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Original article:

A study on acute pancreatitis - incidence, prevalence ,morbidity and mortality, in Western Rajasthan

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Abstract

Introduction: It has been suggested that acute pancreatitis has a benign course in Asian subcontinent, in contrast to Western populations. The aim of the present study was to ascertain the incidence of acute pancreatitis in western Rajasthan.

Material and method: In order to identify the incidence of acute pancreatitis by alcohol, an observational, retrospective and cross-sectional clinical study was performed at a tertiary care hospital in western Rajasthan.

Results: The study consisted of 100 male patients. Alcohol was identified as the predominant etiologic factor in 73.2%, biliary disease in 9.4%, and idiopathic causes in 10.6%. Substantial morbidity was encountered in 32.3% and was caused mainly by pancreatic complications, metabolic derangements, alcohol-related symptoms, and respiratory impairment. A portion (12.3%) of the patients developed further pancreatic pathology, such as pseudocysts, necroses, or an abscess. The overall mortality rate was 7.3%. Patients who died had a higher mean serum amylase. Prospective follow-up after an average of 9.3 month revealed serious morbidity in two-thirds of patients. Fifty-two percent suffered from severe abdominal pain, 33% complained of weight loss, and 21% were shown to have a sonographically abnormal pancreas. Fecal chymotrypsin levels indicated exocrine pancreatic impairment in 32.6%. In terms of age the mean was 40, median 38, mode 34, SD 7.1, minimum 25 and maximum 52 years. Risk factors: 9 cases for Diabetes mellitus, 28 cases for smoking, 11 cases with hypertriglyceridemia, 10 cases with overweight, and 78 cases with positive alcoholism.]

Conclusion: Acute pancreatitis requiring hospital admission is a severe disease associated with a high mortality and significant long-term morbidity in surviving patients. Keywords Alcohol; Acute pancreatitis; Prevalence; Morbidity; Mortality

Introduction

From the earliest century, some authors had found that alcohol intake was associated with clinical pictures of acute pancreatitis, which has been confirmed to this day. Nowadays it is widely recognized that pancreatic injure due to alcohol consumption ranges from isolated episodes of acute pancreatitis (AP) to chronic manifestations that with time could move to pancreatic cancer (PC). However, there is not a consensus in the epidemiology and it is no clear how different drinks or dose of alcohol affect to the development of pancreatic diseases and finally, how drinking trigger pancreatic injury only in a minority of alcoholics [1]. It is emphasized that the consumption of alcohol for a long time as well as the amount of alcohol affects significantly both the presentation of acute pancreatitis and the presentation of pancreatic cancer, in such a way that its ingestion increases the incidence of pancreatitis from 2.5 To 3.0% among alcohol users. Likewise, the amount from 80 to 150 ml per day and for 10 to 15 years leads to the increase of these pathologies [2,3].

Material and Method

In order to identify the incidence of acute pancreatitis by alcohol, and other etiology an observational, retrospective and cross-sectional clinical study was performed at S.P.Medical college and PBM associated group of hospitals, Bikaner,Rjasthan, during 2 years (January 2007-December 2009). Admission criteria: acute inflammatory pancreatitis associated with ingestion of alcohol, Smoking, Diabetes mellitus. The variables observed were: independent variables: diagnosis of pancreatitis, serum amylase, lipase, dependent variables: age, sex, history, severity ratings used, hospitalization days, morbidity and mortality. Descriptive statistics were used for averages, percentages and standard deviation.

Results

During the study period, 100 cases of acute pancreatitis were collected from which 80 cases of alcoholic pancreatitis were present. Regarding sex, all cases obtained were males. In terms of age the mean was 40, median 38, mode 34, SD 7.1, minimum 25 and maximum 52 years. Risk factors: 9 cases for Diabetes mellitus, 28 cases for smoking, 11 cases with hypertriglyceridemia, 10 cases with overweight, and 78 cases with positive alcoholism. Laboratory tests (**Table 1**). Ultrasound: 76 cases diagnosed with acute pancreatitis, 7 cases with hepatic steatosis, 5 case with acute chronic pancreatitis, 3 case with free fluid in the abdominal cavity, 1 case report normal and in 5 cases the study was not performed. CT scan performed 8 studies with Balthazar classification: 2 type C, 1 type D and 5 type C. Of the 100 cases, 14 suffered complications: 11 cases with peripancreatic collections and 3 case of necrohemorrhagic pancreatitis. Cases were classified with APACHE II 5, Bisap 2 and Ranson 1 cases. In terms of management, this was surgical in 7 case of hemorrhagic necrosis and the rest cases with medical treatment. ICU 9 cases required this service. The days of in-hospital stay were: mean 5, median 4, fashion 3, DE 3.7, minimum 1, maximum 11 days).

Table-1

Laboratory Test	Medium	Median	Mode	SD	Minimum	Maximum
Hematocrit	44	43	52	6	33	53
Leukocytes	13,038	12,900	N/A	5	5,500	22,000
Glucose	136	129	112	38	85	118
Total cholesterol	106	85	N/A	118	85	384
Triglicerides	346	84	N/A	483	67	1,324
Lipase	449	293	N/A	605	108	2,120
Amilase	997	537	N/A	981	105	3,378
AST	56	37	42	68	16	310
ALT	51	31	31	51	8	225
LDH	218	N/A	N/A	361	183	1,042
ALP	112	94	N/A	68	94	271
GGT	111	44	N/A	157	16	523
Calcium	5	8	N/A	4.7	8	10
BUN	14	13	13	8	6	37
Creatinine	1	0.88	1.24	0.94	0.52	4.6
Albumin	3.3	4	N/A	1.8	2.2	5.2
DB	0.69	0.45	0.45	0.87	0.09	3.77
IB	1.9	0.56	0.77	5.7	0.24	23.7
PT	8.3	11.3	N/A	7.8	0.28	19.9
PTT	16.3	20.7	N/A	14.9	0.56	37.6

Table -2

Abbreviations and normal values: Leukocytes (4000-11500 × 10^3/mm^3); Glucose (74-109 mg/dl); Lipase (13-60 U/L); Amylase (60-128 UI/L); AST: Aspartate aminotransferase (0-32 U/I); ALT: Alanine aminotransferase (0 - 33 U/I); LDH: Lactic dehydrogenase (103-227 U/l); CRP: C-reactive protein (+ or -); ALP: Alkaline phosphatase (64-306 U/I); GGT: Gama glutamil-transpepidase (0-51 U/l); Calcium (8.5-10.5 mg/dl); BUN: Blood urea nitrogen (10-20 mg/dl); Creatinine (0.4-1.4 mg/ dl); Albumin (3.5-5.3 g/dl); DB: Direct bilirrubin (0-0.20 mg/dl); IB: Indirect bilirrubin (0.075 mg/dl); PT: Prothrombin time (12.8-15.1 sec); PTT: partial thromboplastin time (24.3-35 sec)

Ultrasound Diagnostic		**TAC Balthazar**		**Miscellaneous Aspects**	
Diagnostic	*Cases*	*Type*	*Cases*	*Morbility*	*Cases*
Acute pancreatitis	76	A	0	Collections peripancreatic	3
Hepatic steatosis	7	B	0	Hemorragic necrosis	7
Peripancreatic fluid	3	C	2	Intensive care unit	3
Abdominal free fluid	3	D	1	Hospital-stay (days average)	5
Chronic pancreatitis	5	E	5	Medical management	16
Normal study	1	Total 8		Surgery treatment	11
Without US	5			Mortality	7
Total	100				

Discussion

To date, mechanisms that trigger the clinical picture of acute pancreatitis are poorly understood, so that the responsible enzymatic processes continue to be studied as well as the underlying inflammatory response, since the causative etiology of acute pancreatitis is multifactorial [4,5]. It is known that alcohol alone does not cause pancreatitis and here is proven by knowing the large number of consumers of alchol and those who did develop pancreatitis. Also that hereditary factor and environmental factors play an interesting role in alcohol-Pancreatitis association [6,7,8]. Because ethanol does not cause pancreatitis directly but only sensitizes the pancreas to cause pancreatitis, so it is assumed that the pancreas has developed self-protective effects against ethanol and some of these protective mechanisms have already been identified [9,10]. In other stuy they found that the appearance of pancreatic pseudocysts was more common in alcohol pancreatitis than in pancreatitis of biliary origin, as well as that endoscopicsphincterotomy could differentiate both entities. In our study, there was only elevan case of pancreatic pseudocyst and the clinical picture helped us to differentiate both etiologies [11]. In our study, we only found one case of chronic pancreatitis that developed acute pancreatitis, all other cases were first time, however, it is said that alcoholic individuals with chronic pancreatitis tend to develop acute pancreatitis, a situation not observed in our Patients. Similarly, genetic modification technology in these patients is not available to us for now [12]. The presentation of the pancreatitis cases by alcohol has been increased in the festivities of end of year in our environment due to the high consumption of alcoholic drinks and in individuals who ingestion of these drinks of constant form. Other authors have found the relation of festive periods and the association with morbimortality by pancreatitis [13,14]. Acute pancreatitis in an alcoholic is generally secondary to chronic pancreatitis. Determinants of individual susceptibility to alcoholic pancreatitis still remain uncertain. The studies in progress lead us to a clear understanding of the mechanisms involved in the pathogenesis of alcoholic pancreatitis. In the case of intractable pain, an intrathecal narcotics pump may be offered. In those patients in whom medical therapy failed to obtain persistent pain relief, a surgical approach should be preferred over an endoscopic approach [15].

Conclusion:

In our hospital, acute pancreatitis requiring hospital admission is a severe disease associated with a high mortality and significant long-term morbidity in surviving patients.

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