

Prevalence, Morbidity and Mortality of Acute Alcoholic Pancreatitis in the General Hospital of Southern Mexico: Analysis of Five Years (January 2012-December 2016)

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Abstract

Background: From the earliest century, some authors including Friedrech had found that alcohol intake was associated with clinical pictures of acute pancreatitis, which has been confirmed to this day.

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 the Playa del Carmen Solidaridad Hospital, Quintana Roo, Mexico.

Results: During the study period, 100 cases of acute pancreatitis were collected from which 17 cases of alcoholic pancreatitis. Regarding sex, 3 cases were obtained for females and 14 for males. In terms of age the mean was 40, median 38, mode 34, SD 7.1, minimum 25 and maximum 52 years. Risk factors: 3 cases for Diabetes mellitus, 3 cases for smoking, 7 cases with hypertriglyceridemia, 5 cases with overweight, and 17 cases with positive alcoholism.

Conclusion: In our hospital the percentage it was 17% during five years with predominance for male gender in middle age population and ranking third in place of the lipid etiology.

Keywords: Alcohol; Acute pancreatitis; Prevalence; Morbidity; Mortality

Introduction

From the earliest century, some authors including Friedrech 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 injury 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].

The latest Atlanta review of 2012 has reclassified acute and early as well as mild, moderate and severe acute pancreatitis in such a way that mild acute pancreatitis is the most common and the most rapid recovery and with fewer sequelae, such as As happened in most of the cases presented by us [4].

Materials and Methods

In order to identify the incidence of acute pancreatitis by alcohol, an observational, retrospective and cross-sectional clinical study was performed at the Playa del Carmen Solidaridad Hospital, Quintana Roo, Mexico, during 5 years (January 2012-December 2016). Admission criteria: acute inflammatory pancreatitis associated with ingestion of alcohol. 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

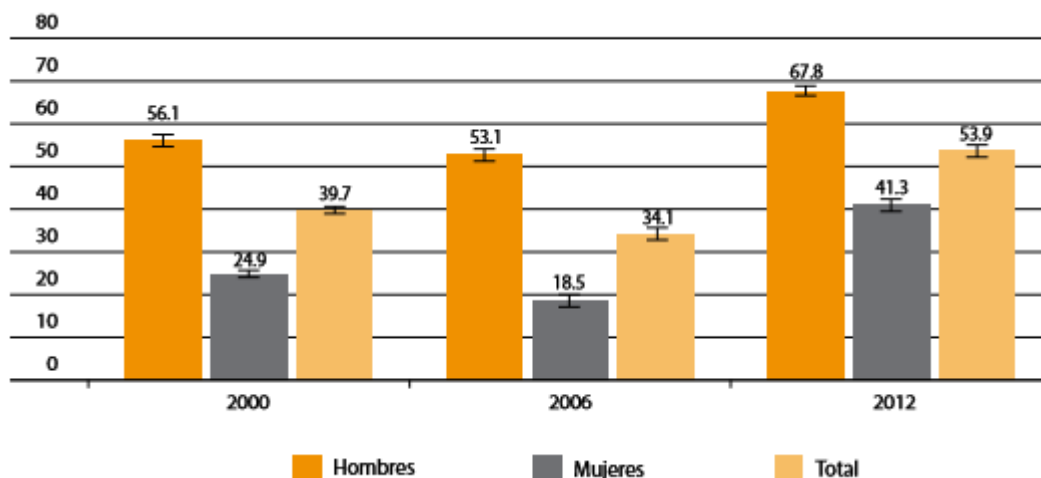
During the study period, 100 cases of acute pancreatitis were collected from which 17 cases of alcoholic pancreatitis were present (Figure 1) (2012=3, 2013=3, 2014: 3, 2015: 4, 2016: 4 cases). Regarding sex, 3 cases were obtained for females and 14 for males. In terms of age the mean was 40, median 38, mode 34, SD 7.1, minimum 25 and maximum 52 years. Risk factors: 3 cases for Diabetes mellitus, 3 cases for smoking, 7 cases with hypertriglyceridemia, 5 cases with overweight, and 17 cases with positive alcoholism. Laboratory tests (Table 1). Ultrasound: 4 cases diagnosed with acute pancreatitis, 3 cases with hepatic steatosis, 1 case with acute chronic pancreatitis, 1 case with free fluid in the abdominal cavity, 1 case report normal and in 7 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 17 cases, 4 suffered complications: 3 cases with peripancreatic collections and 1 case of necrohemorrhagic pancreatitis. Cases were classified with APACHE II 5, Bisap 2 and Ranson 1 cases (Table 2). In terms of management, this was surgical in 1 case of hemorrhagic necrosis and 16 cases with medical treatment. ICU only 3 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). Prevalence of current consumption of

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Note: *People who reported alcohol consumption on a daily, weekly, monthly or occasional basis in the last year. 95% Confidence Intervals

Figure 1: Prevalence of current consumption of alcohol. Population of 20 years or more in Mexico, ENSA 2000, ENSANUT 2006 and 2012.

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

Abbreviations and normal values: Leukocytes (4000-11500 × 10³/mm³); Glucose (74-109 mg/dl); Lipase (13-60 U/L); Amylase (60-128 UI/L); AST: Aspartate aminotransferase (0-32 U/l); ALT: Alanine aminotransferase (0 -33 U/l); LDH: Lactic dehydrogenase (103-227 U/l); CRP: C-reactive protein (+ or -); ALP: Alkaline phosphatase (64-306 U/l); 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 bilirubin (0-0.20 mg/dl); IB: Indirect bilirubin (0.075 mg/dl); PT: Prothrombin time (12.8-15.1 sec); PTT: partial thromboplastin time (24.3-35 sec)

Table 1: Statistical analysis of laboratory analyzes in 17 cases of alcoholic pancreatitis acute of 100 cases acute pancreatitis during five years (2012-2016).

alcohol. Population of 20 years or more in Mexico is shown in (Figure 1). Abuse in alcohol consumption Population aged 20 and over in Mexico (Figure 2).

Discussion

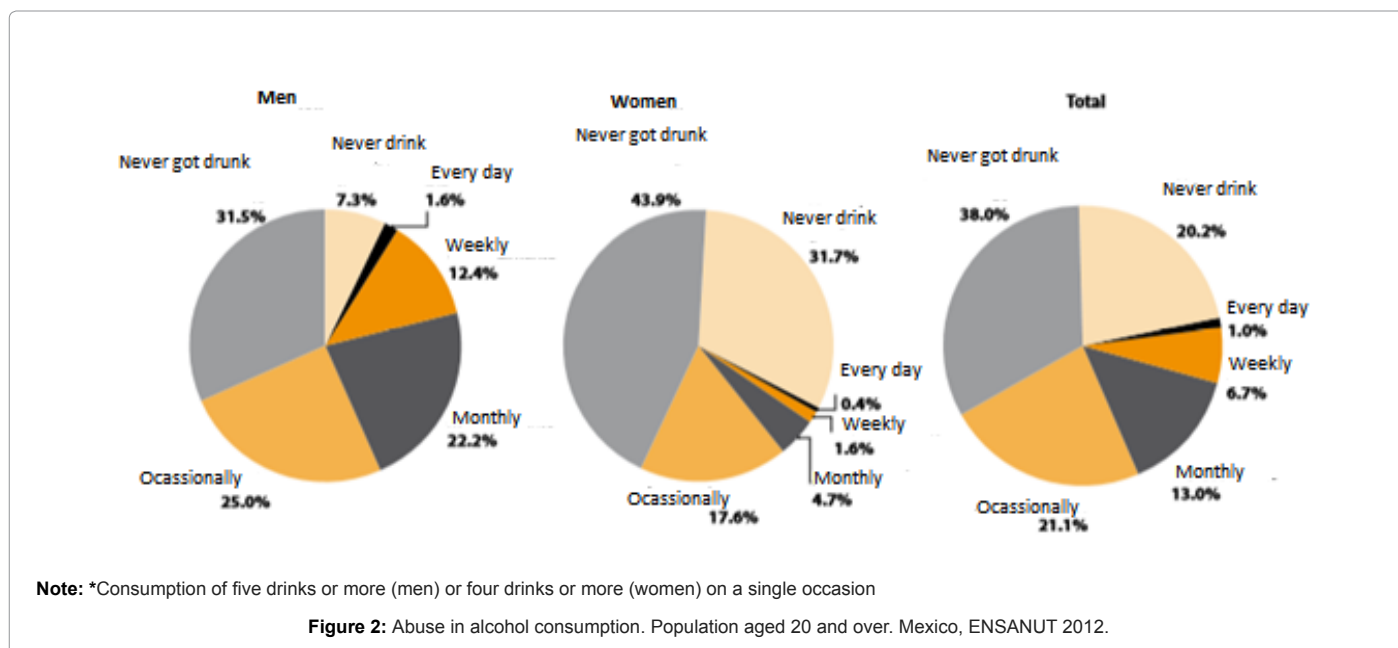
Incidence increased significantly from 27.6 per 100 000 in 1999 to 36.4 in 2010 (average annual increase=2.7% per year), there was little trend in mortality (0.2% average annual reduction).

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 [5].

The relationship was linear for CP and AP in men, but non-linear for AP in women. There was strong evidence supporting a threshold effect for AP in women at the level of alcohol consumption of up to 40

Ultrasound Diagnostic		TAC Balthazar		Miscellaneous Aspects	
Diagnostic	Cases	Type	Cases	Morbidity	Cases
Acute pancreatitis	4	A	0	Collections peripancreatic	3
Hepatic steatosis	3	B	0	Hemorrhagic necrosis	1
Peripancreatic fluid	2	C	2	Intensive care unit	3
Abdominal free fluid	1	D	1	Hospital-stay (days average)	5
Chronic pancreatitis	1	E	5	Medical management	16
Normal study	1	Total		Surgery treatment	1
Without US	5		8	Mortality	0
Total	17				

Table 2: Other results in 17 cases of alcoholic acute pancreatitis out of 100 cases acute pancreatitis during five years (2012-2016).



g/day. Beyond 40 g of pure alcohol/day, the risk of pancreatitis, both acute and chronic, regardless of sex, was higher than previously thought [6].

It is known that alcohol alone does not cause pancreatitis and this is proven by knowing the large number of consumers of alcohol and those who do not develop pancreatitis, so that hereditary factor and environmental factors play an interesting role in this alcohol-pancreatitis association [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 study they found that the appearance of pancreatic pseudocysts was more common in alcohol pancreatitis than in pancreatitis of biliary origin, as well as that endoscopic sphincterotomy could differentiate both entities. In our study, there was only one 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 the percentage it was 17% during five years with predominance for male gender in middle age population and ranking third in place of the lipid etiology.

Conflict of Interest

The authors declare no conflict of interest.

Financing

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