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ORIGINAL RESEARCH

Assessment of Autopsies of Liver in Patients with Chronic Alcoholics: A Cross Sectional Study

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Abstract

Background: Alcohol abuse and dependence are the major cause of morbidity and mortality in the United States. The present study assessed the effects of chronic alcoholism in liver with histopathology.

Materials & Methods: This cross sectional study was conducted on BBMCH, Balangir, Odisha taking 240 medical autopsies patients during the period of December-21 to August-22. 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.

Results: Out of 240 autopsies, 150 were male and 90 were of female. Consistency of liver was found firm, soft, normal and necrosis was 120, 60, 40 and 20 cases respectively. Liver was enlarged in 145 cases, shrunken in 60, normal in 30. Micronodules were seen in 130, macronodules in 90 and non- cirrhotic in 20. Liver was fatty in 110 cases, cirrhotic in 80 cases, cirrhotic in 80 and normal in 20 cases. The difference was significant ($P<0.05$). Microscopic findings of liver were micro and macrovesicular steatosis seen in 45%, microvesicular steatosis in 20%, ballooning degeneration in 28%, Mallory hyaline in 43%, necrosis in 32% and inflammatory portal duct in 27% cases. The difference was significant ($P<0.05$).

Conclusion: Microscopic findings of liver were micro and macrovesicular steatosis, ballooning degeneration, Mallory hyaline, necrosis and inflammatory portal duct.

Key words: Alcoholic liver, Macrovesicular Steatosis, Mallory hyaline

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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.[1]

Many studies have established various relations between alcoholic related liver disease and pancreatitis. There is debate 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%.[2] There is variation in opinion regarding progression of both events. Few suggest co-existence of chronic pancreatitis 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.[3,4]

Some 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.[5]The present study assessed the effects of chronic alcoholism in liver with histopathology.

Materials & methods

The present cross sectional study was conducted on BBMCH, Balangir, Odisha taking 240 medical autopsies patients during the period of December-21 to August-22. Study comprised of 240 medical autopsies of both gender. Family members of the diseased were informed regarding the study and written consent was taken.

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. 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. Results were tabulated and subjected to statistical analysis. P value <0.05 was considered significant.

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Results

Table-1 Distribution of patients

Total- 240		
Gender	**Male n(%)**	**Female n(%)**
Number	150(62.5%)	90(37.5%)

Table-1 shows that out of 240 autopsies, 150 were of males and 90 were of females.

Table- 2 Gross features in liver autopsy

Parameters	Variables	Number	P- value
Consistency	Firm	120	0.01
	Soft	60	
	Normal	40	
	Necrosis	20	
Size	Enlarge	145	
	Shrunken	65	0.01
	Normal	30	
Nodule	Micronodules	130	0.05
	Macronodules	90	
	Non cirrhotic including normal liver	20	
Fatty/cirrhotic	Fatty	110	
	Cirrhotic	80	0.01
	Non cirrhotic	30	
	Normal	20	

Table -2, graph I shows that consistency was firm 120, soft in 60, normal in 40 and necrosis in 20. Liver was enlarged in 145 cases, shrunken in 60, normal in 30. Micronodules were seen in 130, macronodules in 90 and non- cirrhotic in 20. Liver was fatty in 110 cases, cirrhotic in 80 and normal in 20 cases. The difference was significant (P<0.05).

Graph-1 Gross features in liver autopsy

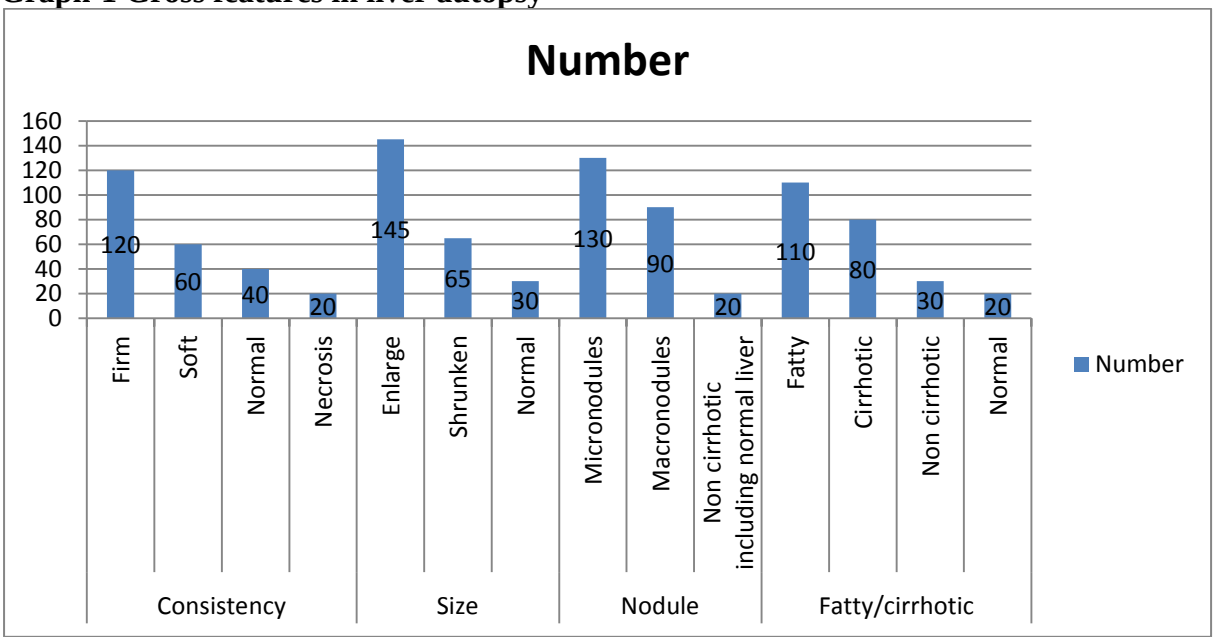

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Table 3 Microscopic findings of liver

Microscopic findings	%	P value
Micro & Macrovesicular steatosis	45%	0.82
Microvesicular steatosis	20%	
Balooning degeneration	28%	
Mallory hyaline	43%	
Necrosis	32%	
Inflammatory portal duct	27%	

Table -3 shows microscopic findings of liver were micro and macrovesicular steatosis seen in 45%, microvesicular steatosis in 20%, ballooning degeneration in 28%, Mallory hyaline in 43%, necrosis in 32% and inflammatory portal duct in 27% cases. The difference was significant ($P<0.05$).

Discussion

There is considerable amount of damage of both liver in alcoholic patients. The confirmation of changes is well established by histopathological study which is the final diagnostic tool.[6,7] In many studies the coexistence of liver diseases and pancreatitis has been explained.[8,9]The present study assessed the effects of chronic alcoholism in liver with histopathology.

We observed that out of 240 autopsies, 150 were of males and 90 were of females. Clark E[10] 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.

We observed that consistency was firm 120, soft in 60, normal in 40 and necrosis in 20. Liver was enlarged in 145 cases, shrunken in 60, normal in 30. Micronodules was seen in 130, macronodules in 90 and non- cirrhotic in 20. Liver was fatty in 110 cases, cirrhotic in 80 cases, cirrhotic in 80 and normal in 20 cases Similar study conducted by Awasthi NP11 found 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

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firm (360), soft (40), normal (30) and necrosis (30). The difference was significant at (P<0.05) equal to our study.

We found that microscopic findings of liver were micro and macrovesicular steatosis seen in 45%, microvesicular steatosis in 20%, ballooning degeneration in 28%, Mallory hyaline in 43%, necrosis in 32% and inflammatory portal duct in 27% cases. Similar study was also found by Umesh et al [12], Patel et al [13]and Selvi et al [14] to found the most common age group of 50-70 years and started with 40 years due to chronic consumption of alcohol in the population of Salem district, Tamil Nadu. In our study Steatohepatitis, steatosis and chronic hepatitis were the leading silent liver diseases compared to other studies. Underlying liver disease can be accurately estimated with the use of severalinvasive methods that spare the necessity of performing a liver biopsy. The widespread use of these methods can help to accurately identify patients at risk for the development of end-stage liver disease. In addition to treatment with direct-acting antivirals for those with HCV infection, abstinence from alcohol consumption should be strongly recommended, given the overlap between alcohol-related and metabolic liver diseases. In summary, asymptomatic fatty liver might be the most common silent liver disease among the general population of this region followed by cirrhosis of liver, hepatitis and chronic venous congestion.

Alcohol abuse generally leads to three pathologically distinct liver diseases viz. fatty liver, hepatitis and alcoholic cirrhosis. One or all of the three can occur at the same time and in the same patient. Fatty change (steatosis) is a very common finding both in biopsies and at post mortem examination. Liver cell involvement may be focal, diffuse, or zonal. Fatty liver develops within a short period (days) of alcohol abuse whereas more severe liver injury requires prolong alcohol abuse for a period of years. Nonalcoholic fatty liver disease (NAFLD) includes a spectrum of liver diseases, ranging from simple steatosis to steatohepatitis, advanced fibrosis and cirrhosis.

Conclusion

Underlying liver disease can be accurately estimated with micro and macrovesicular steatosis, ballooning degeneration, mallory hyaline, necrosis and inflammatory portal duct. The widespread use of these findings can help to accurately identify patients at risk for the development of end-stage liver disease

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