VOLUME - 12, ISSUE - 08, AUGUST - 2023 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Medical Sciences

ASSESSMENT OF EXTRA PANCREATIC MANIFESTATIONS OF ACUTE, SUBACUTE AND CHRONIC CALCIFIC PANCREATITIS

Dr. Suresh Reddy	Associate Professor, Kempegowda Institute of Medical Sciences, Bengaluru
Dr. Sanjay S C	Professor & HOD, Kempegowda Institute of Medical Sciences, Bengaluru
Dr. Shivam Bansal	Postgraduate, Kempegowda Institute of Medical Sciences, Bengaluru
Dr. Srinivas S	Delta Diagnostic Services, Bangalore, Kempegowda Institute of Medical Sciences, Bengaluru

ABSTRACT

Introduction: Pancreatitis accounts for 6-10 per cent of all cases of abdominal pain among patients admitted to hospitals in India. Pancreatitis may sometimes involve multiple organ systems and place demands on the surgeon beyond his or her skills. Patients with systemic complications should be managed by a multidisciplinary team that includes intensive care specialists. It is important to determine the presence and degree of association of these intra-abdominal conditions with acute/subacute/chronic calcific pancreatitis. **Aim &objective:** To analyze the various systemic manifestations of pancreatitis, including both gastrointestinal and extra-intestinal features. **Methods And Materials:** It is a prospective and retrospective observational clinical study in patients who are more than 18 years of age, have typical symptoms like epigastric pain, nausea, vomiting with serum amylase and/or lipase levels elevated 3-4 times above normal and proven imaging diagnosis of acute/subacute/chronic calcific pancreatitis. Contrast Enhanced Computed Tomography (CECT) imaging protocols for abdomen and pelvis with GE 16 Signa Channel were followed. **Results:-** Patients with pancreatitis can have a wide range of other features like ascites, pleural effusion, liver features like fatty hepatomegaly , portal hypertension, chronic liver parenchymal changes, hepatic infarct, gall bladder or biliary tract pathologies like calculous or acalculous cholecystitis, choledocholithiasis, cholangitis, renal changes like pyelonephritis, calculi, cysts, vascular changes like splenic/superior mesenteric/portal vein thrombosis, splenic pathologies like splenomegaly, splenic infarct, lung involvement etc. **Conclusion:** Pancreatitis is one of the leading causes of abdominal pain as well as hospital admission due to gastrointestinal disease and thus amounts to a significant health burden. It can have widespread systemic (both visceral and vascular) associations. These extra-pancreatic features may have important implications for the treating surgeon as they may be able to anticipate the possibility and frequency of these associated manifestations and treat with a more wholesome approach, thus reducing significant morbidity and mortality.

KEYWORDS :

INTRODUCTION

For centuries the pancreas was a *"terra incognita"* hidden behind the stomach and its pathophysiological role remained in the dark. Only in 1761 Jean-Baptista Morgagni described in his book *"de sedibus et causis morborum"* the first case of chronic pancreatitis and it took 60 more years until Kuntzmann was able to connect fatty stool to diseases of the organ.(1) Acute and chronic pancreatitis are distinguished from each other on the basis of structural and functional criteria. In acute pancreatitis, the gland is normal before the attack and can return to normal after resolution of the attack, whereas in chronic pancreatitis, the gland is abnormal before or after the attack, or both (2,3) This classification scheme does not depend on how rapidly symptoms appear or resolve, or on the severity of the symptoms. Thus, it may be impossible to distinguish an exacerbation of chronic pancreatitis from an attack of acute pancreatitis on clinical grounds alone. The hypersecretion of protein from acinar cells in the absence of increased fluid or bicarbonate secretion from duct cells is characteristic of chronic pancreatitis. (4) Plugs formed by the precipitation of protein within interlobular and intralobular ducts are an early finding. (5)

Their importance is evidenced by the observation that endoscopic removal of the plugs may result in transient improvement in the pancreatitis. (6,7) Ductal plugs are initially composed of degenerating cells within a reticular network. They subsequently enlarge to form laminar aggregates through the acquisition of amorphous material. The plugs contain multiple proteins, including secreted digestive enzymes, glycoproteins, and acidic mucopolysaccharides. (8,9) The precipitation of calcium carbonate in the plugs results in the formation of intraductal stones. Ductal plugs can occur in all forms of chronic pancreatitis,(10) but stones are most commonly found in patients with either alcohol induced or tropical (nutritional) pancreatitis.

AIMS & OBJECTIVE

The study is aimed at assessment and analysis of various extra-pancreatic intra-abdominal associations of acute/subacute/chronic calcific pancreatitis.

MATERIALS AND METHODS

This prospective and retrospective observational study was conducted in the Department of Radio-diagnosis at KIMS, BENGALURU with CECT abdomen and pelvis including 60 patients with known diagnosis of acute/subacute/chronic pancreatitis. All 60 patients underwent Contrast Enhanced Computed Tomography imaging (with Pancreatitis protocol) using GE Signa 16 slice CT scanner

The revised Atlanta classification was used for evaluating the severity in cases of acute pancreatitis.

The presence of six intra-abdominal pathologies was analysed in all cases. This included :-

1. LIVER INVOLVEMENT (CLPD,Fatty liver,Hepatomegaly)
2. GB INVOLVEMENT (Cholecystitis,Cholelithiasis)
3. ASCITES
4. SPLEEN INVOLVEMENT (Splenomegaly)
5. VASCULAR THROMBOSIS (Splenic,Portal,Superior Mesenteric Veins)
6. RENAL INVOLVEMENT (Calculus,Pyelonephritis)

RESULTS

In our study the incidence of pancreatitis was 3 times higher in males (table 1 and figure 1) and 30-50 year age group had twice more number of cases as compared to the next most frequent age group(table 2 and figure 2). Also, ascites (58 % cases) and fatty liver (45 % cases) have been found to be

present in highest frequency in patients of pancreatitis (as compared to other lesions).(table 3 and figure 3) Ascites, fatty liver and renal lesions show male predilection when seen in absolute numbers as compared to other conditions (splenomegaly, venous thrombosis)(figure 4-10) which are almost equally distributed between the sexes. Gall bladder lesions like cholecystitis and cholelithiasis were found to be more common in females. (table 5)

DISCUSSION

As we found in our study that incidence of pancreatitis was 3 times higher in males, similarly Yadav et al (11) also stated that incidence was higher for males compared with females in his study. For the 89 clinical cases in his study, the median age at diagnosis was 58 years (interquartile range 48 – 67; range 7 – 87) and 50 / 89 (56.2 %) were males which is very similar to our study. Though Lin et al (12) stated that idiopathic chronic pancreatitis has increased in both males and females. Ascites,pleural and pericardial effusions can be observed during acute pancreatitis. And we also found ascites (58 % cases) to be present in highest frequency in patients of pancreatitis. Similarly Maringhini et al (13) found that ascites,pleural and pericardial effusions are frequent during acute pancreatitis. Pleural effusion and ascites are accurate predictors of severity in these patients. Bush et al (14) though, stated in his study that clinically significant ascites in acute pancreatitis (AP) is rarely encountered and is a result of multifactorial pathogenesis. Early reactionary ascites in AP usually does not require any treatment and resolves spontaneously in majority of patients. A diagnostic analysis should be performed in case of ascites developing in the later stages with increasing pain or worsening organ failure. Ohara et al (15) found 7 cases of AIP with associated systemic extrapancreatic lesions ,the clinical manifestations of the extrapancreatic lesions occurred synchronously with those of AIP. Diagnoses were consequently made simultaneously, and all patients responded satisfactorily to steroid medication.

CONCLUSION

Pancreatitis can have widespread association with other intra-abdominal pathologies. It is particularly important to look for the presence of liver disease like fatty liver , hepatomegaly , CLPD as well as ascites in acute/subacute /chronic pancreatitis cases as they may be found to be co-existing in a significant number of cases. Pancreatic ascites is a well-known entity that results from pancreatic duct injury and subsequent disruption, leading to persistent secretions into the peritoneal cavity. By evaluation of the frequency of ascites, pleural effusion, cholecystitis, liver disease etc, we can draw conclusions regarding what other features should be looked for, in diagnosed cases of pancreatitis. These findings may have important implications for the treating surgeon as they may be able to anticipate the possibility and frequency of these associated manifestations and treat with a more wholesome approach.

Table 1

AGE GROUP	COUNT	COLUMN %
LESS THAN 30 YEARS	17	28.3 %
30 TO 50 YEARS	34	56.7 %
MORE THAN 50 YEARS	9	15.0 %

Table 2

GENDER	COUNT	COLUMN %
FEMALE	15	25 %
MALE	45	75 %

Table 3

Count		Column N %
FATTY LIVER/HEPATOMEGALY		
	27	45.0 %
CHOLECYSTITIS/CHOLELITHIASIS		
	10	16.7 %
ASCITES		
	35	58.3%
SPLENOMEGALY		
	9	15.0%
VENOUS THROMOSIS (PORTAL/SPLENIC/SMV)	5	8.3%
RENAL CALCULUS/PYELONEPHRITIS	17	28.3%

Table 4

	Age_Group					
	Less than 30 years		Between 31 to 50 Years		More than 50 years	
	Count	Column N %	Count	Column N %	Count	Column N %
Cholecystitis / G.B. Lesion	1	5.9%	7	20.6%	2	22.2%
Fatty Liver/Hepatomegaly	6	35.3%	19	55.9%	2	22.2%
Ascites	7	41.2%	24	70.6%	4	44.4%
Splenomegaly	2	11.8%	7	20.6%	0	0.0%
Vascular Thrombosis	0	0.0%	3	8.8%	2	22.2%
Renal Calculus/ Pyelonephritis	2	11.8%	14	41.2%	1	11.1%

Table 5

	Age_Group					
	Less than 30 years		Between 31 to 50 Years		More than 50 years	
	Count	Column N %	Count	Column N %	Count	Column N %
Cholecystitis/ G.B. Lesion	1	5.9%	7	20.6%	2	22.2%
Fatty Liver/Hepatomegaly	6	35.3%	19	55.9%	2	22.2%
Ascites	7	41.2%	24	70.6%	4	44.4%
Splenomegaly	2	11.8%	7	20.6%	0	0.0%
Vascular Thrombosis	0	0.0%	3	8.8%	2	22.2%
Renal Calculus/ Pyelone-phritis	2	11.8%	14	41.2%	1	11.1%

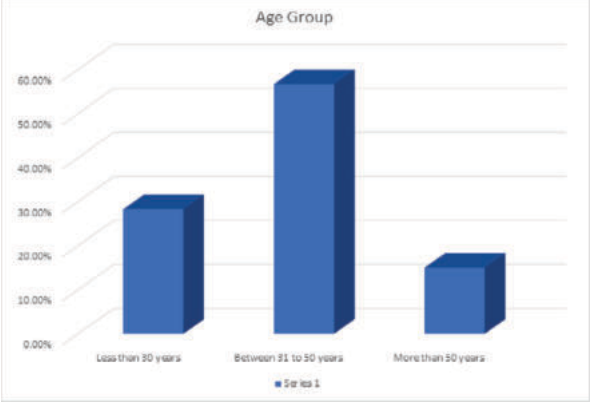

Figure 1

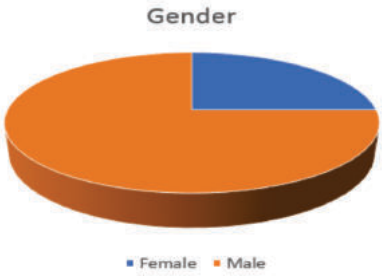

Figure 2

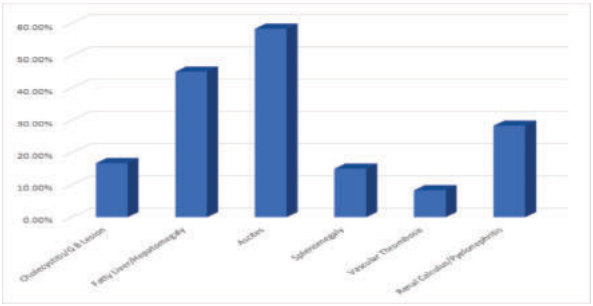

Figure 3

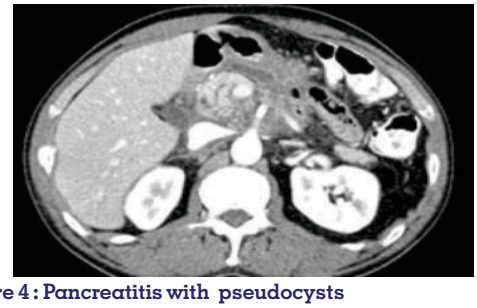

Figure 4 : Pancreatitis with pseudocysts

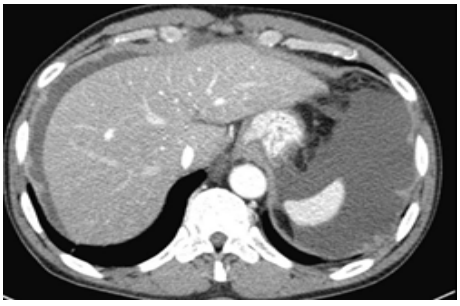

Figure 5 : Ascites

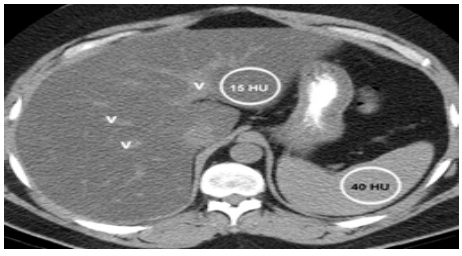

Figure 6 : Fatty liver

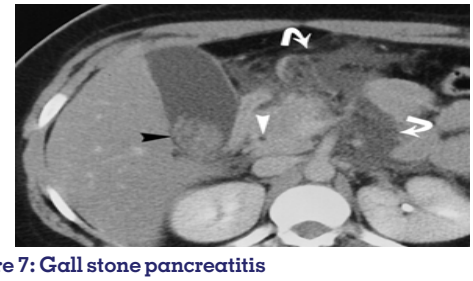

Figure 7: Gall stone pancreatitis

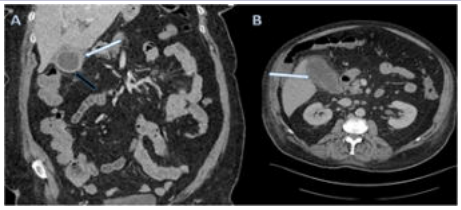

Figure 8 : Acute cholecystitis

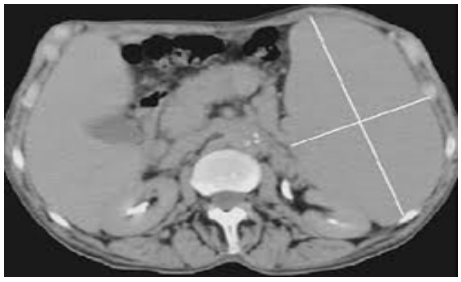

Figure 9 : Splenomegaly

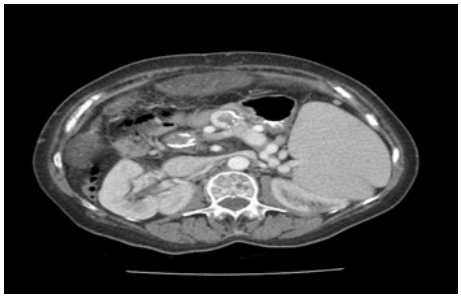

Figure 10 : Vascular thrombosis (portal vein thrombosis is shown)

REFERENCES

1. Keller J, Aghdassi AA, Lerch MM, Mayerle JV, Layer P. Tests of pancreatic exocrine function – Clinical significance in pancreatic and non-pancreatic disorders. Best Pract Res Clin Gastroenterol. 2009 Jun 1;23(3):425–39.
2. Singer MV, Gyr K, Sarles H. Revised classification of pancreatitis. Report of the Second International Symposium on the Classification of Pancreatitis in Marseille, France, March 28-30, 1984. Gastroenterology. 1985;89(3):683–5.
3. Sarner M, Cotton PB. Classification of pancreatitis. Gut. 1984;25(7):756–9.
4. Sahel J, Sarles H. Modifications of pure human pancreatic juice induced by chronic alcohol consumption. Dig Dis Sci. 1979;24:897–905.
5. Nakamura K, Sarles H, Payan H. Three-dimensional reconstruction of the pancreatic ducts in chronic pancreatitis. Gastroenterology. 1972;62(5):942–9.
6. Harada H, Miyake H, Miki H, Kobayashi T, Sasaki T, Kimura I. Role of endoscopic elimination of protein plugs in the treatment of chronic pancreatitis. Gastroenterol Jpn. 1982;17:463–8.
7. Tsurumi T, Fujii Y, Takeda M, Tanaka J, Harada H, Oka H. A case of chronic pancreatitis successfully treated by endoscopic removal of protein plugs. Acta Med Okayama. 1984;38(2):169–74.
8. Harada H, Hayashi T, Miki H, Miyake H, Ochi K, Kimura I, et al. Histochemical studies on enzyme-digested protein plugs of patients with chronic pancreatitis: a preliminary report. Acta Med Okayama. 1983;37(3):227–31.
9. Harada H, Ueda O, Yasuoka M, Nakamura T, Kunichika K, Ikubo I, et al. Histochemical studies on protein plugs obtained by endoscopic retrograde catheterization of the papilla. Gastroenterol Jpn. 1981;16:563–7.
10. Freedman SD, Sakamoto K, Venu RP. GP2, the homologue to the renal cast protein uromodulin, is a major component of intraductal plugs in chronic pancreatitis. J Clin Invest. 1993;92(1):83–90.
11. Yadav D, Timmons L, Benson JT, Dierkhising RA, Chari ST. Incidence, Prevalence, and Survival of Chronic Pancreatitis: A Population-Based Study. Am J Gastroenterol. 2011 Dec;106(12):2192–9.
12. Lin Y, Tamakoshi A, Matsuno S, Takeda K, Hayakawa T, Kitagawa M, et al. Nationwide epidemiological survey of chronic pancreatitis in Japan. J Gastroenterol. 2000 Feb 1;35(2):136–41.
13. Maringhini A, Ciambra M, Patti R, Randazzo MA, Dardanoni G, Mancuso L, et al. Ascites, pleural, and pericardial effusions in acute pancreatitis. Dig Dis Sci. 1996 May 1;41(5):848–52.
14. Bush N, Rana SS. Ascites in Acute Pancreatitis: Clinical Implications and Management. Dig Dis Sci. 2022 Jun 1;67(6):1987–93.
15. Ohara H, Nakazawa T, Sano H, Ando T, Okamoto T, Takada H, et al. Systemic Extrapancreatic Lesions Associated With Autoimmune Pancreatitis. Pancreas. 2005 Oct;31(3):232–7.