ORIGINAL RESEARCH PAPER

Pathology

INCIDENTAL HISTOPATHOLOGICAL FINDINGS OF LIVER - A STUDY OF 100 AUTOPSY CASES

KEY WORDS: Silent liver diseases, Autopsy, Steatosis, Cirrhosis.

Dr. Disha Rai* Postgraduate, M. S. Ramaiah Medical College, Bengaluru, Karnataka, India *Corresponding Author

Dr. Nandakishore Alva Professor, M.S. Ramaiah Medical College, Bengaluru, Karnataka, India

ABSTRACT

Introduction: The silent liver diseases are found accidentally during regular health check-ups, examination of other diseases or autopsy examinations. **Objectives:** To study the spectrum of histopathological findings of liver and to determine the prevalence of silent liver diseases in autopsy cases. **Material And Methods:** A retrospective study was done in the Department of Pathology, Ramaiah Medical College, Bengaluru from January 2019 to June 2020 on 100 liver autopsy specimens. **Results:** The majority of the cases with pathological lesions were in the age group of 31-60 years with Male: Female ratio of 2.8:1. The most common finding in this study was steatosis (31%), followed by chronic venous congestion (26%), cirrhosis (6%), portal triaditis (6%), steatohepatitis (5%), abscess and cholestasis (1%), tuberculosis (1%) and hepatocellular carcinoma (1%). **Conclusion:** The autopsy examination of liver by determining the prevalence of silent liver diseases in particular regional population creates public awareness and brings required life style changes.

INTRODUCTION:

Liver is known as 'the custodian of milieu interior' [1]. Liver performs many functions like excretion, synthesis, metabolism, storage and is at higher risk to toxic, metabolic, microbial, circulatory, and neoplastic insults. The clinical impact of mild liver damage is masked by the functional reserve of the liver. Liver disease is an insidious process. The symptoms and clinical detection of liver damage may occur weeks, months, years after the onset of injury which constitutes the silent liver diseases [2]. These silent liver diseases are found accidentally during regular health check-ups, examination of other diseases or autopsy examinations [1].

AIMS AND OBJECTIVES:

1) To study the spectrum of histopathological findings of liver in autopsy cases.
2) To determine the prevalence of silent liver diseases in autopsy cases in correlation with age and sex.

MATERIAL AND METHODS:

This was a retrospective cross sectional study conducted in Department of Pathology, Ramaiah Medical College, Bengaluru over a period of one and a half years from January 2019 to June 2020. A total of 100 liver autopsy specimens were studied. The liver specimens of medico legal cases were received either as a part of examination of multiple viscera or only liver was taken out for histopathological examination from the Department of forensic medicine. Specimens were received in 10% neutral buffer formalin. In each case details regarding age, sex, clinical findings, habits, cause of death were noted. Gross examinations of the specimens were done. External surface of specimen was examined. Specimens were cut open and sections from representative areas were submitted for processing. After processing paraffin embedded sections were cut and stained with Haematoxylin and Eosin, then examined under the microscope. Microscopically findings were recorded and analysed.

Inclusion Criteria:

- All cases from 2 to 80 years of age on whom medico legal autopsies were conducted to know the cause of death were included.

Exclusion Criteria:

- Markedly autolysed specimens were excluded.
- Autopsies of exhumed bodies were excluded.

RESULTS:

The present study included 100 cases in the age group of 2-80 years, out of which 75 were males and 25 were females.

On histopathological examination among 100 cases 77 liver specimens had pathological lesions, 16 had normal histology and 8 showed autolysis. Out of 75 male cases 57 liver specimens had pathological lesions, 13 had normal histology and 5 showed autolysis. Out of 25 female cases 20 liver specimens had pathological lesions, 3 had normal histology and 2 showed autolysis.

Table 1: Age and sex wise distribution of cases with pathological lesions (n=77)

Age (years)	Male	Female	Total	(%)
2-10	1	1	2	2.6
10-20	3	2	5	6.5
21-30	11	1	12	15.6
31-40	17	5	22	28.6
41-50	12	3	15	19.5
51-60	11	7	18	23.4
61-70	1	1	2	2.6
71-80	1	0	1	1.2
Total	57	20	77	100

Table 2: Distribution of cases according to cause of death (n=100)

Cause of death	Cases (%)
Road traffic accident	40
Poisoning	10
Snake bite	05
Drowning	06
Hanging	10
Fall	10
Assault	01
Pregnancy complication	01
Natural death (Cardiovascular, respiratory and central nervous system causes)	17
Total	100

Table 3: Distribution of cases in relation with habits (n=100)

History	Cases (%)
Alcohol + Smoking	18
Alcohol	21
Smoking	15
None	46
Total	100

Table 4: Gross findings in 100 cases

Cut surface	Cases (%)
Greasy	29
Nutmeg	15
Nodular	08
Greenish yellow	03
Cystic	01
Normal	44
Total	100

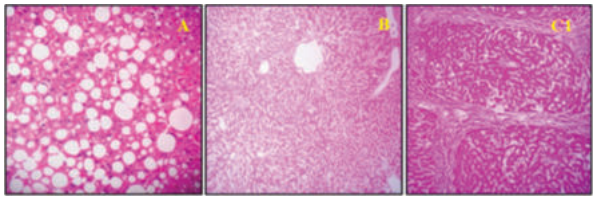
As per age and sex wise distribution of pathological lesions, the majority of cases were in the age group of 31-60 years constituting 71.5% with M: F ratio of 2.8:1 (Table 1). In the study the common causes for death were road traffic accident (40%), followed by natural death (17%) (Table 2). Distribution of cases in relation with habits (Table 3) depicted that 21% of cases were alcoholics, 15% were smokers, 18% were both alcoholics and smokers and 46% of cases did not have the history of smoking or alcoholism. On gross examination of cut surfaces, 29% of cases were greasy, 15% showed nutmeg appearance and 8% of cases showed nodules (Table 4).

Figure 1: A) Fatty change: Enlarged, yellow, greasy B) Cirrhosis: Nodular change (Diffuse nodularity) C) Tuberculosis: Firm tan nodules D) Hepatocellular carcinoma: Well circumscribed, tan yellow, dominant nodule with satellite nodule

Table 5: Sex wise distribution of microscopic findings in 100 cases

Findings	Male	Female	Total (%)
Fatty change (steatosis)	26	05	31
Chronic venous congestion	18	08	26
Normal histology	13	03	16
Autolysis	05	02	07
Cirrhosis	03	03	06
Portal triaditis	04	02	06
Steato hepatitis	03	02	05
Abscess + cholestasis	01	00	01
Granulomatous inflammation (Tuberculosis)	01	00	01
Malignancy (Hepatocellular carcinoma)	01	00	01
Total	75	25	100

On microscopic examination of 100 cases, the most common finding was steatosis (31%) followed by chronic venous congestion (26%), cirrhosis (6%), portal triaditis (6%), steatohepatitis (5%), abscess and cholestasis (1%), tuberculosis (1%) and hepatocellular carcinoma (1%). Also 16% of cases showed normal histology and 7% of cases showed autolysis.



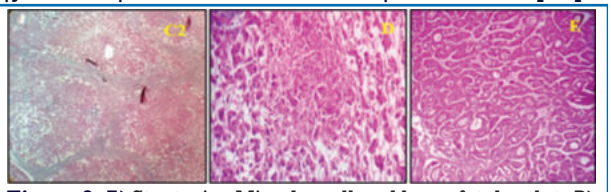


Figure 2: A) Steatosis - Mixed small and large fat droplets B) Chronic venous congestion: Sinusoidal congestion, dilatation and centrilobular distribution of necrosis C1) Cirrhosis:Thick band of collagen separating rounded cirrhotic nodules C2) Massson trichrome stain D) Tuberculosis: Caseating epithelioid granuloma E) Hepatocellular carcinoma-Malignant hepatocytes growing in distorted versions of normal architecture, including pseudo acinar spaces and thickened hepatocyte trabeculae.

DISCUSSION:

In the present study maximum number of cases were seen in the age group of 31-60 years (71.5%) which is similar to the study conducted by Poonam *et al* (64%) [3]. Liver disease was predominated in males in the present study (74%) comparable with the findings of Bhagat *et al* (76%) [1] and Bal. M. *et al* (83%) [4]. This adds to the fact that men are more inclined to consumption of alcohol.

Present study showed fatty change (31%) as the most common silent liver disease which was similar to studies of Poonam *et al* (34%) [3], Bhagat *et al* (32.5%) [1] and Bhavneet *et al* (34.2%) [5]. Cirrhosis was seen in 6% of cases comparable with Bhagat *et al* (7.5%) [1], Selvi *et al* (7.4%) [6]. Steatohepatitis was seen in 5% of cases comparable with Ekta *et al* (2.5%) [7] and Bhavneet *et al* (9.2%) [5]. Alcohol induced liver injury includes steatosis, alcoholic steato hepatitis, fibrosis and cirrhosis. The mild reversible hepatic steatosis is produced by the short term ingestion of 80 gram of alcohol. The daily intake of 80 gram or more of ethanol over years has significant risk of severe hepatic injury. Cirrhosis develops in only 10-15% of alcoholics. Excessive alcohol intake results in inflammation and hepatocyte death.

The microscopic changes in alcoholic liver disease begin in centrilobular zone 3 and extend to the portal tracts. In steatosis grossly liver is enlarged, soft, yellow and greasy. Microscopically there is lipid accumulation which initially forms small droplets later coalesce into large droplets and lead to macrovesicular steatosis. In alcoholic liver disease macrovesicular steatosis is the predominant form. The microscopic findings in alcoholic hepatitis are ballooned hepatocytes, perivenular / pericellular fibrosis, inflammation, and necrosis. Cirrhosis grossly shows parenchymal nodules converting the normal smooth liver capsule into a bumpy surface. Microscopically it shows thick bands of collagen separating regenerating cirrhotic nodules [2].

The presence of fatty liver in people who do not consume alcohol or do so in small quantities and who do not have any other cause of secondary hepatic fat accumulation is called as non-alcoholic fatty liver disease [2]. It is associated with all components of metabolic syndrome like obesity, Diabetes mellitus type 2, hyperlipidemia and insulin resistance. Non-alcoholic fatty liver disease with steato hepatitic injury is known as non-alcoholic steatohepatitis [NASH]. Microscopically non-alcoholic steatohepatitis shows steatosis (equal or more than 5% of hepatocytes), lobular inflammation, ballooned hepatocytes, spider web fibrosis around central vein progressing as periportal fibrosis, bridging fibrosis and cirrhosis. Cirrhosis thus developed is subclinical for years which constitute for silent liver disease [2].

Second most common finding in the study was chronic venous congestion (26%) which is similar to studies of Poonam *et al* (27%) [3] and Sameer *et al* (31.3%) [8]. The chronic venous congestion is the end stage of death due to preterminal

circulatory failure. Grossly centrilobular regions are depressed and red brown and are highlighted against the zones of uncongested tan liver and is called as nutmeg liver. Microscopically shows centrilobular congestion and haemorrhage, hemosiderin laden macrophages, hepatocyte dropout and necrosis [2].

Portal triaditis was seen in 6% of cases and comparable with Ratan *et al* (10.9%) [9]. Portal triaditis can be seen due to intake of drugs, alcoholic fatty liver disease or non- alcoholic fatty liver disease [9].

In the present study tuberculosis was seen in 1% of cases comparable with Bhagat *et al* [1] and Bhavneet *et al* (0.8%) [5]. Hepatic tuberculosis is noted in 50-80% of patients as a part of generalized miliary tuberculosis because of the rich blood supply of the liver [3].

Abscess was seen in 1% of cases as compared to Bal *et al* (2%) [4]. Liver and biliary tree can be involved by bacteria, fungi and parasites as localized infections or as part of a systemic disease. Biliary obstruction creates an environment for bacterial proliferation causing infection of the biliary tree and liver leading to abscess formation [2].

Hepatocellular carcinoma was seen 1% of cases comparable with Venuanand *et al* (0.9%) [10] and Bal *et al* (3%) [4]. Hepatocellular carcinoma is most commonly seen in the setting of chronic liver disease with cirrhosis. The disorders like chronic viral hepatitis, alcoholic liver disease, non-alcoholic fatty liver disease causes chronic injury, inflammation, hepatocyte regeneration contributing to the driver mutation (beta catenin, TERT, P53 mutation) which leads to the development of hepatocellular carcinoma [2]. Grossly it is well circumscribed mass with solitary and multiple satellite nodules, tan yellow to green in colour with areas of necrosis and haemorrhage. Microscopically there are trabecular, solid, pseudo glandular patterns with three cell wide hepatocyte plates. The tumour cells exhibits atypia, intranuclear pseudo inclusions, mallory hyaline bodies and hyaline globules. Tumour shows invasion of portal venules, increased arterialization and reduction of normal reticulin framework [2].

CONCLUSION:

The autopsy examination of liver in a specific regional population aids in determining the prevalence and etiology of silent liver diseases in that population. Thus creates public awareness regarding the same and helps in bringing essential life style changes to reduce the risk of silent liver diseases. Consequently contributing for a healthy population.

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Conflicts of interest: Nil

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