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Acute pancreatitis and its association with dyslipidemia

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ABSTRACT

Background: Pancreatitis is described as any inflammation that manifests in acute or chronic forms in the pancreas. Acute pancreatitis (AP) is a quick attack that results in pancreatic inflammation and affects the peripancreatic tissue and surrounding organ. Aims and Objectives: This study aims to evaluate lipid profile tests in AP patients. Materials and Methods: The present study was conducted in the Department of General Surgery, Government Medical College, Nizamabad, India, prospectively from July 2019 to May 2022. In our study, we enrolled 100 patients between the age of 20 and 60 and divided them into two groups by random selection. We chose 50 consecutive cases of pancreatitis for the study group and 50 other hospitalized patients for the control group. **Results:** Mean age of the case group and control group was found to be 39.17 ± 7.42 and 41.92 ± 9.1 , respectively. We observed that the peak incidence of pancreatitis occurred between the ages of 40 and 70, with a male predominance in all age categories. When compared to control, low-density lipoprotein cholesterol (P=0.715), total cholesterol (P=0.3124), high-density lipoprotein cholesterol (HDL-C) (0.164), non-HDL-C (0.2436), and triglyceride (P=0.1266) of the study group were found to be statistically non-significant. Conclusion: It is determined that serum lipid profile and pancreatitis are frequently linked. To reduce the risk of developing this disease, prevent its underlying complications, and manage the possibility of subsequent recurrences, patients with the aforementioned risk factors should be assessed for any changes in their serum lipid profile. If such an alteration is discovered, they should be treated with antilipidemic medications, dietary modification, lifestyle change, and physical activity.

Key words: Acute pancreatitis; Hypertriglyceridemia; Lipid profile

INTRODUCTION

Acute pancreatitis (AP) is a quick attack that results in pancreatic inflammation and affects the peripancreatic tissue and nearby organs, with an incidence of 12–73/100,000 worldwide.^{1,2} AP is frequently accompanied by excruciating upper abdominal pain. Pain could be really bad and last for several days. Frequent headaches, nausea, vomiting, diarrhea, bloating, and fever are further signs of AP.¹ The mortality rate for AP, a disease with a wide range of etiologies, can reach 20%. In the industrialized world, gallstones and excessive alcohol use are the primary etiologies, with congenital, metabolic, and iatrogenic factors accounting for 20–25% of cases.³

A relatively uncommon cause of pancreatitis is hypertriglyceridemia (HTG). Recent research has demonstrated that a sizable fraction of individuals who had experienced AP has aberrant exogenous triglyceride (TGL) clearance, suggesting that this population already has a metabolic problem. Acute or recurring pancreatitis and very infrequently chronic pancreatitis are the most common manifestations of pancreatitis secondary to HTG.^{3,4} When the levels of HTG are >1000 mg/dL (11.2 mmol/L), pancreatitis is thought to be at risk. Early clinical diagnosis of HTG-associated pancreatitis is crucial for providing the necessary care and preventing additional occurrences.⁵

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Aims and objectives

The aim of this study is to evaluate lipid profile tests in Acute Pancreartitis patients.

MATERIALS AND METHODS

The present study was conducted in the Department of General Surgery, Government Medical College, Nizamabad, India, prospectively from July 2019 to May 2022. In our study, we enrolled 100 patients between the age of 20 and 60 and divided them into two groups by random selection. A random table generated by a computer was used to choose the patients for the study population. We chose 50 consecutive cases of pancreatitis from both sexes and diverse age groups for the study group. Equal number of controls of each age and sex was used. After obtaining ethical approval, all the patients who participated in the study were informed of its aim, and their written agreement was obtained. All patients who presented with the clinical signs and symptoms of pancreatitis were admitted to the hospital and had routine blood tests, including lipase and amylase levels as well as an abdominal X-ray. Serum lipid profiles of those who had signs of pancreatitis were assessed.

Sample collection

A 10 mL of blood was taken from each person using disposable syringes to puncture a vein. The blood was then collected in a gel tube and centrifuged at 5000 rpm for 10 min to separate the different components.

Cases

The study comprised 50 cases with a pancreatitis diagnosis.

Controls

The study also included 50 additional hospitalized patients (individuals admitted for other surgical issues such as hernia, peptic ulcer, intestinal perforation, hydrocele of transvaginal tape, trauma case, and cellulitis and without antilipidemic medications).

Inclusion criteria

The following criteria were included in the study:

- 1. Patients who were newly or already diagnosed with pancreatitis
- 2. Age group >18 years.

Exclusion criteria

The following criteria were excluded from the study:

- 1. Individuals who were not willing to participate in the study
- 2. Patients on antilipidemic drugs
- 3. Patients who were undergoing emergency interventions
- 4. Age <18 years.

Statistical analysis

The SPSS statistical software, version 20.0, was used for statistical analysis. Independent samples Student's t-test and Pearson's correlation analysis for assessment of mean differences between the patients and control groups were performed with P < 0.05 as significant.

RESULTS

Mean age of the case group and control group was found to be 39.17 ± 7.42 and 41.92 ± 9.1 , respectively. Out of 100 patients, 65 were male and 35 were female. In this study, out of 50 patients in the case group, 25 were smokers, 20 consumed alcohol, and 17 had hypertension and 10 were smokers, eight consumed alcohol, and 10 had hypertension out of the 50 patients in the control group (Table 1).

Mean T-C level of the case group was 157.42 mg/dL, with a standard deviation (SD) of 32.14. 211.56 mg/dL was determined to be the mean serum TGL in cases, with a SD of 32.57. The mean high-density lipoprotein cholesterol (HDL-C) value was 38.50 mg/dL, with a SD of 4.761. The mean serum level of non-HDL-C was 128.41 mg/dL with a SD of 31.25 and the mean serum level of low-density lipoprotein cholesterol (LDL-C) was determined to be 101.84 mg/dL with a SD of 29.52. The mean serum levels of T-C, TGL, HDL, LDL, and non-HDL-C in the control group were found to be 166.10 mg/dL, 135.49 mg/dL, 33.82 mg/dL, 99.56 mg/dL, and 116.16 mg/dL, respectively, with SDs of 23.46, 27.58, 3.42, 27.89, and 25.89. When compared to control, LDL-C (P=0.715), total cholesterol (P=0.3124), HDL-C (0.164), non-HDL-C (0.2436), and TGL (P=0.1266) of the study group were found to be statistically non-significant (Table 2).

DISCUSSION

Up to 7% of all instances of AP have severe HTG as the underlying cause, including chylomicronemia.^{6,7} Persistent high serum TGL level for weeks may be a sign of underlying lipid problems.^{8,9}

There is a theory that when pancreatic lipase hydrolyzes TGL, the ensuing free fatty acids cause inflammatory

Table 1: Demographic data				
Demographic data	Case (n=50)	Control (n=50)		
Age Sex	39.17±7.42	41.92±9.1		
Male	36	29		
Female	14	21		
Smoking	25	10		
Alcohol consumption	20	08		
Hypertension	17	10		

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Table 2: Statistical analysis of lipid parameters				
Lipid parameters	Case group	Control group	P-value	
Total cholesterol (mg/dl)	157.42±32.14	166.10±23.46	0.3124	
Triglyceride (mg/dl) (TGL)	211.56±32.57	135.49±27.58	0.0658	
HDL-C (mg/dl)	38.50±4.761	33.82±3.42	0.164	
LDL-C (mg/dl)	101.84±29.52	99.56±27.89	0.715	
Non-HDL-C (mg/dl)	128.41±31.25	116.16±25.89	0.2436	
HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol				

alterations and free radical damage, which support severe pancreatitis. TGL-rich lipoproteins are called chylomicrons. The acinar structure that exposes TGLrich molecules to the pancreatic lipase may be disturbed by inadequate fat flow in the pancreatic capillaries, which can cause ischemia.³

By degrading chylomicrons-HTG enzymatically, proinflammatory non-esterified free fatty acids are produced, which could cause more harm to pancreatic acinar cells and microvasculature.^{10,11} Necrosis, edema, and pancreatitis eventually result from the subsequent amplification of the released inflammatory mediators and free radicals. AP in various experimental models has also been found to be aggravated by HTG.¹² It is not quite apparent how the HTG induces the AP; the precise process. The majority of accepted ideas describe how pancreatic lipase breaks down excess TGs to liberate fatty acids that cause ischemia and harm to pancreatic cells.^{13,14}

The findings of studies done by Teshima et al.,¹⁵ and Yadav et al.,¹⁶ are similar to that of this study. This study found that the peak incidence of pancreatitis occurred between the ages of 40 and 70, with a male predominance in all age categories.

In this study, it was found that the case group's lipid profile had changed when compared to the control group. In the case group, the mean serum levels of T-C, TGL, HDL-C, LDL-C, and non-HDL-C were noted. The case group has greater values for each parameter than the control group. Compared to other causes, AP is more likely to be linked to HTG, but no difference in mortality has been noted.^{17,18}

A clinical investigation by Michalakis et al.,¹⁹ concluded that pancreatitis is associated with a considerable drop in blood HDL-C, and the astounding rise of TGL and cholesterol found in the lipid profile validates our observation.

Limitations of the study

The small sample size could be considered a limitation to this study.

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CONCLUSION

Pancreatitis affects men more frequently than women. The TC, TG, LDL, and very LDL lipid profile parameters are greater in pancreatitis patients than in the control group, but serum HDL-C is lower. Finally, it is determined that serum lipid profile and pancreatitis are frequently linked. To reduce the risk of developing this disease, prevent its underlying complications, and manage the possibility of subsequent recurrences, patients with the aforementioned risk factors should be assessed for any changes in their serum lipid profile. If such an alteration is discovered, they should be treated with antilipidemic medications, dietary modification, lifestyle change, and physical activity.

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GR- Concept and design of study, review of literature, acquisition of data, and original draft preparation; RC- Preparation of manuscript, review and editing, and revision of final manuscript; and SKA- Review of literature, statistical analysis, and interpretation of results.

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