Research Article
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The outcome of acute pancreatitis patients with acute kidney injury- Single centre study from North India

Abstract

Introduction: Acute Pancreatitis (AP) results in 100,000 hospitalizations per year and the incidence of acute pancreatitis ranges from 13 to 45 per 100,000 population-years. Eighty percent of cases of AP are interstitial and mild; the remaining 20% to 30% are necrotizing and severe with hospital mortality rates of 15%.[1] Acute kidney injury (AKI) is a common serious complication of AP and an important marker of morbidity and mortality in critically ill patients. The prognosis of AP patients with AKI is extremely poor with mortality rates ranging between 25-75%.[2,3]

Methodology: Study type and design: observational**, c**ross sectional study, single centre study. The main source of data for the study was collected from patients with AP admitted in Department of General Medicine and Gastroenterology department of Santokba Durlabhji Memorial Hospital, Jaipur, India.

Results: AKI was found in 27 out of total 144 study subject resulting in an incidence of 18.75% in our study. Mean age of patients who developed AKI was significantly higher when compared to patients without AKI with significant p value. In our study, we found that high BMI and high SOFA score at admission were risk factors for AKI. Patients of AP with AKI had longer average hospital stay and ICU stay when compared to patients without AKI. We also found high mortality in AP patients who developed AKI had (48.15% Vs 1.71%) as compared to patients of AP without AKI.

Conclusion: From current study we can conclude that, AKI is a serious complication of AP and leads to a poor outcome.

Keywords: Acute kidney injury, severe acute pancreatitis, mortality

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Abbreviations: AP, acute pancreatitis; AKI, acute kidney injury; SAP, severe acute pancreatitis; CBC, complete blood counts; CKD, chronic kidney disease; NIV, non invasive ventilation, CAD, coronary artery disease; CLD, chronic liver disease; COPD, , chronic obstructive pulmonary disease; SOFA, Sequential Organ failure assessment

Introduction

Acute pancreatitis (AP) is defined as an acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems. According to JPN criteria 2002,[4] AP is best diagnosed clinically by a patient presenting with two of the following criteria:

1. Acute abdominal pain and tenderness in the upper abdomen;
2. Elevated pancreatic enzyme levels in serum, urine or ascitic fluid; and
3. Ultrasonographic (US) or radiologic abnormalities characteristic of AP

Worldwide, the incidence of acute pancreatitis ranges between 20 and 80 per 100,000 populations.[5,6] Pathologically, AP varies from mild *interstitial pancreatitis*, which is generally self-limited to severe *necrotizing pancreatitis*, in which the extent of necrosis may correlate with the severity of the attack and its systemic complications.

The courses of most AP cases are auto- restricted, but about 20%-30%[7] of all patients develops severe acute pancreatitis (SAP) and may become worse, due to development of multiple organ malfunctions or local complications like necrosis, pseudocyst and abscess. The inception of SAP is hazardous with relatively high mortality estimated as from 7% to 47%.[8–10] One common complication of SAP in critically ill patients is acute kidney injury (AKI). The mortality in SAP with AKI is 10 fold higher as compared to patients without AKI. [2,11–13] The exact mechanism of AKI in patients with AP is complex and not very well understood. Different key pathophysiologic processes include release of pancreatic enzymes with resulting impairment of renal microcirculation, hypoxemia, hypovolemia, intra-abdominal hypertension, endotoxin and cytokines mediated injury.

The aim of our study is to access the risk factors for the development of AKI in patients of AP, and the disease spectrum and outcome of such patients.

AIM of the study

The aim of our study is to access the risk factors for the development of AKI in patients of AP, and the disease spectrum and outcome of such patients.

Materials and methods

Study type and design: This is observational, cross sectional, single centre study.

Study population: The diagnosed patients of AP admitted in the department of General Medicine and department of Gastroenterology at Santokba Durlabhji Memorial Hospital, Jaipur, India were included in the study.

The patients were followed till discharge or death.

Inclusion criteria

All patients above 18years of age admitted in ward/ICU with first episode of acute pancreatitis with AKI at admission or developed during hospital stay were included in the study.

Exclusion criteria

a) Patients not willing to give consent for the study.

b) Patients of chronic pancreatitis.

c) Patients of recurrent pancreatitis.

d) Patients having malignancy.

Study period: 2years

Method of data collection

After obtaining informed written consent from the patient, detailed thorough general and systemic examination was carried out. Reports of all relevant laboratory and radiological investigation were generated and procured. All information & data thus generated was recorded on a pre-designed study proforma. Separate proforma was used for each patient and was used to prepare master chart.

Methodology

After giving full explanation regarding the study, patients were requested to give written informed consent. Once the eligibility was verified a detailed history and thorough clinical examination including vitals, general physical examination, systemic examination and investigations were carried out.

AP was diagnosed based on JPN criteria 2002.[4] Etiology of AP was studied in each patient on the basis of history given by patient, evidence of gall stones by radiological investigations like Ultrasonography, CT scan etc. and laboratory investigations like serum calcium, triglycerides etc. The severity of the AP was assessed according to Modified Atlanta Classification.[14] Investigations like Complete Blood Counts (CBC), liver function tests (serum bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, PT, INR, serum albumin), serum creatinine, Serum electrolytes, serum amylase, lipase were carried out. Local complications of AP like fluid collection, pseudocyst, necrosis, WOPN etc. were studied in each patient. Sequential Organ failure assessment score (SOFA) was calculated for each patient at admission (Figure 1).[15] AKI was defined as per AKIN criteria and once AKI was diagnosed, patients were further categorized to various stages of AKI by measuring Serum creatinine and Urine output of each patient according to AKIN criteria (Figure 2).[16] Patients having AKI were labeled as having stage I, II or III based on AKIN criteria. The worst value of S.creatinine was taken to stage AKI. For those patients requiring RRT number of days the patient was on RRT, the modality of RRT utilized (HD, SLED, CRRT etc.) and the status of renal recovery at the time of discharge were studied by recording serum creatinine and urine output. Risk factors for the development of AKI in these patients e.g. smoking, alcohol, pre-existing renal disease, hypoxemia, sepsis, abdominal ascites etc. were studied in detail. Extra renal involvement, duration of hospital stay, duration of ICU stay and complete course of treatment throughout admission in each of these patients were studied.

System	Score				
	0	1	2	3	4
Respiration					
PaO_2/FIO_2, mmHg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation					
Platelets, $\times 10^3$ μL^{-1}	≥150	<150	<100	<50	<20
Liver					
Bilirubin, mg dL^{-1} (μmol L^{-1})	<1.2 (20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0 (204)
Cardiovascular	MAP ≥ 70 mmHg	MAP < 70 mmHg	Dopamine < 5 or dobutamine (any dose)[a]	Dopamine 5.1–15 or epinephrine ≤ 0.1 or norepinephrine ≤ 0.1[a]	Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1[a]
Central Nervous System (CNS)					
Glasgow Coma Scale score[b]	15	13–14	10–12	6–9	<6
Renal					
Creatinine, mg dL^{-1} (μmol L^{-1})	<1.2 (110)	1.2–1.9 (110–170)	2.0–3.4 (171–299)	3.5–4.9 (300–440)	>5.0 (440)
Urine output, mL per day				<500	<200

FIO_2: fraction of inspired oxygen; MAP: mean arterial pressure; PaO_2: partial pressure of oxygen.
[a]Catecholamine doses are given as $\mu g kg^{-1} min^{-1}$ for at least 1 h.
[b]Glasgow Coma Scale scores range from 3 to 15; higher score indicates better neurological function.

Figure 1 SOFA score

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Stages	Serum creatinine criteria	Urine output
Stage 1	Increase in serum creatinine >O.3mcVd1 (È26Alm01/1) or increase to ÈI 50% to 200% (1.5 fold to 2 fold) from baseline	<0.5 ml/kg/h for >6 h
Stage 2	Increase to >200% to 300% (2 fold to 3 fold) from baseline	for <0.5 ml/kg/h for >12h
Stage 3	Increase in serum creatinine to >300% (>3 fold) from baseline, or serum creatinine >4.0mg/dl (È354ktmol/1) with acute increase 12 hours of atleast 0.5m dl	<0.3m1/kg/hr for 24 hours, or anuria for 12 hours

Figure 2 AKIN criteria

Statistical analysis

Continuous data will be presented as mean and standard deviation whereas categorical data will be presented as number, percentages. Student T Test will be applied for continuous data whereas Fischer's exact test will be applied for categorical data appropriately. Sensitivity, specificity, positive predictive value, negative predictive value, likelihood ratio for positive test result and negative test result was calculated as per standard formulae. P-value less than 0.05 were taken as significant. Medcalc. Software was used for all statistical analysis.

Observations and results

A total of 144 patients with AP were included in our study and out of these 27 patients (18.75%) developed AKI. Out of total 144 patients, 76.39% (n=110) were male and 23.61% (n=34) were female. Out of the total 27 patients with AKI, 77.78% (n=21) were male and 22.22% (n=6) were female. Mean age of patients who developed AKI was 50.48±20.45 which was significantly higher when compared to patients without AKI (40.97±16.13) with a P value of 0.009.

We observed that BMI of AP patients, who developed AKI, was higher (24.56±3.08kg/$m^{2)}$ when compared to patients without AKI (22.90±2.36kg/$m^{2)}$ with p value 0.002. In our study, AP patients who had higher SOFA score at admission had greater chance of developing AKI. The score was significantly higher in patients with AKI (5.11±2.93) compared to patients without AKI (0.85±1.12) with p value of <0.001.

Mean±standard deviation serum creatinine at admission in patients with AKI group was 3.53±3.11 while in non AKI group it was 0.73±0.20. Patients with AKI had lower urine output in first 24 hours (858.91ml±720.86 ml) compared to those who didn't develop AKI (1775.56 ml±1249.61 ml). Values of other laboratory parameters like amylase and lipase are given in Table 1.

Table 1 Demography and laboratory data

Parameters	AKI (N= 27)	Non-AKI (N=117)	Total (N=144)	P value
Age	50.48±20.45	40.97 ±16.13	42.75±17.35	0.009
BMI	24.56±3.08	22.90±2.36	23.21± 2.58	0.002
SOFA score	5.11±2.93	0.85±1.12		<0.00
Serum Creatinine	3.53±3.11	0.73±0.20		
Urine Output	858.91±720.86	1775.56±1249.61		0.04
Amylase	1162.64±1772.39	1260.61±1494.97	1236.11±1558.80	0.80
Lipase	5784.59±10163.55	8088.54±10395.73	7512.56±10328.77	0.37
Co morbidities				
DM	6 (22.22)	19 (16.24)	25 (17.36)	0.57
HTN	11 (40.74)	25 (21.37)	36 (25)	0.04
CAD	5 (18.52)	6 (5.13)	11(7.64)	0.03
CLD	5 (18.52)	11 (9.40)	16 (11.11)	0.18
COPD	3 (11.11)	1 (0.85)	4 (2.78)	0.02
Risk factors				
Smoking	3 (11.11)	18 (15.38)	21 (14.58)	0.76
Alcohol	11 (40.74)	40 (34.19)	51 (35.42)	0.51
Hypoxemia	19 (70.37)	7(5.98)	26 (18.06)	<0.001
Pre existing CKD	6 (22.22)	4 (3.42)	10 (6.94)	0.003
Other associated factors				
Pleural Effusion	28 (19.58)	11 (40.74)	17 (14.66)	0.005
Ascites	21 (14.69)	12 (44.44)	9 (7.76)	<0.001
Sepsis	17 (11.89)	12 (44.44)	5 (4.31)	<0.001
Shock	6 (4.20)	6 (22.22)	0 (0)	<0.001
Hospital stay (Days)	12.22±8.60	8.41±5.87	9.13±6.60	0.006
ICU stay (Days)	8.07±8.99	1.74±4.31	2.93±5.99	<0.001
Mortality	13 (48.15)	2 (1.71)	15 (10.42)	<0.001

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Presence of associated co morbidities like diabetes, coronary artery disease (CAD), chronic liver disease (CLD), hypertension, chronic obstructive pulmonary disease (COPD) did not make any significant impact on development of AKI in our study as evident from Table 1. Also traditional risk factors like smoking and alcohol were not associated with development of AKI in patients with AP. However, presence of hypoxemia (p value 0.001) requiring noninvasive ventilation (NIV) or mechanical ventilation at admission and preexisting chronic kidney disease (CKD) (p value 0.003) had significant impact on development of AKI in patients with AP which was statistically significant.

In our study, AP with AKI had significant impact on overall outcome of patients in terms of longer hospital & ICU stay and mortality. Patients of AP with AKI had average hospital stay of 12.22±8.60 days Vs 8.41±5.87 in non AKI patients. ICU stay in AKI group (8.07±8.99) was also longer when compared to non AKI group (1.74±4.31). AP patients who developed AKI had significantly high mortality (48.15% Vs 1.71%) as compared to patients of AP without AKI.

In our study, we found that presence of possible reasons of abdominal compartment syndrome (ACS) (third spacing of fluid) in terms of pleural effusion (p-value=0.005) and ascites (p-value <0.001) were associated with AKI. AP patients who developed sepsis (p-value <0.001) and shock (p-value <0.001) were associated with AKI.

Etiology of AP had no correlation with development of AKI in our study (Table 2). Similarly complications of AP were no different in both the groups. But, severity of AP was associated with higher incidence of AKI. It was observed in our study that 35 patients (24.31%) out of 144 developed severe AP. Only 10 % patients developed AKI in patients with mild to moderately severe AP while nearly 50% patients had AKI in patients with SAP. Conversely, those who had AKI, nearly 63% (17 out of 27) had severe AP while SAP was only 15% (18 out of 117) in non AKI group (Table 2).

Table 2 Characteristics of Acute Pancreatitis

S. No	Characteristics of AP	Total (N=144)	AKI (N=27)	Non-AKI (N=117)	P value
1.	Etiology of AP				
	Alcohol	51 (35.42)	12 (44.44)	39 (33.33)	
	Biliary	39 (27.08)	6 (22.22)	33 (28.21)	
	Unknown	45 (31.25)	5 (18.52)	40 (34.19)	0.08
	Others	9 (6.25)	4 (14.81)	5 (4.27)	
2.	Severity of AP				
	Mild AP	71 (49.31)	8 (11.26)	63 (88.74)	
	Moderately Severe AP	38 (26.39)	2 (5.26)	36 (94.74)	
	Severe AP	35 (24.31)	17 (48.57)	18 (51.42)	<0.001
3.	Complications of AP				
	Collection	28 (19.44)	4 (14.81)	24 (20.51)	0.59
	Pseudocyst	13 (9.03)	2 (7.41)	11 (9.40)	0.74
	Necrosis	18 (12.50)	3 (11.11)	15 (12.82)	0.80
	Cholelithiasis	4 (2.78)	0 (0)	4 (3.42)	0.33
	Choledocholithiasis	19 (13.19)	4 (14.81)	15 (12.82)	0.78
	WOPN	4 (2.78)	0 (0)	4 (3.42)	0.33
	Cholecystitis	4 (2.78)	0 (0)	4 (3.42)	0.33

In our study, 27 (18.75%) out of 144 developed AKI. In these 27 patients of AP with AKI, 14 (51.85%) had Stage I AKI, 4 (14.81%) had Stage II AKI and 9 (33.33%) had Stage III AKI. Out of total 27 patients of AP with AKI, 9 (33.33%) required RRT whereas 18 (66.66%) did not require RRT (Table 3). At the time of discharge, 14 (51.85%) recovered their renal functions completely, 1 (3.70%) had partial recovery, while 12 (44.44%) did not recover renal function. Patients of AP with AKI had very high mortality 48.15% (13 out of 27) as compared to total of 117 patient of AP without AKI where mortality was 1.71%. Total of 15 (10.42%) patients expired in our study. Of those patients, who expired, 13 (85%) had AKI and 2 didn't have AKI. Out of 9 patients requiring dialysis, 6 (66.6%) patients expired and only 3 survived.

Table 3 Characteristics of AKI

AKI (n= 27	AKI Stage			RRT		Recovery		
	Stage I	Stage II	Stage III	AKI with RRT	AKI without RRT	Complete	Partial	Non recovery
	14	4	9	9	18	12	1	14

Discussion

Acute Pancreatitis results in 100,000 hospitalizations per year and the incidence of acute pancreatitis ranges from 13 to 45 per 100,000 population-years.[17] Eighty percent of cases of AP are interstitial and mild; the remaining 20% to 30% are necrotizing and severe with hospital mortality rates of 15%.[1]

AKI is a common serious complication of AP and an important marker of morbidity and mortality in critically ill patients. The prognosis of AP patients with AKI is extremely poor with mortality rates ranging between 25-75%.[2,3] The combination of renal disease and SAP can occur as a result of systemic conditions that affect many organs not only kidney and pancreas. The mechanism of AKI that occurs in patients with SAP remains unclear and may involve many factors. Knowledge of the pathophysiology and diagnosis of AKI following SAP might improve the therapeutic outcome of critically ill patients.

Several studies[10,12,18] attempted to identify risk factors for the development of AKI in patients of AP. With the objective of studying risk factors for AKI in patients of AP and to study spectrum and

outcome for AKI in AP, we conducted this study in SDM hospital, Jaipur (India).

Total of 144 patients of AP, 35 (24.35%) had severe acute pancreatitis. Overall, 18.75 % patients (27 out of 144 AP patients) developed AKI. We found that the incidence of AKI was significantly higher in patients of SAP (48.15%) when compared to mild to moderate AP where it was only (9.15%). Overall mortality in our study was around 10.4% but the mortality in patients who developed AKI was very high 48.15% compared to those without AKI (1.7%). Furthermore, the patients who died in our study, nearly 85% of them had AKI. Retrospective study from a tertiary care centre in India reported 19.4 % incidence of AKI in SAP patients and very high mortality (57%) in such patients which is consistent with our study.[19] Devani et al.[13] in their study reported an overall AKI incidence of 7.9% and it's independent association with higher mortality.[13] A multicenter retrospective study of critically ill SAP patients found 69% incidence of AKI.[20] In our study, approximately 33% (9 out of 27) patients of AKI required dialysis and 44.4 % did not recover renal function i.e serum creatinine didn't touch baseline and the patient was still on renal replacement therapy at the time of discharge or death. In a study by Naqvi, complete renal recovery was observed in 54% patients.[21]

In our study, we found that advanced age, higher SOFA score, high BMI, presence of sepsis, shock, hypoxemia, third spacing of fluid and pre-existing renal dysfunction were risk factors for development of AKI in AP. Etiology of acute pancreatitis had insignificant impact on AKI in our while study by Ravindra et al. found alcohol as a significant risk factor for AKI in SAP.[19] Our results were consistent with another study where the investigators found that age, Acute Physiology and Chronic Health Evaluation II scores, pre-existing renal dysfunction, abdominal compartment syndrome (ACS) were risk factors for AKI in patients with AP.(2) Similarly, study of etiology and epidemiology of AKI in SAP patients in one study, age, APACHE, renal dysfunction, ACS, sepsis were found to be independent risk factors for AKI.[20] In the same study, investigators concluded that the presence of AKI was associated with longer hospital and ICU stay which was again consistent with our study. A retrospective study of 139 AP patients found higher acute physiologic assessment and chronic health evaluation II score, higher Sequential Organ Failure Assessment score in patients with AKI.[22] This study also found lower survival rate in AKI patients.

Conclusion

In conclusion, the overall prognosis of AP patients with AKI is guarded with significantly high morbidity and mortality. Further multicentre, randomized prospective studies are required to identify etiology, risk factors and outcomes of AKI in patients with acute pancreatitis.

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Conflicts of interest

Author dealer there are no conflicts of interest.

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