International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 13, No. 12, December 2024 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.12.2024.142

ORIGINAL RESEARCH

A study on biochemical parameters and presentation of patients diagnosed with normal and complicated pseudocyst of pancreas

[1]Dr. S.Latha, [2]Dr. A.Waheetha Begum, [3]Dr. M.P.Saravanan

[1]Assistant Professor, Department of Biochemistry, Government Thiruvallur Medical College, Thiruvallur, India
[2]Assistant Professor, Department of Biochemistry, Chengalpattu Medical College, India
[3]Professor & HOD, Department of Biochemistry, Stanley Medical College, Chennai, India

Corresponding Author
Dr. S.Latha
Assistant Professor, Department of Biochemistry, Government Thiruvallur Medical College, Thiruvallur, India

Received Date: 16 November, 2024 Accepted Date: 20 December, 2024

ABSTRACT

Background: Pseudocysts are either directly or indirectly related to the pancreatic duct system through the pancreatic parenchyma. The usefulness of serum testing is limited. Although they may be within reference ranges, amylase and lipase levels are frequently high. Certain laboratory tests may offer hints about the underlying cause of pancreatitis. The current study's objective was to examine the biochemical makeup of pseudocysts that developed during acute and chronic pancreatitis, with a particular emphasis on proteolytic activity, and to link the biochemical results with each patient's clinical trajectory. **Methodology:** This study was done in Government Stanley Medical College for a period of 12 months as a ccomparative cross-sectional study in 29 patients. Pseudocyst Pancreas of patients with age 18 to 60 admitted in Surgical Gastroenterology department was included in this study.5 ml of venous blood was collected in plain tube under aseptic precautions. Sample allowed to clot by waiting for 20 minutes. After that Blood and cystic fluid samples was centrifuged at 2500rpm for 15 minutes. Serum was separated and subjected to biochemical analysis and cystic fluid supernatant are stored separately at -20^{o}C for analysis. **Results:** The mean Serum Amylase among the subjects was 576.07(± 818.14. The mean Serum Lipase among the subjects was 444.17 (± 440.78). The mean Total bilurubin among the subjects was 1.28 (± 0.78). The mean Direct bilirubin among the subjects was 0.63 (± 0.53). The mean Serum protein among the subjects was 6.34 (± 0.77). The mean Serum albumin among the subjects was 3 (± 0.58. The mean Serum Urea among the subjects was 34.93 (± 27.88). The mean Serum creatinine among the subjects was 0.86 (± 0.39). **Conclusion:** The idea behind the present study began to reduce morbidity and mortality in cases with vascular complications both in acute and chronic pancreatitis with pseudo cyst. Patients diagnosed as pseudocyst by ultrasound or CT scan, should be investigated along with routine parameters , which will give us a clue ,which patients will go for vascular complications and who will recover normally with conservative management, and frequently watch observe and monitor the susceptible patients going for complications in a tertiary care hospital, so that mortality and morbidity can be prevented.

Keywords: Pseudocyst of pancreas, biochemical parameters.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

Rich in amylase and other pancreatic enzymes, the pseudocyst of the pancreas is a confined fluid collection encased in a fibrous tissue wall devoid of epithelium [1]. Pseudocysts are either directly or indirectly related to the pancreatic duct system through the pancreatic parenchyma. They are brought on by pancreatic ductal disruption after elevated pancreatic ductal pressure, which can be brought on by pancreatic necrosis after an acute pancreatitis attack or by stenosis, calculi, or protein plugs blocking the primary pancreatic ductal system [2,3]. Thirty to forty percent of patients with chronic pancreatitis experience complications from pseudocysts, a common clinical issue [4].

Although pseudocyst formation is less common after acute pancreatitis than chronic pancreatitis and more common after alcohol-induced pancreatitis than after non-alcohol-related pancreatitis, the occurrence of pseudocysts is similar to that of pancreatitis, and their etiology is similar to that of pancreatitis. According to research from nations with significant alcohol consumption, alcohol-related pancreatitis appears to be the primary etiology, accounting for 59%–78% of

©2024Int. J. LifeSci.Biotechnol.Pharma.Res.

DOI: 10.69605/ijlbpr_13.12.2024.142

all pseudocysts [5].Although there was no concrete proof, the majority of the individuals in the idiopathic category were assumed to have alcohol-related conditions [6].

The incidence of pseudocyst is minimal, ranging from 1.6% to 4.5%, or 0.5-1 per 100,000 persons annually, regardless of its cause [9, 10]. Pseudocysts formed in 86 participants in an Imrie trial following emergency hospitalization for an acute pancreatitis episode [11]. Of the 86 pseudocysts resulting from acute pancreatitis, sixty-two were obtained from the local hospital population, which included 879 patients who were hospitalized for acute pancreatitis over the same time period. Pseudocysts as a consequence of acute pancreatitis occurred in 7% of cases overall [11].

Unlike acute pseudocysts, which can occur in patients who have had chronic pancreatitis for 10, 20, or more years, there is a lack of accurate data based on the long-term follow-up of patients with chronic pancreatitis. This means that patients with chronic pancreatitis are at a higher risk of developing a pseudocyst at least once during a prolonged period of illness [14].A pancreatic pseudocyst may present clinically as an asymptomatic patient or as a significant abdominal disaster as a result of complications [16–18]. Bleeding (typically from a pseudoaneurysm in the splenic artery), infection, and rupture are examples of acute consequences.

The usefulness of serum testing is limited. Although they may be within reference ranges, amylase and lipase levels are frequently high. If the bile duct is blocked due to a stone, extrinsic compression from the pseudocyst, or an underlying liver disease (such as alcoholic hepatitis), the blood bilirubin and liver chemistries may be high. Certain laboratory tests (such as high triglycerides or calcium levels) may offer hints about the underlying cause of pancreatitis. Biliary pancreatitis is suspected when liver chemistries are elevated. The current study's objective was to examine the biochemical makeup of pseudocysts that developed during acute and chronic pancreatitis, with a particular emphasis on proteolytic activity, and to link the biochemical results with each patient's clinical trajectory.

METHODOLOGY

This study was done in Government Stanley Medical College – involving the followingdepartments - department of Biochemistry,Institute of Surgical Gastroenterology And Liver Transplantation for a period of 12 months as a ccomparative cross-sectional study in 29 patients. Pseudocyst Pancreas of patients with age 18 to 60 admitted in Surgical Gastroenterology department was included in this study.

This study was approved by Institutional Ethics Committee. The samples were collected after obtaining a clearly written informed consent from patients and their attenders admitted in Department of Gastroenterology. Patients with acute and chronic pancreatitis going for complications of Pseudocyst formation diagnosed with clinical examination, diagnostic procedures like USG abdomen, CT abdomen.Admitted patients shall be of both sexes and above 18 years of age group were included in this study. Whereas patients with congenital cysts of pancreas, patients with neoplastic swelling, Hydatid cyst of pancreas and all the true cysts of pancreas were excluded.

All the participants or their attenders was informed about the study andinformed consent was obtained from them or from their attenders, if the patients is not able to sign. A brief history about their personal details such as life style, physical activity, food habits, their socioeconomic status, history of any illness was obtained. General examination and Systemic examination wasdone.

After getting the informed consent from the patient or the attender, 5 ml of venous bloodwas collected in plain tube under aseptic precautions. Sample allowed to clot by waiting for 20 minutes. After that Blood and cystic fluid samples was centrifuged at 2500rpm for 15 minutes. Serum was separated and subjected to biochemical analysis and cystic fluid supernatant are stored separately at -20^{o}C for analysis.

The collected data were analyzed with IBM.SPSS statistics software 23.0 Version.To describe about the data; descriptive statistics frequency analysis, percentageanalysiswereusedforcategoricalvariablesan dthemean&S.D were used for continuous variables.To find the significant difference between the bivariate samples in Independent groups; the Unpaired sample t-test wasused.In all the above statistical tools the probability value .05 is considered as significant level.

RESULTS

Among the 50 subjects in our study population, 11 (37.93%) was in 31 - 40 years followed by 9 (31.03%) in 21 - 30 years and 1 (3.45%) was < 20 years. Also 28 (96.55%) were Male and 1 (3.45%) were Female.Among the subjects, 25 (86.21%) had H/o alcohol intake.

Among the subjects, 25 (86.21%) had Alcohol induced pancreatitis followed by 1 (3.45%) had Idiopathic pancreatitis,1 (3.45%) had Hypertriglyceridemiaand 1 (3.45%) had Trauma. Among the subjects, 19 (65.52%) had Nil comorbidities followed by 2 (6.9%) had COPD and least 1 (3.45%) had Aortic aneurysm.

Table 1: Cause of Pancreatitis distribution.

Cause of Pancreatitis	Frequency	Percent
Alcohol induced	25	86.21
Hypertriglyceredemia	1	3.45

©2024Int. J. LifeSci.Biotechnol.Pharma.Res.

DOI: 10.69605/ijlbpr_13.12.2024.142

Idiopathic	1	3.45
Trauma	1	3.45
Total	29	100.00

Among the subjects, 19 (65.52%) had High Serum Amylase and 10 (34.48%) had Normal Serum Amylase. Among the subjects, 19 (65.52%) had High Serum Lipase and 10 (34.48%) had Normal Serum Lipase

Among the subjects, 15 (51.72%) had Normal Total bilirubin and 14 (48.28%) had High Total bilirubin. 20 (68.97%) had High Direct bilirubin and 9 (31.03%) had Normal Direct bilirubin.21 (72.41%) had Normal Serum protein and 8 (27.59%) had Low Serum protein. 23 (79.31%) had Low Serum albumin and 6 (20.69%) had Normal Serum albumin

Among the subjects, 16 (55.17%) had Normal Serum Urea and 13 (44.83%) had High Serum Urea24 (82.76%) had Normal Serum creatinine and 5 (17.24%) had High Serum creatinine

The mean Serum Amylase among the subjects was 576.07 (± 818.14) ranging from 24 to 3411. The mean Serum Lipase among the subjects was 444.17 (± 440.78) ranging from 34 to 1823. The mean Total bilurubin among the subjects was 1.28 (± 0.78) ranging from 0.28 to 3.59. The mean Direct bilirubin among the subjects was 0.63 (± 0.53) ranging from 0.1 to 2.5. The mean Serum protein among the subjects was 6.34 (± 0.77) ranging from 4.6 to 7.8. The mean Serum albumin among the subjects was 3 (± 0.58) ranging from 1.7 to 4.1. The mean Serum Urea among the subjects was 34.93 (± 27.88) ranging from 9.2 to 110. The mean Serum creatinine among the subjects was 0.86 (± 0.39) ranging from 0.4 to 3.

Table 2: Blood parameters

	Mean	Std. Deviation	Minimum	Maximum
Serum Amylase	576.07	818.14	24.0	3411.0
Serum Lipase	444.17	440.78	34.0	1823.0
Total bilirubin	1.28	0.78	0.3	3.6
Direct bilirubin	0.63	0.53	0.1	2.5
Serum protein	6.34	0.77	4.6	7.8
Serum albumin	3.00	0.58	1.7	4.1
Serum Urea	34.93	27.88	9.2	110.0
Serum creatinine	0.86	0.39	0.4	2.0

DISCUSSION

In the present study group of 29 patients, of whom 22 patients had vascular complications is pseudocyst or in acute necrotizing pancreatitis and 7 patients with normal uncomplicated pseudocyst who were treated at our institute over a period of 26 months. Even though this study was first of its kind to ensure the trypsin activity in pseudocyst for predicting the vascular complications in these patients who actually needs early intervention than to wait and watch phenomenon in pseudo cysts. similar study to measure proteolytic activity was done in 1993 by Ake Lasson etal[19] in 15 cases of pseudo cysts were biochemically analyzed ,measured over all proteolytic activity in acute vs chronic pseudocyst .

Trypsin, protrypsinogen, pancreatic elastase, amylase, lipase have been identified in fluid of pseudocysts or in fluid with acute necrotizing pancreatitis and walled off pancreatic necrosis (WOPN). Raised levels of routinely measured amylase or lipase both in serum or cystic fluid couldnot predict any complications or morbid clinical course after acute or chronic pancreatitis and its complications by S.amylase and S.lipase at the time of presentation. These parameters were evaluated at the admission. As the most established fact that elevation exceeding three times the normal upperlimit of serum amylase or lipase supports the diagnosis. The upper limit of normal serum amylase in our institute was 90 IU.So values of serum amylase above 270 IU wereuseful in making the diagnosis of pancreatitis.

Most of the cases in our present study are complicated cases of acute or acute on chronic pancreatitis who were admitted elsewhere at the initial onset of pain or hematemesis or melena and was later referred to our institute. The mean value of serum amylase in the present study group was 576.07 did not signify any clinical course or predicted the complications. Etiology did not correlate to the biochemical findings as 26/29 cases were alcoholic and the other three cases of which two are in complicated group with hypertriglyceridemia in a 15 year old male and trauma in 35 year old male , one case in control group, the cause of pseudocyst was idiopathic.Thus, bleeding within a pseudocyst, the development of pseudoaneurysms may very well be explained by proteolytic digestion of the vascular tissue by activated trypsin activity in contact with the pseudocyst or in the necrotic fluid, pseudocysts are accompanied by a high complication rate, 30-40%, and a mortality rate of 3-10% in earlier series.

Cases with preexisting comorbid conditions was identified in about 10 out of 29 patients , which constitutes to 34% of the present study group did not influence on the clinical course during the hospital stay neither mortality. Radiological confirmation of Pseudoanuersym of vessels surrounding the pancreas was identified in 8/22 cases which constitute 36% of

©2024Int. J. LifeSci.Biotechnol.Pharma.Res.

DOI: 10.69605/ijlbpr_13.12.2024.142

the present study.

Even though all these cases were presented in emergency or semi emergency timely intervention with interventional radiological management with coil embolization and early surgical intervention have prevented most of the mortality in the present study group. Lack of availability of sophisticated services, delay in diagnosis, acute blood loss, chronic malnutrition and sepsis would be the most common causes of mortality in these cases.

CONCLUSION

The idea behind the present study began to reduce morbidity and mortality in cases with vascular complications both in acute and chronic pancreatitis with pseudo cyst. Ultrasonography was the most useful investigation for diagnosis and follow-up.CT-scan was required in selected cases. Patients diagnosed as pseudocyst by ultrasound or CT scan, should be investigated along with routine parameters , which will give us a clue ,which patients will go for vascular complications and who will recover normally with conservative management, and frequently watch observe and monitor the susceptible patients going for complications in a tertiary care hospital, so that mortality and morbidity can be prevented.

REFERENCES

1. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg. 1993; 128:586–590.
2. Sanfey H, Aguilar M, Jones RS. Pseudocysts of the pancreas, a review of 97 cases. Am Surg. 1994; 60:661–668.
3. Gumaste VV, Pitchumoni CS. Pancreatic pseudocyst. Gastroenterologist. 1996; 4:33–43.
4. Boerma D, Obertop H, Gouma DJ. Pancreatic pseudocysts in chronic pancreatitis. Surgical or interventional drainage? Ann Ital Chir. 2000;71:43–50.
5. Pitchumoni CS, Agarwal N. Pancreatic pseudocysts. When and how should drainage be performed? Gastroenterol Clin North Am. 1999;28:615–639.
6. Walt AJ, Bouwman DL, Weaver DW, Sachs RJ. The impact of technology on the management of pancreatic pseudocyst. Fifth annual Samuel Jason Mixter Lecture. Arch Surg. 1990;125:759–763.
7. D'Egidio A, Schein M. Pancreatic pseudocysts: a proposed classification and its management implications. Br J Surg. 1991;78:981–984.
8. Nealon WH, Walser E. Main pancreatic ductal anatomy can direct choice of modality for treating pancreatic pseudocysts (surgery versus percutaneous drainage) Ann Surg. 2002;235:751–758.
9. Sandy JT, Taylor RH, Christensen RM, Scudamore C, Leckie P. Pancreatic pseudocyst. Changing concepts in management. Am J Surg. 1981;141:574–576.
10. Wade JW. Twenty-five year experience with pancreatic pseudocysts. Are we making progress? Am J Surg. 1985;149:705–708.
11. Imrie CW, Buist LJ, Shearer MG. Importance of cause in the outcome of pancreatic pseudocysts. Am J Surg. 1988;156:159–162.
12. Maringhini A, Uomo G, Patti R, Rabitti P, Termini A, Cavallera A, Dardanoni G, Manes G, Ciambra M, Laccetti M, et al. Pseudocysts in acute nonalcoholic pancreatitis: incidence and natural history. Dig Dis Sci. 1999;44:1669–1673.
13. Kourtesis G, Wilson SE, Williams RA. The clinical significance of fluid collections in acute pancreatitis. Am Surg. 1990;56:796–799.
14. Ammann RW, Akovbiantz A, Largiader F, Schueler G. Course and outcome of chronic pancreatitis. Longitudinal study of a mixed medical-surgical series of 245 patients. Gastroenterology. 1984;86:820–828.
15. Grace PA, Williamson RC. Modern management of pancreatic pseudocysts. Br J Surg. 1993;80:573–581.
16. Zdanyte E, Strupas K, Bubnys A, Stratilatovas E. [Difficulties of differential diagnosis of pancreatic pseudocysts and cystic neoplasms] Medicina (Kaunas) 2004;40:1180–1188.
17. O'Malley VP, Cannon JP, Postier RG. Pancreatic pseudocysts: cause, therapy, and results. Am J Surg. 1985;150:680–682.
18. Gouyon P, Levy P, Ruszniewski P, Zins M, Hammel P, Vilgrain V, Sauvanet A, Belghiti J, Bernades P. Predictive factors in the outcome of pseudocysts complicating alcoholic chronic pancreatitis. Gut. 1997;41:821–825.
19. Kruse, Peter & Lasson, Åke & Hage, Esther. (1999). Proteases and Protease Inhibitors in Cerulein-Induced Acute Pancreatitis in Rats. The Journal of surgical research. 85. 294-300.

©2024Int. J. LifeSci.Biotechnol.Pharma.Res.