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Original Research

Assessment of histomorphological features of pancreas and liver in chronic alcoholics

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ABSTRACT:

Background: The present study was conducted to assess histomorphological features of pancreas and liver in chronic alcoholics. **Materials & Methods:** 114 liver and pancrease autopsies of both genders were was differentiated from ante-mortem changes histologically. H and E staining was done in all the sections, reticulin, Mason's trichrome and Elastic Van Giesen stainings were also carried out. **Results:** Gross features were enlarge in 73, shrunken in 30 and normal in 11 cases. Micronodules were seen in 64, macronodules in 36 and non- cirrhotic with normal liver in 14. Consistency was firm in 71, soft in 15, normal in 11 and necrosis was 28. The difference was significant (P< 0.05). Distribution of pancreatic fibrosis found to be Focal in 1 and 0, diffuse in 2 and 1, perilobular in 11 and 2, intralobular in 4 and 0, periductal in 2 and 1, calcifying pancreatitis in 3 and 0, ductal epithelial mucinous metaplasia in 2 and 1 in cirrhotic and non- cirrhotic in pathologic pancreatic features.

Conclusion: Parenchymal fibrosis was more frequently seen in alcoholic cirrhosis cases than that in non-cirrhotic alcoholic liver disease.

Key words: Alcoholic liver disease, Parenchymal fibrosis, Pancrease

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INTRODUCTION

Alcohol abuse and dependence are the major cause of morbidity and mortality in the United States. About three-fourths of individuals of 18-26 years of age and two-thirds of those 26 and older are current drinkers. The age related pattern for concurrent alcohol and tobacco dependence was similar to that found for tobacco dependence. In a recent survey it was found that approximately 17.6 million adult Americans abuse alcohol or is alcoholic.[1] Alcohol related problems cost society approximately $185 billion per year. Pancreatitis due to alcohol abuse is a very painful and potentially fatal condition. About one-third of acute pancreatitis cases in the United States are alcohol induced and 60%-90% of pancreatitis patients have a history of chronic alcohol consumption.

Co-existence of alcoholic related liver disease and pancreatitis has been a matter of debate and the involved mechanisms have been variously explained by many investigators. There are two schools of thoughts with respect to the events involved in disease development both in live rand pancreas. One school suggests frequent co-existence of chronic pancreatits and chronic alcohol related liver disease.[2]

The other school however believes that alcoholic pancreatitis is infrequently seen in alcoholic liver cirrhosis. Chronic pancreatitis and chronic liver disease are reportedly two conditions that have well-defined precursor lesion.[3] Recent studies have proposed chronic pancreatitis to develop through stimulation of stellate cells into myofibroblasts, which are responsible for the production of collagen and parenchymal fibrosis, and similarly there is also activation of stellate cells in liver along with steatosis and steatofibrosis ultimately resulting in liver parenchymal fibrosis and cirrhosis.[4,5]

The present study was conducted to assess

histomorphological features of pancreas and liver in chronic alcoholics.

MATERIALS & METHODS

The present study was conducted in the department of general pathology. It comprised of 114 autopsies of both genders. Ethical approval for the study was obtained before starting the study.

Patients had history of chronic alcohol abuse and clinically presented with one or more of the following problems, *i.e.* clinical and biochemical evidences of severe liver damage and died of either hepatic encephalopathy, gastrointestinal bleeding, hepatorenal failure, pancreatitis or sepsis.

All autopsies were performed within the first 2 hours. Post-mortem autolysis was differentiated from ante-mortem changes histologically. Multiple representative tissue blocks were taken from different areas of liver and pancreas. H and E staining was done in all the sections, reticulin, Mason's trichrome and Elastic Van Giesen stainings were also carried out. Degree of chronic inflammatory cells was graded as mild, moderate and heavy. Results were assessed statistically. P value less than 0.05 was considered significant (P< 0.05).

RESULTS

Table I Distribution of patients

Total- 114		
Gender	**Males**	**Females**
Number	103	11

Table I shows that out of 114 cases, 103 were seen in males and 11 in females.

Table II Assessment of Gross features

Gross features	Number	P value
Enlarge	73	0.021
Shrunken	30	
Normal	11	
Cirrhotic		
Micronodules	64	0.05
Macronodules	36	
Non- cirrhotic with normal liver	14	
Consistency		
Firm	71	0.01
Soft	15	
Normal	11	
Necrosis	28	

Table II shows that gross features was enlarge in 73, shrunken in 30 and normal in 11 cases. Micronodules were seen in 64, macronodules in 36 and non- cirrhotic with normal liver in 14. Consistency was firm in 71, soft in 15, normal in 11 and necrosis was 28. The difference was significant (P< 0.05).

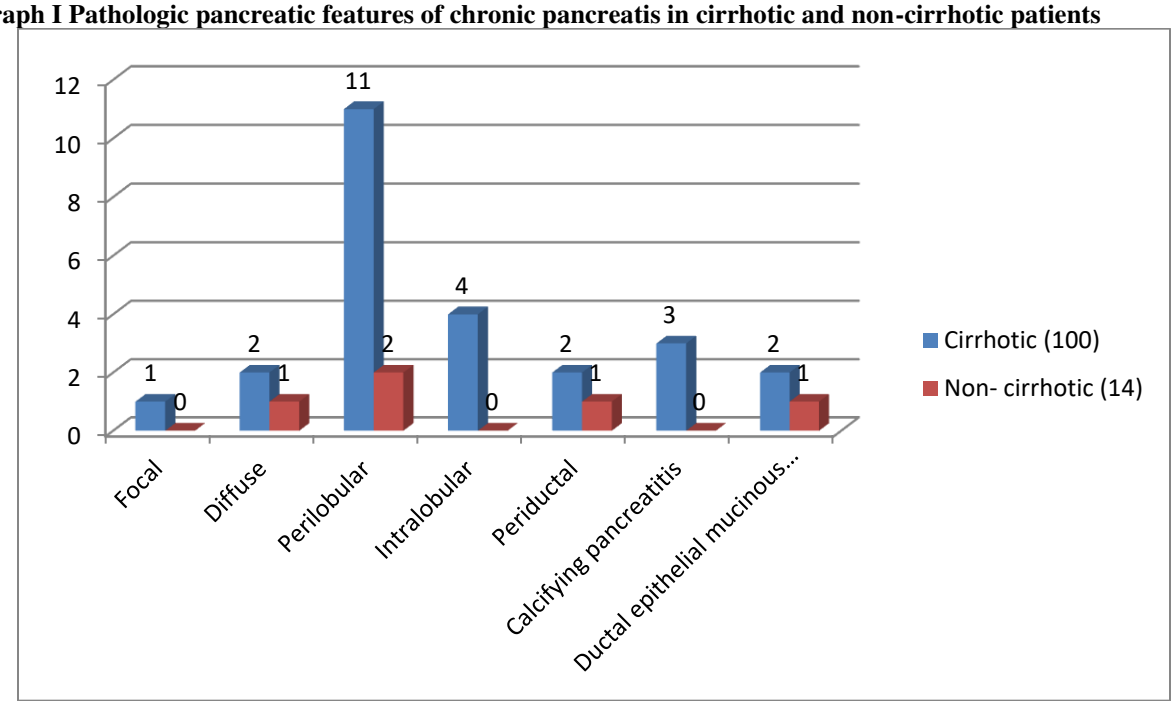
Table III Pathologic pancreatic features of chronic pancreatis in cirrhotic and non-cirrhotic patients

Distribution of fibrosis	Cirrhotic (100)	Non- cirrhotic (14)	P value
Focal	1	0	0.021
Diffuse	2	1	
Perilobular	11	2	
Intralobular	4	0	
Periductal	2	1	
Calcifying pancreatitis	3	0	
Ductal epithelial mucinous metaplasia	2	1	
Total	25	5	

Table III, graph I shows that distribution of pancreatic fibrosis found to be Focal in 1 and 0, diffuse in 2 and 1, perilobular in 11 and 2, intralobular in 4 and 0, periductal in 2 and 1, calcifying pancreatitis in 3 and 0, ductal

epithelial mucinous metaplasia in 2 and 1 in cirrhotic and non- cirrhotic in pathologic pancreatic features. The difference was significant (P< 0.05).

Graph I Pathologic pancreatic features of chronic pancreatis in cirrhotic and non-cirrhotic patients



DISCUSSION

Cigarette smoking might have an additive effect with alcohol in inducing pancreatitis. In the rat model of alcohol-induced pancreatitis, ethanol induces pancreatic ischemia while cigarette smoke potentiates the impairment of pancreatic capillary perfusion caused by ethanol.[6] Cigarette smoking accelerates progression of alcoholic chronic pancreatitis. A dietary component may also interact and modify effects of alcohol on the pancreas. A protein and fat rich diet along with continued consumption of alcohol exacerbate the course of chronic pancreatitis.[7] African Americans are affected more than Caucasians and this could be due to differences in diet, type or quantity of alcohol consumption. It can be due to differences in metabolism of alcohol in liver and pancreas. Alcohol consumption at intoxicating concentrations induces pancreatic cellular injury that may involve class III isoenzymes of ADH.[8] The present study was conducted to assess histomorphological features of pancreas and liver in chronic alcoholics.

In present study, out of 114 cases, 103 were seen in males and 11 in females. Gross features was enlarge in 73, shrunken in 30 and normal in 11 cases. Micronodules were seen in 64, macronodules in 36 and non- cirrhotic with normal liver in 14. Consistency was firm in 71, soft in 15, normal in 11 and necrosis was 28. Agrawal et al[9] in their study found that a total of 390 autopsies over 11 year's period were included in the study. Age ranged from 22 to 65 years with a mean age of 45.32 years. All 390 consecutive patients included in the study were males. Majority of the patients had primarily presented with alcohol related liver diseases whereas few had presented with features of pancreatitis. Micronodular cirrhosis was present in 292 cases. Features of chronic pancreatitis were observed in 42 cases and 8 of these cases had associated changes of acute hemorrhagic pancreatitis. Prevalence of pancreatitis was more in cirrhotics as compared to non-cirrhotics, and acute pancreatitis was mostly seen in non-cirrhotics. Dominant pattern of fibrosis was perilobular followed by periductal, intralobular and diffuse.

We found that distribution of pancreatic fibrosis found to be Focal in 1 and 0, diffuse in 2 and 1, perilobular in 11 and 2, intralobular in 4 and 0, periductal in 2 and 1, calcifying pancreatitis in 3 and 0, ductal epithelial mucinous metaplasia in 2 and 1 in cirrhotic and non-cirrhotic in pathologic pancreatic features. Cigarette smoking is a known risk factor for alcoholic and chronic pancreatitis. About 80%-95% of people who abuse alcohol also smoke while 25%-30% of smokers do not drink alcohol. The incidence of alcoholism is 10 times more likely in smokers than nonsmokers. Cigarette smoking accelerates the progression of alcohol induced pancreatitis.[10]

Despite the strong association between excessive alcohol consumption and development of chronic

pancreatitis, alcohol alone is not sufficient to lead to the disease, only a small proportion of chronic alcoholics (5-10%) develop chronic pancreatitis. Role of environmental factors, genetic profile or their interaction in determining susceptibility is still poorly understood. Amongst the environmental factors, smoking has been found to be independent factor associated with chronic pancreatitis. After all pancreatic disease is a complex disorder resulting from multiple defects, which, when combined, lead to failure of control systems and metabolic homeostasis. Understanding the roles played by certain genes, proteins, types of cells and pathways involved in interactions may provide better understanding of the pathogenesis of the disease.[11] Hayakawa et al[12] have proposed more than one theory in an attempt to explain the mechanisms responsible in the development of pancreatitis of alcoholic etiology. The first one based pancreatic histology showed ductal closures secondary to increased protein content in pancreatic juice, which led to obstruction, fibrosis, and calcification. Most patients already have some degree of parenchymal injury when presenting with the first acute crisis. The second theory refers to acinar cell injury secondary to direct toxic effect of ethanol which leads to inflammation which culminates in fibrosis.

The limitation of the study is small sample size.

CONCLUSION

Authors found that parenchymal fibrosis was more frequently seen in alcoholic cirrhosis cases than that in non-cirrhotic alcoholic liver disease.

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