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EPIDEMIOLOGY, INCIDENCE, ETIOLOGY, COMPLICATIONS, AND OUTCOME OF ACUTE PANCREATITIS IN KASHMIRI POPULATION: A RETROSPECTIVE HOSPITAL-BASED SINGLE CENTRE STUDY.

Dr. Neeraj Dhar¹, Dr. Sayed Mushfiq Shafi¹, Dr. Jaswinder Singh², Dr. G.M. Gulzar²

1. DM, Gastroenterology, Consultant, Accord Superspeciality, Faridabad 2. Professor, Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar, JK UT

ARTICLE INFO	ABSTRACT ORIGINAL RESEARCH ARTICLE
Article History Received: June 2022 Accepted: July 2022 Key Words: Acute Pancreatitis, Inflammatory disease, Gallstone, Clinical profile and Severity of pancreatitis.	Introduction: Acute pancreatitis is an inflammatory disease of the pancreas. It can be initiated by several factors, including gallstones, alcohol, trauma, infections and hereditary factors. About 75% of pancreatitis is caused by gallstones or alcohol. According to the physical examination, radiological findings and laboratory results the etiology of acute pancreatitis is diagnosed as biliary or non-biliary. Aims and Objectives : The present study aimed to study the clinical profile (incidence, etiology, and outcome) of Kashmiri patients with acute pancreatitis. Material and Methods : This was a hospital-based retrospective study conducted in Srinagar for a period of 2 years w.e.f. September 2017 to September 2019 on 650 patients of all age groups with clinical/Laboratory/imaging findings suggestive of acute pancreatitis. Results: Our data revealed gallstone as the most common etiological agent followed by idiopathic acute pancreatitis. Mild, moderately severe acute pancreatitis constitutes 48.61%, 24.61% and 26.76% of patients respectively. There is a statistically significant association between high body mass index >25 Kg/m ² , waist circumference >100 cm, high blood sugar >200 mg /dl, hematocrit >44, CRP >150 mg/d and worsening of acute pancreatitis.

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INTRODUCTION

Acute pancreatitis is an inflammatory disease of the pancreas. It is estimated that acute pancreatitis approximately 75% of

pancreatitis is caused by gallstones or alcohol.^[1] However, more women develop this disorder since gallstones occur with increased frequency in women.^[2,3]

The diagnosis of acute pancreatitis is made on the basis of abdominal pain characteristics, Serum lipase activity (3 findings) and contrast-enhanced computed tomography (CECT) which is the gold standard in diagnosis (figure 01) and less commonly magnetic resonance imaging (MRI) or trans abdominal ultrasonography.^[4]



Figure 1. Acute biliary pancreatitis

AIM AND OBJECTIVES:

The present study aimed to study the clinical profile (incidence, etiology and outcome) of Kashmiri patients with acute pancreatitis.

MATERIAL AND METHODS

This was a hospital based retrospective study conducted in Srinagar for a period of 2 years w.e.f. September 2017 to September 2019 on 650 patients of all age groups with clinical/ Laboratory/ imaging findings suggestive of acute pancreatitis after obtaining approval from the institute ethical committee. Informed consent was obtained from all the patients prior to enrolment in the study.

Inclusion criteria

- 1. Patients with acute abdominal pain and tenderness suggestive of pancreatitis.
- 2. Serum amylase/lipase \geq 3 times the normal.
- 3. Imaging findings (USG and/or CT)

suggestive of acute pancreatitis. **Exclusion criteria:**

- 1. Chronic calcific pancreatitis
- 2. Those patients refusing consent for participation

All the study participants were thoroughly interviewed and subjected to clinical examinations and laboratory abdominal ultrasound was performed on all the patients on the day of admission. Contrast enhanced computerized tomography (CECT) and Computed Tomography Severity Index (CTSI) and MRCP was done when required. Grading of acute pancreatitis was done according to Revised Atlanta classification. Severity was assessed by BISAP scoring system at presentation and after 48 hours of admission. Data was tabulated and analyzed with SPSS 20.0 version.

Table 1: Age distribution of study patients					
Age (Years)FrequencyPercenta					
11-25	108	16.6			
26-40	260	40			

Observations and Results

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41-55	173	26.6			
56-70	87	13.4			
71-85	22	3.4			
Mean±SD=40.1±12.83					



Fig 1: Age distribution of study patients

Table 1 depicted that majority of patients i.e. 260 (40%) in this study were 26-40 years of age whereas only 22 (3.4%) patients aged 71-

85 years. The mean age of our study patients was 40.1 ± 12.83 .

Gender	Frequency	Percentage
Male	235	36.2
Female	415	63.8
	36.2	■ Male

Table 3: Distribution of study patients as per residence						
Residence Frequency Percentage						
Rural	396	60.9				
Urban	Urban 254 39.1					

Table 2 depicted that majority of the patients were females 415 (63.8%) as compared to males 235 (36.2%) with female to male ratio 1.8:1.



Table 3 showed that out of a total of 650 patients studied, 396 (60.9%) belonged from rural area whereas 254 (39.1%) were from urban areas.

Table 4: Etiological profile with gender distribution						
	Male Female		e	Total		
Etiology	No.	%	No.	%	No.	%
Gall stone pancreatitis	74	31.5	196	47.2	270	41.5
Idiopathic	76	32.3	120	28.9	196	30.2
Ascariasis induced acute pancreatitis	19	8.1	36	8.7	55	8.5
Drug	14	6	13	3.1	27	4.2
Hyperparathyroidism	14	6	6	1.4	20	3.1
Hypertriglycredemia	13	5.5	7	1.7	20	3.1
Pancreatico biliary tumor	7	3	13	3.1	20	3.1
Pregnancy	0	0	14	3.4	14	2.2
Alcohol	8	3.4	0	0	8	1.2
Trauma	4	1.7	4	1	8	1.2
Autoimmune acute pancreatitis	1	0.4	4	1	5	0.8
Pancreatic Divisum	3	1.3	2	0.5	5	0.8
Annular pancreas	2	0.9	0	0	2	0.3

Table 4 represented that gall stone pancreatitis was seen in 74 (31.5%) males and 196 (47.2%) females, Idiopathic 76 (32.3%) males and 120 (28.9%) females, ascariasis induced acute pancreatitis was observed in 19 (8.1%) males and 36 (8.7%) females, Drug in 14 (6.0%) males and 13 (3.1%) females, Hyperparathyroidism in 14 (6%) males and 6 (1.4%) females, Hypertriglycredemia was observed in 13 (5.5%) males and 7 (1.7%) females, Pancreatico biliary tumor in 7 (3.0%) males and 13 (3.1%) females, Pregnancy in 14 (3.4%) females, Alcohol consumption in 8 (3.4%) males, Trauma in 4 (1.7%) males and 4 (1.0%) females, autoimmune acute pancreatitis was observed in 1 (0.4%) males and 4 (1.0%) females, Pancreatic Divisum was seen in 3 (1.3%) males and 2 (0.5%) females and, Annular pancreas was found in 2 (0.9%) males.

Table 5: Symptoms obs	Table 5: Symptoms observed at presentation				
Symptoms	Frequency	Percentage			
Pain Abdomen	624	96			
Nausea/vomiting	403	62			
Abdominal Distension	150	23.1			
Fever	169	26			
Jaundice	96	14.8			
Jaundice 1 Fever Abdominal Distension Nausea/vomiting Pain Abdomen 0	4.8 26 23.1 62 50 10	96 0 150			

Fig 4: Symptoms observed

Table 5 depicted that majority of the patients 624 (96%) had abdominal region followed by nausea / vomiting in 403 (62%) patients,

abdominal distension in 150 (23.1%), fever in 169 (26%) and jaundice in 96 (14.8%) patients.

Table 6: Grade of severity					
Grade Frequency Percentage					
Mild acute pancreatitis	316	48.6			
Moderate acute pancreatitis	160	24.6			
Severe acute pancreatitis	174	26.8			



Table 6 presented that out of 650 patients, 316 (48.6%) had mild acute pancreatitis, 174 (26.8%) had severe acute pancreatitis whereas moderate acute pancreatitis was observed in 160 (24.6%) patients.

Table	Table 7: Mortality as per severity of acute pancreatitis in study patients					
Mortality	Mild acute pancreatitis [n=316]	Mod severe acute pancreatitis [n=160]	Severe acute pancreatitis [n=174]	P-value		
Yes	0 (0%)	13 (8.1%)	72 (41.4%)	< 0.001*		
No	316 (100%)	147 (91.9%)	102 (58.6%)			

*Statistically Significant (P-value<0.05)

Table 7 depicted that there was statistically significant mortality (41.4%) in severe acute pancreatitis followed by (8.1%) moderately severe acute pancreatitis, with (0%) mortality in mild acute pancreatitis.

Table 8: Comparison of various parameters with severity in						
patients of acute pancreatitis						
	Mild Acute	Moderate Acute	Severe Acute			
	Pancreatitis	Pancreatitis	Pancreatitis			
Parameter	[n=316]	[n=160]	[n=174]			
Pain abdomen	316	160	174			
	-100%	-100%	-100%			
Vomiting	55	85	112			
	-17.39	-53.06	-64.28%			
Fever	14	23	44			
	-4.34%	-14.28%	-25.63%			

Jaundice	0	16	16
	0%	-10.20%	-9.45%
Pleural effusion	0	109	158
	0%	-68.12%	-90.81%
Ascites	0	47	104
	0%	-29.38%	-59.77%
Hypoxia	3	127	174
	0%	-79.59%	(100%0
CRP >150 mg/dl	13	22	118
	-4.17%	-14%	-67.85%
HCT >44 mm	27	50	117
	-8.69%	-31.61%	-67.52%
BMI >25			
(kg/m2)	61	55	87
	-19.36%	-34.69%	-50%
Mortality	0	13	72
	0%	-8.10%	-41.40%
Hospital stay	17	75	131
(> 1 week)	-5.44%	-46.94%	-75%

Table 8 depicted that patients with severe acute pancreatitis had statistically significant vomiting, fever, jaundice, pleural effusion, ascites, hypoxia, high CRP, high HCT, high BMI and mortality.

Table 9: Percentage of necrotizing pancreatitis				
Percentage of necrosis Frequency Percentage				
<30%	38	26.8		
30-50%	42	29.6		
>50%	62	43.7		
Total	142	100		

Table 9 showed that percentage of necrotizing pancreatitis was >50% in majority of patients i.e. 62 (43.7%) followed by 30-50% in 4 (29.6%) patients, 38 (26.8%) had <30% and overall 142 patients had necrotizing pancreatitis.

Table 10: Infected necrotizing pancreatitis				
Necrotizing pancreatitis Frequency Percentage				
Sterile Necrosis	44	31		
Infected Necrosis	98	69		
Total	142	100		

Table 10 represented that out of 142 patients of NP 98/142 hadinfected NP and 44/142 had sterile NP.

Table 11: Infected pancreatic necrosis with culture positivity				
IPN Frequency Percentage				
Culture +IPN	98	100		
Gm -ve	74	75.5		
Polymicrobial	60	61.2		

Table 11 described that Culture + IPN was found in 98 (100%) patients followed by Gram negative in 74 (75.5%), polymicrobial in 60 (61.2%) patients.

Table 12: Microbiology profile of organisms in IPN			
	Organism	Frequency	Percentage
	E.coli	52	53.1
	Klebsiella pneumonia	21	21.4
Gram negative	Pseudomonas Aeruginosa	14	14.3
Gram positive	Enterococcus faecium	11	11.2
	Total	98	100

Table 12 showed that E. coli was isolated in 52 (53.1%), followed by Klebsiella pneumonia in 21 (21.4%) and pseudomonas aeruginosa in 14 (14.3%) whereas in Gram positive, enterococcus faecium was seen in 11 (11.2%).

Table 13: Outcome in infected pancreatic necrosis			
Intervention No. Mortality			
Antibiotics	26	5 (19.2%)	
Antibiotics with Percutaneous	72	24 (33.3%)	
Drainage			

Table 13 showed that IPN (98), 26/98 were managed conservatively out of which 5 expired, 72/98 were managed with antibiotics and percutaneous drainage, out of which 24 expired.

Table 14: Outcome in severe acute pancreatitis (severe acute pancreatitis)		
severe acute pancreatitis	No.	Mortality
Early Severe Acute Pancreatitis	90	45 (50%)
OF+IPN	84	27 (32.1)
Total	174	72 (41.4)

Table 14 depicted that out of 174 patients of severe acute pancreatitis 90/174 had (early organ failure) early severe acute pancreatitis, 45/90 expired and 84/174 had Late OF+IPN, 27/84 expired.

Table 15: Risk factors for worsening of acute pancreatitis				
	Mild Pancreatitis		Moderate- Severe Pancreatitis	
Risk Factors	No.	%age	No.	%age
BMI >25 Kg/m ²	61	19.3	142	42.5
Waist circumference >100cm	23	7.3	129	38.6
High blood sugar >200 mg/dl	37	11.7	151	45.2
Hematocrit > 44	7	8.5	167	50
CRP >150 mg/dl	3	4.1	140	41.9

Table 15 represented that there is statistically significant association between high BMI >25 Kg/m², waist circumference >100 cm, high blood sugar >200 mg /dl, hematocrit >44, CRP >150 mg/d, and worsening of acute pancreatitis.

DISCUSSION:

In this retrospective study 650 patients were included. Detailed examinations and investigations were carried out in all the cases. Data was analyzed and discussed with previous literature.

Majority of patients i.e. 40% in this study were 26-40 years of age while as 3.4% patients aged 71-85 years. The mean age of our study patients was 40.1±12.83. Similar observations were made by Balthazar EJ et al ^[5] average age was 45 years. In another study on pancreatitis done by Khanna AK et al^[6], mean age of presentation was 40.5 years.

It was observed that majority of the patients were females 415 (63.8%) females versus 235 (36.2%) males. The female to male ratio in our study was 1.8:1. Chand P et al ^[7] who enrolled 30 patients in their study 73.3% patients were males and 8 patients 26.67% were females. Balthazar EJ et al^[8] in which there were 75% male patients.

Majority of our patients were from rural areas, probably because of higher

number of referrals from peripheral hospitals due to lack of intensive care facilities in those hospitals. The study revealed gallstone is the major cause of acute pancreatitis irrespective of age and gender, although stones were more prominent in females(47.2%) as compared to males (31.5%). In a study conducted by Nesvaderani M et al^[9] found that 40% study subjects had gallstones, 25.6% idiopathic, 22% alcohol induced and 3.9% post ERCP. Similarly Vidarsdottir H et al^[10] reported the causes of acute pancreatitis was drugs in 8 (6.3%), idiopathic causes were found in 16 (12.7%) and miscellaneous causes were observed in 8 (6.3%) with male to female ratio of 4:3.

Further presenting symptoms in our study were, abdominal pain (96%) followed by nausea and vomiting (62%), distension (23.1%), fever (26%), jaundice (14.8%). Webster PD et al^[11] and Shah SSH et al^[12] reported similar results in their study.

It was found that there was statistically significant mortality (41.4%) in severe acute pancreatitis followed by (8.1%) moderately severe acute pancreatitis, with (0%) mortality in mild acute pancreatitis and patients with severe acute pancreatitis had statistically significant vomiting, fever. jaundice, pleural effusion, ascites, hypoxia, high CRP, high HCT, high BMI and

mortality. In a similar study conducted by Jain S et al^[14], reported that All the 56 patients with mild acute pancreatitis survived, 4 of 72 (5%) patients with severe acute pancreatitis died and 42 of the 81(52%)patients with severe acute pancreatitis died. Similarly Singh VK et al^[15], observed that among 397 cases, there were 14 (3.5%) deaths. In our study the percentage of necrotizing pancreatitis was >50% in majority of patients i.e. 62 (43.7%) followed by 30-50% in 4 (29.6%) patients, 38 (26.8%) had <30% and overall 142 patients had necrotizing pancreatitis. Out of 142 patients of NP had infected NP and 44 had sterile NP. In similar study conducted by Pacute pancreatitis achriston

Culture + IPN was found in 98 (100%) patients followed by Gram negative in 74 (75.5%), polymicrobial in 60 (61.2%) patients. E. coli was isolated in 52 (53.1%), followed by Klebsiella pneumonia in 21 (21.4%) and pseudomonas aeruginosa in 14 (14.3%)whereas in Gram positive. enterococcus faecium was seen in 11 (11.2%). Jain S et al¹⁴ concluded in an observational study concluded that out of Eighty-seven out of 108 (81%) patients had culture positive IPN and 86% of cultures grew MDR organism(s). Fungal infection was present in 13% (27/209) of patients. Seventy-five out of 87 (86%) patients had Gram negative bacteria. Poly-microbial infection was seen 44/87 (51%) patients. The in most common Gram-negative bacterium was Escherichia coli in 48/149 (32%)isolates and most common Gram-positive bacterium was Enterococcus faecium in 10/149 (7%) isolates.

It was observed that 26 patients were managed conservatively out of which 5 expired, 72 patients were managed with antibiotics and percutaneous drainage, out of which 24 expired and out of 174 patients of sacute pancreatitis 90/174 had (early organ failure) esacute pancreatitis, 45/90 expired

and 84/174 had Late OF+IPN, 27/84 expired. Zerem E et al^[16], reported that 17.4% were successfully treated with conservative treatment only. Percutaneous catheter drainage was performed in 80.2% and 9.3% died, two at week 1 without drainage or surgery and 6 after percutaneous catheter drainage and surgery. 12.8% patients were converted to surgery. Similarly Jain S et al^[14], found that Among patients with severe acute pancreatitis, patients with early severe acute pancreatitis had a mortality of 43%. Of these patients with early severe acute pancreatitis, 79% had single OF, 21% had 2 OF with higher mortality in those with multiple OF. This concludes that early mortality in severe acute pancreatitis mostly due SIRS and multiorgan failure and late onset mortality is due to persistent organ failure and infected necrosis.

In our study high BMI >25Kg/m², waist circumference >100 cm, high blood sugar >200 mg/dl, hematocrit >44 and CRP> 150 mg/dl are risk factors associated with worsening of acute pancreatitis which was found to be statistically significant with p value 0.001. In similar study conducted by Jin Z et al^[13] indicated that there were 5 significant differences between patients who developed mild severe acute pancreatitis or severe acute pancreatitis and those who did not: VFA (>100 cm²) (p=0.003), BMI (>25 kg/m^2) (p=0.001), Ranson score (p=0.004), APACHE-II (\geq 5) (p=0.001), and blood level admission glucose on (>11.1 mmol/L) (p=0.040). Further multivariate logistic regression analyses revealed that BMI $(\geq 25 \text{ kg/m}^2)$ (p=0.005), APACHE-II (≥ 5) (p=0.001), and blood glucose level on admission (>11.1 mmol/L) (p=0.004) were independent risk factors for developing mild severe acute pancreatitis or severe acute pancreatitis in patients admitted with mild acute pancreatitis.

CONCLUSION:

The present study concluded that the Kashmir valley has increased number of

cases of acute pancreatitis in recent years which is one of the major causes of admission in the gastroenterology ward. The increased incidence of acute pancreatitis in recent years is thought to be due to change in life style and food habits of patients. The sedentary life style has increased the incidence of obesity, dyslipidemia, diabetes and gall stones, all presumed to be risk factors for acute pancreatitis.

REFERENCES

- 1. Ilhan M, Alis H. Acute Biliary Pancreatitis. In: Rodrigo L. (Editor) Acute Pancreatitis. Chacute pancreatitister 1, Rijeka, Croatia, 2011, PP 1.
- Eland IA, Sturkenboom MJ, Wilson JH, Stricker BH. Incidence and mortality of acute pancreatitis between 1985 and 1995. Scand J Gastroenterol 2000; 35: 110-16.
- 3. Chen DB, Wang L, Wang PH, et al. Insulin-like growth factor I retards acute pancreatitisoptotic signaling induced by ethanol in cardiomyocytes. Life Sci 2000; 67: 1683-93.
- Tenner S, Baillie J, DeWitt J, Vege SS; American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol 2013; 108(9): 1400-15.
- 5. Balthazar EJ, Ranson JHC, Naidich DP, et al. Acute-pancreatitis prognostic value of CT. Radiology 1985; 3: 767–72.
- Khanna AK, Meher S, Prakash S, Tiwary SJ, Singh U, Srivastava A Et al. Comparison of Ranson, Glasgow, MOSS, SIRS, BISAP, APACHE-II, CTSI Scores, IL- 6, CRP, and Procalcitonin in Predicting Severity, Organ Failure, Pancreatic Necrosis, and Mortality in Acute Pancreatitis. HPB Surgery. 2013; 367581:1-10.
- 7. Chand P, Singh R, Singh DP, Rani N.

Evaluation of the outcome of acute pancreatitis by Ranson's criteria and modified CT severity index. International Journal of Contemporary Medicine Surgery and Radiology. 2017; 2(2):58-61.

- 8. Balthazar EJ, Ranson JHC, Naidich DP, et al. Acute-pancreatitis—prognostic value of CT. Radiology 1985; 3: 767–72.
- 9. Nesvaderani M, Eslick GD, Vagg D, Faraj S, Cox MR. Epidemiology, aetiology and outcomes of acute pancreatitis: A retrospective cohort study. Int J Surg. 2015; 23(Pt A):68-74.
- Vidarsdottir H, Moller PH, Vidarsdottir H, Thorarinsdottir H, Bjornsson ES. Acute pancreatitis. European Journal of Gastroenterology and Hepatology 2013;25(9):1068–75.
- Webster PD. Pathophysiology and management of acute pancreatitis. Hosp Prac. 1974; 56-66.
- 12. Shah SSH, Ansari MA, Ali S. Early prediction of severity and outcome of acute severe pancreatitis. Pak J Med. Sci. 2009; 25: 619-623.
- 13. Jin Z, Xu L, Wang X and Yang D. Risk factors for worsening of acute pancreatitis in patients admitted with mild acute pancreatitis. International Journal of Science and Research (IJSR). 2016; Volume 5, Issue 7.
- 14. Jain S, Mahacute pancreatitisatra SJ, Gupta S, Garg MK. Infected pancreatic necrosis due to multi-drug resistant organism and persistent organ failure predict mortality in acute pancreatitis. Clinical and Transnational Gastroenterology 2018; 9: 190.
- 15. Singh VK. Prospective Evaluation of the Bedside Index for Severity in Acute Pancreatitis Score in Assessing Mortality and Intermediate Markers of Severity in Acute Pancreatitis. Am J Gastroenterol 2009; 104:966–971.
- 1. Zerem E, Imamovic G, Susic A, Haracic

B. Step-up acute pancreatitisproach to infected necrotizing pancreatitis: a 20 year experience of percutaneous drainage in a single center. Dig Liver is 2011; 43(6): 478-83.