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ORIGINAL ARTICLE

Analysis of Autopsies of Liver and Pancreas in Patients with Chronic Alcoholics

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***ABSTRACT*:**
Background: The frequency of co-existing alcoholic related pancreatitis (AP) and liver disease (ALD) is less well-studied and the reported estimate is just about 0.04 to 5%. The present study was performed to evaluate the effects of chronic alcoholism in liver and pancreas with the help of histopathology. **Materials & Methods:** It involved 460 medical autopsies. In all cases, autopsies were performed within the 2 hours of death. Multiple representative tissue blocks were taken from different areas of liver and pancreas. In addition to routine H and E staining done in all the sections, reticulin, Mason's trichrome and Elastic Van Giesen stainings were also carried out in selected liver and pancreatic sections according to indication. Any liver weight < 1200 gms was considered as having reduced in weight and any liver weight > 1300 gms was considered as having increased in weight. **Results:** Out of 460 autopsies, 320 were from males and 140 were from females. The difference was significant (P<0.05). Liver is enlarged in 345 cases, shrunken (92), normal (23). Cirrhotic in 345 cases which shows macronodules (200), macronodules (145) and non- cirrhotic (115). Non cirrhotic including norma liver was seen in 115 cases, fatty in 250 cases, cirrhotic (14) and normal in 20 cases. Consistency is firm (360), soft (40), normal (30) and necrosis (30). The difference was significant (P<0.05). Microscopic findings of liver such as micro and macrovesicular steatosis (51%), microvesicular steatosis (24%), ballooning degeneration (26%), Mallory hyaline (42%), necrosis (35%) and inflammatory portal duct in 28% cases. The difference was significant (P<0.05). Histopathological findings in pancreas such as parenchymal acute inflammation (7), parenchymal necrosis with hemorrhage (6), fat necrosis (12), vessel necrosis (4), peri and intra lobular necrosis (30), ductal ectasis (12), fibrin thrombus (5) and parenchymal calcification (3). The difference was significant (P<0.05).
Conclusion: Pancreatitis is not uncommon phenomenon in patients with alcoholic liver disease. Removal of alcohol resulted in reduction in pancreatic injury.
Key words: Alcoholic liver, Ductal ectasis Pancreatitis

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INTRODUCTION

Many investigators have established various relations between alcoholic related liver disease and pancreatitis. There is debate over the concerned matter and the clinical and pathological association between pancreatitis and alcohol abuse has been documented. The frequency of co-existing alcoholic related pancreatitis (AP) and liver disease (ALD) is less well-studied and the reported estimate is just about 0.04 to 5%.[1]

There is variation in opinion regarding progression of both events. There are two schools of thoughts with respect to the events involved in disease development both in liver and pancreas. Few suggest co-existence of chronic pancreatits and chronic alcohol related liver disease. Few say 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.[2,3]

Few authors 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. Recently, molecular study has referred to important role of genetic polymorphism in the pathobiology of pancreatic diseases.[4] Few authors from southern part of the country in their study had shown that alcoholic cirrhosis patient had significantly higher cumulative intake of alcohol and more number of years of intake of alcohol compared to alcoholic chronic pancreatitis. Thus one can say that there are considerable amount of damage of both liver and pancreas in alcoholic patients. The

confirmation of changes is well established by histopathological study which is the final diagnostic tool.[5] Considering this, the present study was performed to evaluate the effects of chronic alcoholism in liver and pancreas with the help of histopathology.

MATERIALS & METHODS

The present study was conducted in the department of general pathology. It involved 460 medical autopsies. Ethical clearance was obtained from the institutional ethical committee. First degree relative of the diseased in all cases were informed regarding the study and written consent in local language was taken.

All 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. Cases which showed pancreases showing focal fine intralobular fibrosis, and cases showing terminal peripancreatic fat necrosis with no inflammatory cell response were excluded from the study.

In all cases, autopsies were performed within the 2 hours of death. Post-mortem autolysis was differentiated from ante-mortem changes histologically. Multiple representative tissue blocks were taken from different areas of liver and pancreas. In addition to routine H and E staining done in all the sections, reticulin, Mason's trichrome and Elastic Van Giesen stainings were also carried out in selected liver and pancreatic sections according to indication.

Any liver weight < 1200 gms was considered as having reduced in weight and any liver weight > 1300 gms was considered as having increased in weight. Histopathology of the alcoholic liver disease included steatosis, steatohepatitis, sclerosing hyaline necrosis and cirrhosis.

Acute pancreatitis was characterized by necrosis of pancreatic parenchyma and surrounding fat, variable amount of hemorrhage and necrotizing inflammation. Changes in pancreas were documented as fibrosis which was graded subjectively as mild, moderate and severe depending on the extent of fibrosis at perilobular and intralobular parenchyma based on Kloppel and Maillet. Similarly, degree of chronic inflammatory cells was also graded subjectively as mild, moderate and heavy. Results were tabulated and subjected to statistical analysis. P value <0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 460		
Males	Females	P value
320	140	0.02

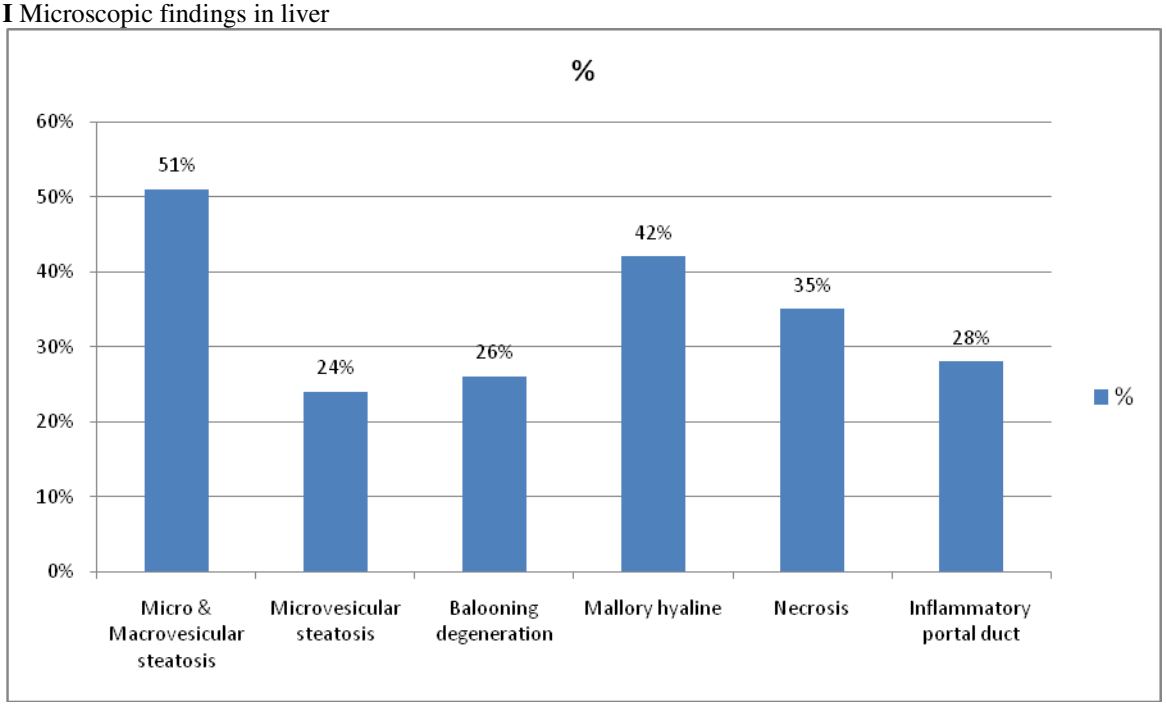
Table I shows that out of 460 autopsies, 320 were from males and 140 were from females. The difference was significant (P<0.05).

Table II Gross features observed in liver autopsy

Gross feature	Number	P value
Enlarge	345	
Shrunken	92	0.01
Normal	23	
Cirrhotic	345	
Micronodules	200	0.05
Macronodules	145	
Non cirrhotic including normal liver	115	
Fatty	250	
Cirrhotic	140	0.01
Non cirrhotic	50	
Normal	20	
Consistency		
Firm	360	
Soft	40	0.01
Normal	30	
Necrosis	30	

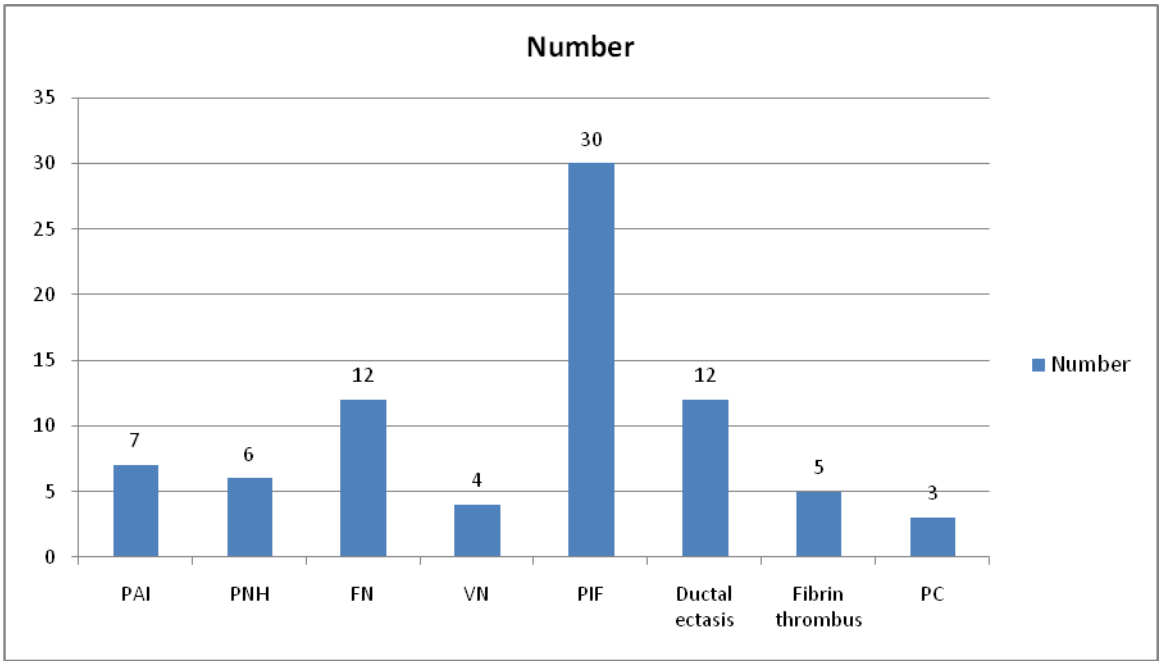
Table II shows that liver is enlarged in 345 cases, shrunken (92), normal (23). Cirrhotic in 345 cases which shows macronodules (200), macronodules (145) and non- cirrhotic (115). Non cirrhotic including norma liver was seen in 115 cases, fatty in 250 cases, cirrhotic (14) and normal in 20 cases. Consistency is firm (360), soft (40), normal (30) and necrosis (30). The difference was significant (P<0.05).

Graph I Microscopic findings in liver



Graph I shows microscopic findings of liver such as micro and macrovesicular steatosis (51%), microvesicular steatosis (24%), ballooning degeneration (26%), Mallory hyaline (42%), necrosis (35%) and inflammatory portal duct in 28% cases. The difference was significant (P<0.05).

Graph II Histopathological finding in pancreas



Graph II shows histopathological findings in pancreas such as parenchymal acute inflammation (7), parenchymal necrosis with hemorrhage (6), fat necrosis (12), vessel necrosis (4), peri and intra lobular necrosis (30), ductal ectasis (12), fibrin thrombus (5) and parenchymal calcification (3). The difference was significant (P<0.05).

DISCUSSION

In many studies the coexistence of liver diseases and pancreatitis has been explained. 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 develop chronic pancreatitis.[6] The present study was performed to evaluate the effects of chronic alcoholism in liver and pancreas with the help of histopathology.

In this study, out of 460 autopsies, 320 were from males and 140 were from females. We found that liver is enlarged (345), shrunken (92), normal (23). Cirrhotic in 75% cases which shows macronodules (200), macronodules (145) and non- cirrhotic in 20% cases. Non cirrhotic including norma liver was seen in 115 cases, fatty in 250 cases, cirrhotic (14) and normal in 20 cases. Consistency is firm, soft, normal and necrosis. This is in accordance to Sarles et al.[7]

Smoking has been found to be independent factor associated with chronic pancreatitis. This is considered to be the most common environmental factor. After all pancreatic disease is a complex disorder resulting from multiple defects, which, when combined, lead to failure of control systems and metabolic homeostasis. In this study we observed microscopic findings of liver such as micro and macrovesicular steatosis, microvesicular steatosis, ballooning degeneration, Mallory hyaline, necrosis and inflammatory portal duct. This is similar to Eghoje et al.[8]

In a study conducted by Clark E[9] 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. In this study we found that histopathological findings in pancreas were parenchymal acute inflammation, parenchymal necrosis with hemorrhage, fat necrosis, vessel necrosis, peri and intra lobular necrosis, ductal ectasis, fibrin thrombus and parenchymal calcification. This is in accordance to study by Yipp.[10]

Minics et al.[11], in their study have shown that in contrast to the persistence of established injury upon continuation of alcohol, withdrawal of alcohol resulted in a significant reduction in the indices of pancreatic injury in the form of edema, hemorrhage, acinar cell vacuolization and acinar necrosis.

CONCLUSION

Pancreatitis is not uncommon phenomenon in patients with alcoholic liver disease. Removal of alcohol resulted in reduction in pancreatic injury.

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