the severity of acute pancreatitis can help identify the patients at increased risk of morbidity and mortality, therefore to make an early decision to triage these patients and treat accordingly.
Methods: An observational study was undertaken over a span of 6 months from January 2018 to June 2018 in K.R Hospital, Mysore, Karnataka, India. 50 Patients admitted with the diagnosis of acute pancreatitis, who had serum triglyceride levels measured within the first 24 hrs of admission, were divided into 2 groups. The study group consisted of patients with a triglyceride level ≥200 mg/dl (group 2) and the control group consisted of triglyceride level of ≤ 200 mg/dl (group 1). We collected the detailed history, data of laboratory values, incidence of complications like pleural effusion, acute kidney injury, pancreatic necrosis, shock, admission to ICU, length of total hospital stay and death.
Results: A total of fifty patients were included in the study out of which 42 patients had triglycerides of less than 200 mg/dl and 8 patients had triglycerides of more than 200 mg/dl. There was no significant difference in the incidence of pleural effusion and shock but there was significant difference in the incidence of acute kidney injury, pancreatic necrosis and Intensive care unit stay between the two groups.
Conclusions: A triglyceride level of more than 200 mg/dl on admission in acute pancreatitisis is an independent predictor of developing local and systemic complications, hospital stay, admission to ICU and death.

Keywords: Acute pancreatitis, Triglycerides, Complications

INTRODUCTION

Acute pancreatitis, an inflammatory disorder of the pancreas, is the most frequent cause of admission to hospital due to gastrointestinal disorders in the USA.[1,2] With an annual incidence ranging from 4.9 to 35 per 100,000 population approximately.

Hypertriglyceridemia is defined as increase in fasting plasma triglyceride levels typically above the 95th percentile for age and sex, although additional quantitative or qualitative lipoprotein abnormalities can also be present.[3,4] Hypertriglyceridemia is one of the known causes of acute pancreatitis, seen in 1.3-3.8% of patients as a primary etiological factor or secondary to other factors prior to the increase of lipid levels but constituting clinically relevant proportion of cases.[3]

Eighty to 90% of the adipocyte volume is composed of TGs, which can be hydrolyzed by lipases that are released during pancreatitis; serum TGs and adipose tissue are hydrolyzed, generating free fatty acids (FFAs).[5,6] Elevated TGs and FFAs are high risk factors that lead to toxic effects and are necessary to evoke damage to isolated pancreatic acinar cells.[7] It has been seen that triglyceridemia would worsen pancreatic injury induced

by acute pancreatitis when it reaches 500 mg/dl or more, thus playing an important role in predisposing mild pancreatitis to the vicious episode.[8]

Severe acute pancreatitis patients with hypertriglyceridemia can have pancreatic necrosis, pseudo cysts, abscesses and other complications with the different clinical course.[9] Many reports have shown that triglyceride elevation on admission for acute pancreatitis is a predictor of a poor prognosis as well as local and systemic complications. The impact of different levels of HTG on the severity and complications of AP has not been clearly defined. In this study, we aimed to analyze the influence of elevated triglyceride level in acute pancreatitis (AP) and its prognosis. The ability to predict the severity of acute pancreatitis can help identify patients at increased risk for morbidity and mortality, therefore helping clinicians to make an early decision to triage these patients to intensive care units as well as selection of patients for specific interventions.

METHODS

Patient selection

Patients ≥18 years of age admitted to our hospital with the diagnosis of acute pancreatitis (AP), who had serum triglyceride levels measured within the first 24 hours of admission from January 2018 to June 2018 in surgical units in K. R Hospital, Mysore, Karnataka, India were included in the study population. The diagnosis of AP was made when any two of the following three criteria were met: classic abdominal pain; elevation of amylase and/or lipase three times the upper limit of normal; and radiographic evidence of acute pancreatitis. The initial study population consisted of 56 patients. Patients with end stage renal disease, those with chronic kidney disease and those with missing information/data were excluded from the study. A total of 50 patients were finally included and divided into two study groups.

Group division

The study group consisted of patients with a triglyceride level ≥2.26 mmol/L (≥200 mg/dl) (group 2) and the control group consisted of triglyceride level of ≤2.26 mmol/L (≤200 mg/dl) (group 1). All the values were measured within the first 24 hours of presentation.

Data collection

Baseline demographic data including age, gender, vital signs on admission, complete blood count, liver function test, serum amylase, serum lipase, and serum triglyceride levels were collected. We calculated the incidence of local and systemic complications including pleural effusion, shock, acute kidney injury, pancreatic necrosis, and admission to ICU, length of hospital stay and death.

Definition and criteria

Complications were defined as follows: shock as a systolic blood pressure of less than 90 mm of Hg, and Pancreatic necrosis is defined as diffuse or focal areas of nonviable pancreatic parenchyma >3 cm in size or >30% of the pancreas. Acute kidney injury (AKI) was defined as increase in the serum creatinine concentration of ≥26.5 μmol/l (≥0.3 mg/dl) from baseline; a percentage increase in the serum creatinine concentration of ≥50 percent; or oliguria of <0.5 ml/kg per hour for more than six hours.

Statistical methods

Statistical analysis was performed with IBM SPSS 20 (Statistical packages for the Social Sciences). For comparison of continuous variables between the two groups, we used the independent sample tests *t*-test.

RESULTS

There were fifty acute pancreatitis patients included in the study, out of which 42 had a triglyceride level of ≤2.26 mmol/L (group 1) and 8 had a triglyceride level of ≥2.26 mmol/L (group 2). Below is the table depicting the sex distribution of the study population. It includes 45 men and 5 women with male to female ratio being (37:5) in group 1 and (8:0) in group 2. As most of the males are abused to alcohol compared to females, which forms the major aetiological factor for the acute pancreatitis, the male to female ratio is very high.

Table 1: Table depicting the sex distribution of the study population.

Sex	Triglyceride ≤200 mg/dl (n=42)	Triglyceride ≥200 mg/dl (n=8)
	N (%)	**N (%)**
Male	37 (88)	8 (100)
Female	5 (12)	0 (0)

This cohort study includes 45 men and 5 women with male to female ratio being (37:5) in group 1 and (8:0) in group 2. As most of the males are abused to alcohol compared to females, which forms the major aetiological factor for the acute pancreatitis, the male to female ratio is very high.

As alcohol pancreatitis, biliary pancreatitis, hypertriglyceridemia and others forms the major etiological factors for acute pancreatitis, in our study there were more cases of acute biliary pancreatitis (52% versus 12%) in group 1 compared to group 2 which was more likely to be due to the presence of more females (12% versus 0%) was consistent with the epidemiological studies that showed gallstones are more common in females and group 2 consisted of more cases of alcoholic pancreatitis (50% versus 23%) compared to group 1 as it consists of more males (100% versus 88%) who are

likely to abuse alcohol. The same has been depicted by the below bar diagram.

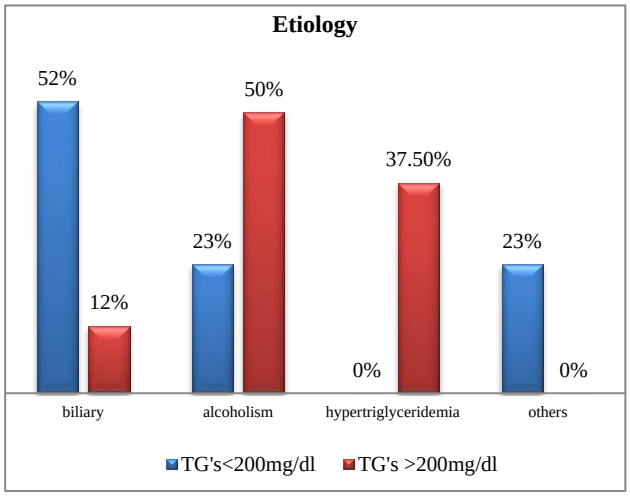

Figure 1: Bar diagram showing the various etiological factors.

There were more cases of acute biliary pancreatitis(52% versus 12%) in group 1 compared to group 2 which was likely due to the presence of more females (12% versus 0%) and group 2 consisted of more cases of alcoholic pancreatitis(50% versus 23%) compared to group 1, as it consists of more males(100% versus 88%) who are likely to abuse alcohol.

The prevalence of diabetes mellitus (3% vs 12%; p<0.001), smoking (21% vs 37%; p<0.001), and alcoholism (16% vs 37%; p<0.001) were more common in the HTG group which may represent these factors as the risk factors for worse outcomes in patients with acute pancreatitis. No significant difference was identified with regard to hypertension (19% vs. 12%; p=0.559). A bardiagram was constructed to study the associated co-morbidities between the two groups and their impact on the outcome and complications.

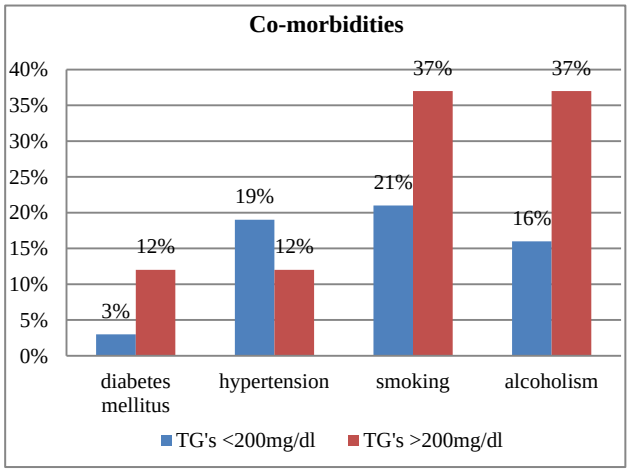

Figure 2: Bar diagram showing the associated co-morbidities between the two groups.

History of diabetes mellitus (3% vs 12%; p<0.001), smoking (21% vs 37%; p<0.001), and alcoholism (16% vs 37%; p<0.001) were more common in the HTG group. No significant difference was identified with regard to hypertension (19% vs. 12%; p=0.559).

To learn the incidence of complications in acute pancreatitis patients related to increased or decreased level of triglycerides, the complications between the two groups were plotted on a bar diagram below. Patients with a triglyceride level ≥200 mg/dl had higher incidence of acute kidney injury (87% versus 35%), pancreatic necrosis (37.5% versus 9.5%) and number of admissions to ICU (87% versus 4.7%) compared to the patients with triglyceride level ≤200 mg/dl. Pancreatic lipase hydrolyzes excess TG in serum resulting in the accumulation of free fatty acids (FFAs), which are toxic to organ function and TG depositing around kidney tubules is hydrolyzed by pancreatic lipase with production of high levels of toxic FFAs around the renal cells, which may directly impair renal function. The levels of pancreatic enzymes are much higher in glomerulus because of concentration and aggravate the damage of renal function.

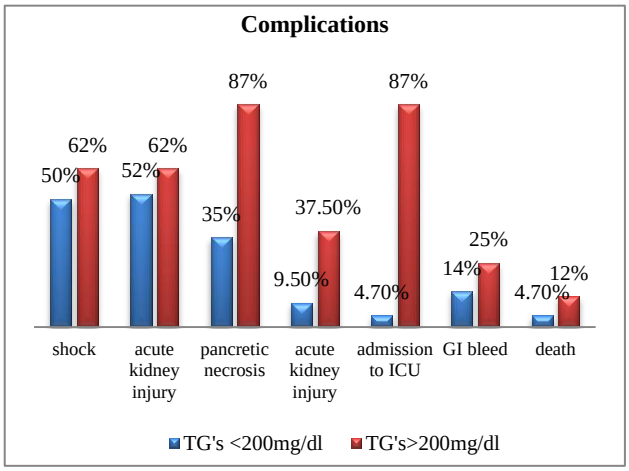

Figure 3: Bar diagram showing the differences in the incidence of complications between two groups.

Patients with a triglyceride level ≥200 mg/dl had higher incidence of acute kidney injury (87% versus 35%), pancreatic necrosis (37.5% versus 9.5%) and number of admissions to ICU (87% versus 4.7%) compared to the patients with triglyceride level ≤200 mg/dl.

DISCUSSION

Many predictive models have been developed to identify patients at increased risk for morbidity and mortality from acute pancreatitis. The ideal predictor would be a single marker that can be reliably and rapidly measured with cost effectiveness while causing no discomfort to the patient. Recent studies have suggested that triglyceride level ≥2.26 mmol/L in patients with acute pancreatitis increases the incidence of complications compared to

normal triglyceride levels.[10] Pedersen et al reported that nonfasting mild-to-moderate HTG levels of 177 mg/dl (2 mmol/L) and above were associated with a high risk of AP in 116,550 individuals.[11] Tariq et al showed that a TG level of ≥2.26 mmol/L on admission of AP patients was an independent predictor of developing local and systemic complications, the hospital length of stay, admission to the ICU, and ICU length of stay.[12]

So in our study, we aimed to study the serum triglyceride level as a marker that can predict the development of complications and the need for admission to ICU among patients admitted with acute pancreatitis. It is generally believed that a serum triglyceride (TG) level of more than 11.3 mmol/L (1000 mg/dl) is needed to precipitate AP, the reduction of which to levels well below 11.3 mmol/L is often preventative.[13]

We observed that in our study, group 1 had more patients with biliary pancreatitis (52% versus 12%), which was likely due to the presence of more females (12% versus 0%) in this group consistent with the epidemiological studies that showed gallstones are more common in females. Group 2 consists of more male patients (100% versus 88%) and more cases of alcoholic pancreatitis (50% versus 23%) were seen likely because males are more likely to abuse alcohol.[14]

Hypertriglyceridemia was found to be an independent risk factor for development of acute kidney injury in patients with AP and development of AKI in acute pancreatitis is associated with a higher mortality.[15] We excluded patients with chronic kidney disease and end stage renal disease in our study population to validate these results. Patients with a triglyceride level >200 mg/dl had higher incidence of acute kidney injury (87% versus 35%), pancreatic necrosis (37.5% versus 9.5%) and number of admissions to ICU (87% versus 4.7%) compared to the patients with triglyceride level <200 mg/dl. Pancreatic lipase hydrolyzes excess TG in serum resulting in the accumulation of free fatty acids (FFAs), which are toxic to organ function and TG depositing around kidney tubules is hydrolyzed by pancreatic lipase with production of high levels of toxic FFAs around the renal cells, which may directly impair renal function. The levels of pancreatic enzymes are much higher in glomerulus because of concentration and aggravate the damage of renal function.

CONCLUSION

In summary, a TG ≥2.26 mmol/L on admission in acute pancreatitis is an independent predictor of developing local and systemic complications (organ failure), hospital length of stay, admission to ICU.

The final conclusion of this study is that patients of acute pancreatitis with elevated triglyceride levels form a morbid group and these should be monitored aggressively for the development of any complications.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Lankisch PG, Apte M, Banks P. A. Acute pancreatitis. The Lancet. 2015;386(9988):85–96.
2. Peery AF, Dellon ES, Lund J, Crockett SD, McGowan CE, Bulsiewicz WJ, et al. Burden of gastrointestinal disease in the United States: 2012 update. Gastroenterology. 2012;143(5):1179–87.e3.
3. Hegele RA. Monogenic dyslipidemias: window on determinants of plasma lipoprotein metabolism Am J Hum Genet. 2001;69:1161.
4. Hodis HN, Mack WJ, Krauss RM, Alaupovic P. Pathophysiology of triglyceride-rich lipoproteins in atherothrombosis: Clinical aspects. Clinical cardiology. 1999 Jun;22(S2):II-15.
5. Zechner R, Kienesberger PC, Haemmerle G, Zimmermann R, Lass A. Adipose triglyceride lipase and the lipolytic catabolism of cellular fat stores. J Lipid Res. 2009;50:3–21.
6. Lam PP, Cosen BL, Lugea A, Pandol SJ, Gaisano HY. Alcohol redirects CCK mediated apical exocytosis to the acinar basolateral membrane in alcoholic pancreatitis. Traffic. 2007;8:605–17.
7. Wang Y, Sternfeld L, Yang F, Rodriguez JA, Ross C, Hayden MR, et al. Enhanced susceptibility to pancreatitis in severe hypertriglyceridaemic lipoprotein lipase-deficient mice and agonist like function of pancreatic lipase in pancreatic cells. Gut. 2009;58:422–30.
8. Oh RC, Lanier JB. Management of hypertriglyceridemia. Am Family Physician. 2007;75(9):1365-71.
9. Balachandra S, Virlos IT, King NK, Siriwardana HP, France MW, Siriwardena AK. Hyperlipidaemia and outcome in acute pancreatitis. Int J Clin Pract. 2006;60(2):156-9.
10. Zeng Y, Zhang W, Lu Y, Huang C, Wang X. Impact of hypertriglyceridemia on the outcome of acute biliary pancreatitis. Am J Med Sci. 2014;348(5):399–402.
11. Pedersen SB, Langsted A, Nordestgaard BG. Nonfasting mild-to-moderate Hypertriglyceridemia and risk of acute pancreatitis. JAMA Intern Med. 2016;176:1834–42.
12. Tariq H, Gaduputi V, Peralta R, Abbas N, Nayudu SK, Thet P, et al. Serum Triglyceride Level: A Predictor of Complications and Outcomes in Acute Pancreatitis? Can J Gastroenterol Hepatol. 2016;2016:8198047.
13. Toskes PP. Hyperlipidemic pancreatitis. Gastroenterol Clin N Am. 1990;19(4):783–91.
14. Hasin DS, Stinson FS, Ogburn E, Grant BF. Prevalence, correlates, disability, and comorbidity of DSMIV alcohol abuse and dependence in the United States: results from the national

epidemiologic survey on alcohol and related conditions. Arch General Psychiatr. 2007;64(7):830–42.
15. Wu C, Ke L, Tong Z, Li B, Zou L, Li W, et al. Hypertriglyceridemia is a risk factor for acute kidney injury in the early phase of acute pancreatitis. Pancreas. 2014;43(8):1312-6.

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