IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 19, Issue 12 Ser.5 (December. 2020), PP 01-05
www.iosrjournals.org

Relationship of Gall stone with Severity of Acute Pancreatitis.

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Abstract
Background: *Acute pancreatitis is now the most common reason for hospital admission among all gastrointestinal disorders. In most countries the presence of gallbladder stones represents the most frequent and significant risk factors for developing acute pancreatitis and underlying gallstone disease accounts for between 30 and 50% of cases with pancreatitis. Little is known about whether the severity of pancreatitis depends upon persistent stone impaction or stone passage into the duodenum. Multiple theories have been proposed to describe how gallstones set off the inflammatory response in acute pancreatitis, and a commonly accepted mechanism involves a transient obstruction of the bile or pancreatic duct by an impacted or passing stone.* ***Objective:*** *In our study we aimed to assess the relationship of gall stone with severity of acute Pancreatitis. The others specific objectives were to find out the prevalence of acute pancreatitis in patients who presented with sudden attack of upper abdominal pain as well its etiological factors and complications.* ***Methodology:*** *All patients admitted with acute attack of upper abdominal pain were considered as target population from which the study population was purposively selected after clinical and biochemical confirmation of acute pancreatitis. Severity was assessed by Ranson score. This observational study was carried out from January 2018 to December 2018 at Enam Medical College and Hospital, Savar. Statistical analysis was carried out by Student's t-Test.* ***Results:*** *Total 187 patients got admitted within above mentioned time span with acute attack of upper abdominal pain. The prevalence of acute pancreatitis among them was 30% (56). Majority of the age group (40%) belonged to 41-50 year. Gall stone was the most common etiological factors in our study (38%). Idiopathic being the next common (27%). Based on Ranson score 17 patients had severe attack. Pleural effusion developed in 60% of the patients and 2 patients died due to MODS. The severity of pancreatitis was not statistically related to the presence of gall stones (p value 0.3).* ***Conclusion:*** *In our study though gallstone being the most common etiological factor it was not related to the severity of acute pancreatitis. So the presence of gallstones should not be considered to assess the severity of the disease.*

Keywords: *Acute pancreatitis (AP). RANSON score, Gallstone*

Date of Submission: 28-11-2020 Date of Acceptance: 13-12-2020

I. Introduction

Acute Pancreatitis is an inflammatory disorder of the exocrine pancreas caused, in most cases, by immoderate alcohol consumption or the passage of gallstones. Gallstone disease is the most common cause of acute pancreatitis in the Western world[1–4]. In most cases, gallstone pancreatitis (GSP) is a mild and self-limiting disease, and patients may proceed without complications to cholecystectomy to prevent future recurrence. Severe disease occurs in about 20% of cases and is associated with significant mortality, and meticulous management is critical. A thorough under-standing of the disease process, diagnosis, severity stratification, and principles of management is essential to the appropriate care of patients presenting with this common disease. A connection between gallbladder stones and pancreatitis has been suspected since at least the 17th century[5] but how gallstones confer that risk has been a matter of much debate. Claude Bernard discovered in 1856 that bile is an agent that, when injected into the pancreatic duct of laboratory animals, can cause pancreatitis[6]. It is firmly established today that the initiation of pancreatitis requires the passage of a gallstone from the gallbladder through the biliary tract[7] and gallstones that remain in the gallbladder will not cause pancreatitis. In 1901 Eugene Opie postulated that impairment of the pancreatic outflow due to obstruction of the pancreatic duct causes pancreatitis[8]. This initial 'duct obstruction hypothesis' was somewhat forgotten when Opie published his second 'common channel' hypothesis during the same year[9]. This later hypothesis predicted that an impacted

gallstone at the papilla of Vater creates a communication between the pancreatic and bile duct (the so called 'common channel') through which bile flows into the pancreatic duct and thus causes pancreatitis (Figure 1). There are several ways to define severe acute pancreatitis. It is characterized by pancreatic necrosis, severe SIRS and often multi organ failure. According to revised Atlanta classification (2013) severe acute pancreatitis is persistent organ failure (>48 hours), single or multiple organ failure[10]. The Ranson score of 3 or more at 48 hours indicates several attacks and this scoring system was used to assess the severity in this study. Other predictors of severity of interest include biochemical markers C-reactive protein has been shown to correlate with severe disease, and has been advocated as a useful single biochemical marker[11]. There is no established pathological correlation between gallstone and severity of pancreatitis but passage of stones through biliary channels may precipitate early and severe cholangitis causing severe SIRS followed by organ failure[12]. This concept provoked us to find any statistical relation between the severity and the gall stones.

Little is known about whether the severity of pancreatitis depends upon persistent stone impaction or stone passage into the duodenum. In this study we tried to find out any positive correlation between severe pancreatitis and passed stone.

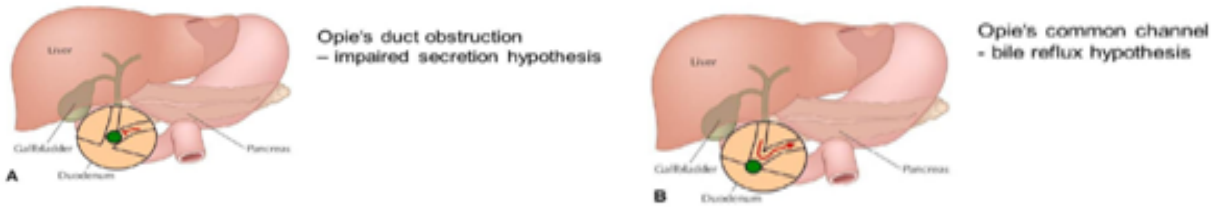

Figure I

The two "Opie hypotheses" for the pathogenesis of gallstone induced pancreatitis, both reported in 1901. A: A gallstone on its passage through the biliary tract obstructs the pancreatic duct. The impaired flow from the exocrine pancreas triggers acinar cell or duct cell damage. Whether or not the common bile duct is also obstructed is immaterial to the triggering mechanism of pancreatitis in this scenario. B: A gallstone, impacted at the duodenal papilla, creates a communication between the pancreatic duct and the common bile duct. Behind it, bile can flow through this "common channel" into the pancreatic duct and would trigger the onset of acute pancreatitis.

II. Materials And Methods

This study was carried out from January 2018 to December 2018 at Enam Medical College & hospital, Savar. All patients admitted with acute attack of upper abdominal pain were considered as the target population. Among them the patients who were diagnosed as Acute pancreatitis from history and by 3 fold rise of S. Amylase, S. Lipase was taken under study population group on the basis of simple random purposive sampling. Patients with sudden severe attack of pain as well as recurrent attack (<4 weeks) were included in the study. Those having any other systemic comorbid illness were excluded. Severity of pancreatitis was assessed by RANSON score after 48 hours of admission. Demographic variables (age, sex), were analyzed along with local (acute peripancreatic fluid collection, sterile pancreatic necrosis, infected pancreatic necrosis, pleural effusion) complication and systemic (cardiovascular, respiratory, renal failure, hematological) complication. Gallstones, alcohol, post ERCP, post trauma, Idiopathic were considered as etiological factors in our study. Among those the gall stone (independent variable) was statistically analyzed with severity of pancreatitis (Dependent variable) by Student's t-Test. p value <0.05 was considered to be statistically significant.

Data were collected with a structured form filled by the investigator after interviewing with the sample unit and were presented as tables. Statistical analysis was carried out by the software SPSS version 23.

III. Results

Total 187 patients got admitted within above mentioned time span with acute attack of upper abdominal pain. The prevalence of acute pancreatitis among them was 30% (56). And that of severe acute pancreatitis was 30.3 %(17 out of 56) (Table II). Majority of the age group (40%) belonged to 41-50 year (Table I). Gall stone was the most common etiological factors in our study (38%). Idiopathic being the next common (27%), followed by post ERCP (16%), blunt abdominal trauma (15%) and alcohol (5%) (Figure II). Based on Ranson score at 48 hours of hospital admission 17 patients had severe attack. Pleural effusion developed in 60% of the patients and 3 patients died due to MODS (Figure III). The severity of pancreatitis was not statistically related to the presence of gallstones (p value 0.3). Acute peripancreatic fluid collection was

found in 19 patients (33%) evident in USG of whole abdomen on the 1st day of admission and on subsequent USG in next 7 days evidence of regression of collection was found in 13 patients. Pleural effusion was found in 30 patients (53%) as evident by USG of whole abdomen but not causing any major respiratory complication and all responded with the ongoing conservative management on 7 days follow up. Seven patients (13%) developed SIRS but all resolved in first 72 hours. Sterile pancreatic necrosis was found in 13 patients evident by CT scan with oral and I/V contrast, among them 7 patients responded conservative management the rest went on a state of infected pancreatic necrosis. Four patients developed MODS on 4th to 5th day of monitoring and three of them died on the 6th day of follow up. Eleven patients developed ARDS, Six patients (11%) developed AKI, and 07 patients (13%) developed DIC all managed conservatively (Figure III). Among the etiological factors 21 patients had gallstone among which only seven had RANSON score > 3. Those without gall stone (35); ten had RANSON score > 3. After statistical analysis, the p value came out to be 0.3. So it was concluded from the analysis that severity of the disease is not related to the presence of gallstones (Table II).

Age Distribution (years)	n=56	%
21 years to 30 years	04	07.14
31 years to 40 years	11	19.64
41 years to 50 years	22	39.29
51 years to 60 years	16	28.57
>60 years	03	05.36
Total	56	100.00

Table I: Age distribution of the study population (n=56)

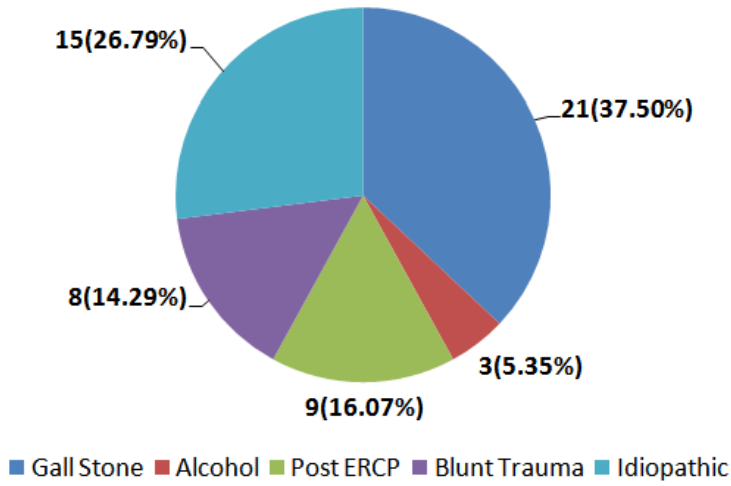

Figure II: Etiology of pancreatitis (n=56)

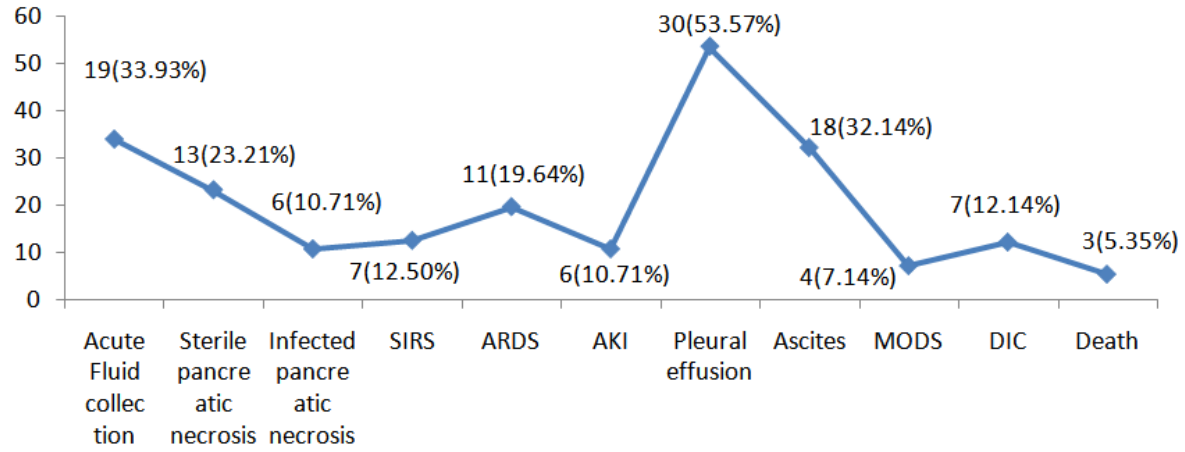

Figure III: Complications developed in the patients of Acute Pancreatitis (n=56)

Gall Stone	Ranson Score		Total	p value
	<3	>3		
Present	14	7	21	0.3
Absent	25	10	35	
Total	39	17	56	

Table II: Relationship of gall stone with RANSON Score

IV. Discussion

Gallstone pancreatitis is caused by the transient blockage of the ampulla of Vater by a migrating gallstone. The obstruction causes extracellular or intracellular activation of pancreatic enzymes. Little is known about whether the severity of pancreatitis depends upon persistent stone impaction or stone passage into the duodenum. The aim of this study was to find out any relation between the severity of pancreatitis with presence of gallstones. The main etiology of AP in this work was biliary (37%), not similar to that found by Hernandez and colleagues (72%)[11]. It is also not similar to those found in American investigations[12], but was similar to those found in European studies where alcoholic etiology is most frequent [13]. This etiology accounted for only 5% of the patients in our study. Idiopathic AP accounted for 27% of the cases in our study, in contrast to the 17.6% of Hernandez . One thing that stands out is the prevalence of severe AP (30.3%) that we found. This was higher than what has been reported in other works which indicate prevalence ranging from 12% to 25%[14,15]. The prevalence rate that we found is similar to that found by Hernandez (39%) in the Military Hospital . This suggests that in our country AP is more frequently severe. This is an alert for doctors who handle these patients. Among the causes that could explain this high prevalence of severe pancreatitis is the fact that the main cause of AP in our patients was biliary. As is known, biliary AP has a more unfavorable course [16]. Many other studies have been done to find out the factors predicting the severity of acute pancreatitis. Mayer et al[17] have recently studied the role of CRP in the assessment and monitoring of acute pancreatitis. They found that the main value of CRP is to provide a guide to the severity of inflammation and to indicate the patients' risk of developing pancreatic collections when the CRP values remain high (>100 mg/I) at the end of the first week of the illness.. Mayer also concluded that CRP could differentiate mild and severe attacks of pancreatitis better than the white blood cell count, erythrocyte sedimentation rate, body temperature or concentration of anti proteases.

As gall stone has been found as the prime etiological agents behind pancreatitis in this study so our aim was to see whether there is any statistical relationship between the severity and the presence of gallstones and we found it to be a negative predictor of severe acute pancreatitis. No study has been found so far evaluating this result. The limitations of this study were it was a single center study, sample size was small, no methods of randomization were followed and it was merely an observational study. The results of this study can be used to do a multicentre comparative analytical study in the future.

V. Conclusion

Severe Acute pancreatitis is a serious disease which imposes several local as well as systemic effects which are sufficient to cause MODS. So prediction of severity in this disease must be done early enough to prevent its progression. Presence of gall stone merely helps to know an etiology but it does not foretell anything regarding the severity. That what we analysed in this study but if we consider opie's common channel hypothesis the obstructing calculi should be extracted from ampulla to relieve cholangitis and biliary reflux in order to prevent the progression of pancreatitis.

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Dr. Ashok Kumar Sarker, et. al. "Relationship of Gall stone with Severity of Acute Pancreatitis.." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(12), 2020, pp. 01-05.