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SPECTURM OF SILENT LIVER DISEASES IN ROAD TRAFFIC ACCIDENT CASES – A 3 YEAR REVIEW

Pathology

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ABSTRACT

Background : Liver is the site of many diseases, some of which became symptomatic & have clinical manifestation while some remain silent and diagnosed only on autopsy. Histology is the unique method for diagnosis of liver diseases like acute & chronic hepatitis, steatosis, steatohepatitis, focal nodular hyperplasia, cirrhosis, cholestasis, hemangioma & hepatocellular carcinoma. The main histopathological alteration in liver tissue are steatosis followed by inflammation & fibrosis as studied in various centers.

Objective : To analyze the incidence of various liver diseases in road traffic accident cases, which gives us a data regarding incidence in general population of Orissa and histopathological study to diagnose steatosis, steatopatitis,cirrhosis, hepatocellular carcinoma etc. The study will also show the long term effects of various toxins, infection, enzymatic alteration and metabolic storage disorders on liver.

Results: histopathological findings show Portal fibrosis & inflammation occupies majority of cases, 35 cases out of 139 cases (25.18%).

- Fatty change/steatosis also occupies a majority of cases, 27 cases out of 139 cases (19.42%).

KEYWORDS

Silent Liver Disease, Road Traffic Accident, Autopsy.

INTRODUCTION

An autopsy is a medical procedure that consists of a thorough examination of a corpse to determine the cause of death and to evaluate any disease or injury that may be present. Most of the chronic liver diseases, even in advanced stages, may cause no prominent clinical signs or symptoms. They either go undiagnosed or are found incidentally during general health checkups, investigation for other diseases, surgery, or autopsy.

Quite rightly liver is, called as "The custodian of milieu interior" Autopsy study is useful to monitor the cause of death and to plan medical strategy[1]

The underlying cause of chronic liver diseases vary in different geographic areas and are based on various factors such as socioeconomic status, life style, diet, local or regional infections & other endemic diseases.[2] Abnormal findings in liver autopsy can be fatty change, hepar lobatum, glycogen storage diseases , acute phosphorus poisoning, hemosiderosis, syphilis, actinomycosis, infarcts, cloudy swelling, tuberculosis, acute passive hyperemia , amylodosis , abscess, hydatid cyst, malignancy, cirrhosis & acute yellow atrophy.

Alcohol abuse generally leads to 3 pathological distinct liver disease; fatty liver, hepatitis & alcoholic cirrhosis.[3]

In HIV patients also liver autopsy shows tubercular granuloma, extensive fatty change, sinusoidal congestion, focal necrosis, portal inflammation resembling chronic Hepatitis & kupffer cell hyperplasia[4]

In IV heroine addicts, the autopsy samples show degenerative vesicular & fatty changes, chronic Active & persistent hepatitis, cirrhosis, reduction in the amount of glycogen in hepatocytes as well as kupffer cell hypertrophy[5]

MATERIALS & METHODS

The study was carried out in the department of pathology & department of pathology VIMSAR Burla. This is a retrospective study on liver autopsy from September 2017 to October 2019.

The study includes the road traffic accident cases. After opening up of abdomen, liver is viewed for any abnormal gross findings. Then liver is resected into 2cm slices inside the abdomen to view the cut surface for any focal change of infarction, fibrosis, tumor, abscess, hemorrhage area, cholestasis etc.

Then a piece of liver from the abnormal site taken like a wedge biopsy. The liver piece is fixed in 10% buffered formalin in a small sealed plastic container & carried to the pathology department by proper labeling with postmortem no & date of postmortem.

In the pathology department the tissue is kept for 24hr for proper fixation & grossing is done on next day. Liver tissue is sliced into 4 to 5 micron thickness & 1section or multiple section given according to requirement. In automated tissue processor the tissue Kept in formalin for 9hrs .Then 3 changes of graded alcohol for dehydration (70, 80 & 90) for 1hr each. Then 2 changes of xylene for clearing – 1 hr each then embedding in paraffin – 3hr at 58*-60* C. After embedding tissue taken out & block is prepared. Tissue section are taken at 4 to 5 micrometer thick after trimming the block. Tissue sections are taken over albuminized slide & prepared for staining and Hematoxylin & eosin staining done.

Brunt grading system for non alcoholic steatohepatitis

Grade	Steatosis	Ballooning	Inflammation
Grade-1	1-2	Minimal	Lobular=1-2 Portal =0-1
Grade-2	2-3	Mild	Lobular=1-2 Portal=1-2
Grade-3	2-3	Marked	Lobular=3 Portal=1-2

Lobular Inflammation (0-3)	Portal inflammation (0-3)	Steatosis (1-3)
0 None	0 None	1 <33%
1 <2 foci/20x field	1 Mild	2 33%-66%
2 2-4/20xfield	2 Moderate	>66%
3 >4/20.field	3 Marked	

In chronic hepatitis for staging there is a histopathological activity index (HAI).

OBSERVATIONS

Histopathological findings

Finding	No. of cases	%
Portal fibrosis & inflammation	35	25.18
Fatty change/steatosis	27	19.42
Steatohepatitis	7	5.03
Sinusoidal dilatation & congestion	29	20.86
Hepatitis (both ac. & chronic)	1	0.72
Cirrhosis/bridging fibrosis	16	11.51
Cholestasis	3	2.16
Focal nodular hyperplasia	1	0.72
Hepatic adenoma	1	0.72
Hepatocellular ca	1	0.72
Autolytic change/ PM change	5	3.59
Normal	13	9.35

- Table 4 shows cases divided according to histopathological findings.
- Portal fibrosis & inflammation occupies majority of cases, 35 cases out of 139 cases (25.18%). Fatty change/steatosis also occupies a majority of cases, 27 cases out of 139 cases (19.42%).
- Grading of steatosis (n=26)

Grading	Percentage	No. of cases	%
Grade 1 (0)	< 5%	3	11.54
Grade 2 (1+)	5-33%	11	42.31
Grade 3 (2+)	33-66%	8	30.77
Grade 4 (3+)	>66%	4	15.38

- This table shows grading of all cases of steatosis. Out of the 26 cases grade 2 steatosis includes majority of cases (42.31%) Next to this is grade 3 steatosis which includes second majority (30.77%).
- Age & sex of cases of sinusoidal dilatation & congestion. Among the 29 cases 8 cases are within age range 21-30 while another 8 cases in age group 31-40. So all total 16 cases out of 29 cases (55.17%) are within age range 21-40.
- Out of 16 cases 7 cases (43.75%) are from age group 31-40 are cases of cirrhosis/bridging fibrosis.
- Females are most commonly affected in age range 31-40 (8.57%) of portal fibrosis & inflammation cases. Most common age group affected are within 21-30 (22.85%)
- Males are most commonly affected in fatty change of liver as the data shows , 21 cases out of 27 cases (77.77%).10 cases out of 26 cases (37.03%) are within age group 31-40yr.

Discussion:

This study on silent liver disease in RTA cases in Odisha an autopsy study showed that steatosis is the most common silent liver disease. Portal fibrosis & inflammation also occupies majority of cases.

Inflammatory disorders including NASH comprise a minority of cases. The true incidence & prevalence of NASH & steatosis, periportal fibrosis, precirrhotic stage are not well known in different populations of Odisha. This is partly because liver biopsy is required as a gold standard for diagnosing these condition. Because of the relatively invasive procedure & having high risk complications, clinicians avoid liver biopsy for management of these silent liver disorder.

Most of the reports about the prevalence rates of this disease are based on ultrasonographic studies & elevated liver enzymes.

The prevalence of NAFLD in patients undergoing liver biopsy for any reason ranges between 15% & 39%. This wide range is naturally related to the difference in the population studied.

In our autopsy study the range is between 1.8% to 5.07%. In clinical practice, diagnostic liver biopsy is only performed for highly selected patients. Therefore the reported rates which are based on liver biopsies cannot reflect the true prevalence of NAFLD/NASH in general population. Autopsies performed for those who have passed away for reasons other than liver disease like in random samples in case of RTA are a better source for determination of prevalence of NAFLD.

In our study fatty liver disease/steatosis occupies 18.7% of cases. In general population clinician do not advice for liver biopsy as USG can have some diagnostic value. But the general incidence & prevalence could not be estimated by these procedure. So autopsy study gives us idea about the incidence rates which help clinicians for the study purpose & clinical trials and find out the causes & prevention of the of the disease process. Modification of life style & change in diet is most essential. In odisha most people take staple foods like rice & dal in heavy amount. Workload is not so high. They prefer to a sedentary life. Few people do exercise like morning walk or heavy exercises, so they can bornout fat. But most people unwilling to do heavy work and exercise with diet rich in cholesterol and fat suffer from fatty liver disease. Fat is oxidized in liver but excess fat (>5% of liver wt.) leads to accumulation of lipids within hepatocytes.

Potential pathophysiological mechanism include enhanced delivery of fatty acids to the liver from adipose tissue or diet, increased endogenous fatty acid synthesis, decreased mitochondrial fatty acid oxidation and deficient incorporation or export of triglycerides in the form of very low density lipoprotein.

Among 139 cases enrolled in 2 years study period between Sep 2017 to Oct 2019, which is a random study, includes RTA cases and there is more number of males are in the age group between 30-50(52 cases.)

As our study is on silent liver diseases, and the most common toxic substance affecting liver architecture is alcohol, history of alcoholism is taken in all of the cases. As per history taken from close relatives alcoholics are 33 cases from 139 cases.

In our study fatty change is around 19.42% as in sameer et al around 20%.(7)

But the study by M.S. bal et al included cases of RTA, along with drowning, burn and poisoning. Poisoning induces fatty change so steatosis cases are increased.

According to study by Rasoul Sotodemanesh et al(2) fatty change/ steatosis 31.6%, chronic hepatitis 2.6%, steatohepatitis 2.1%, cirrhosis 0.8%, focal nodular hyperplasia 0.2% and hepatocellular carcinoma 0.1%. In comparision to that our study includes fatty change 19.42%, cirrhosis/bridging fibrosis 11.51%,steatohepatitis 5.03%, , chronic hepatitis 0.72% focal nodular hyperplasia 0.72% and hepatocellular carcinoma 0.72%.

In our study group most common age group affected are 31-40 (37.03%). Second most common age group 21-30 (22.22%). Mostly young people are affected because of excess intake of fat in diet, hectic lifestyle and alcohol consumption. M.S. Bal et al , Fubara DS et al and patel et al also found that fatty change is most prevalent in age group i.e. 53.85% , 28% and 31% respectively.(3,6,7)

Cirrhosis or bridging fibrosis is more common in age group 31-40, 7 cases out of 16 cases (43.75%) and study by SS Oberoi and SP Singh also revealed cirrhosis more common in age group 41-50, 6 cases out of 14 cases (42.85%).

Sinusoidal dilatation and congestion more common in age group 21-40 around 16 cases out of 29 cases (55.17%), as compared to study by M.S. bal in age group 41-50 around 7 cases from 9 cases (77.8%). Copeland (1985) reported congestion with fatty change in 3.4% of liver autopsies of alcoholics who died suddenly.(9)

Cholestasis more common in females 2 out of 3 cases around 66.66%. No comparative study is available.

Portal fibrosis and inflammation included 35 cases out of 139 cases (25.18%) no cases till date.

Grading of steatosis based on percentage of affection of hepatocytes. Most of the cases in our study are of grade 2, 11 cases out of 26 cases (42.31%). As compare to study by Seyed Mohammad Tavangar grade 1 change is more common 196 out of 283 cases (64.8%).

Our study in Odisha shows a good number of fatty liver cases (19.42%) & Oriya males have a greater predilection for fatty liver (66.66%) than female (33.33%). So males of Odisha should try to avoid excess alcohol intake and sedentary life style.

Since the metabolic syndrome and insulin resistance are known to be associated with coronary artery disease and cardiovascular morbidity and mortality, if fatty liver constitutes an independent marker for metabolic syndrome, then we have a gloomy forecast for the large number of individuals with fatty liver, who would not only be at risk of developing cirrhosis, terminal liver failure, and hepatocellular carcinoma, but would also carry an increased risk of cardiovascular morbidity and mortality.

There is no established treatment for NAFLD/NASH. Most patients improve by moderate weight loss, achieved by diet and/or exercise. A large body of evidence indicates that insulin resistance may be the primary reason for fat accumulation in the liver, hence the rationale for treating patients with insulin sensitizing agents.

Conclusion:

Even though autopsy & histopathological study of liver is the best method to determine the undiagnosed liver diseases, there has been a decline in autopsy rate recently.

To improve autopsy rate, clinicians & pathologist should have a active

role & encourage family to give consent for autopsy in organ failure cases.

By providing informative preliminary & final autopsy reports pathologists could convey their message to clinicians regarding the liver damage by various exogenous factors which remain undiagnosed but could be prevented.

Better support should be provided from administration of institution by allocating adequate resources, improving administrative requirements and encouraging greater pathologist involvement.

Also community awareness about the value of liver autopsies should be enhanced by information about the histopathological finding & its significance.

Alcoholic, obesity, DM, HTN are the major causes of steatosis along with lack of exercise & sedentary habits leads to all the disease states.

So public should be informed & they should modify their life style & avoid excess alcohol intake to prevent the pathological changes in liver.

So to conclude my topic this study of silent liver disease in RTA cases in Odisha gives us information regarding:

a) Incidence and prevalence of silent liver disease in general population of Odisha & its etio-pathogenesis.
b) By knowing the etio-pathogenesis, community awareness could be done to prevent the disease process & clinicians could inform patients regarding the hazards of alcoholic intake, injudicious use of drugs & dietary modifications.

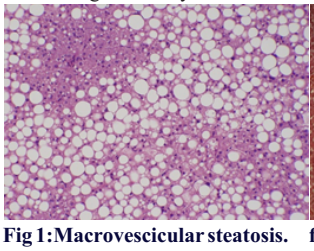


Fig 1:Macrovescicular steatosis.

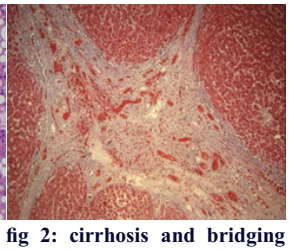


fig 2: cirrhosis and bridging fibrosis.

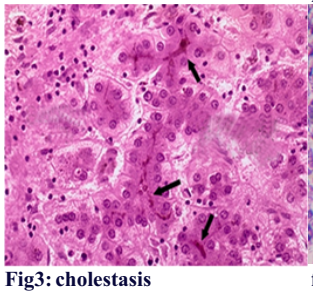


Fig3: cholestasis

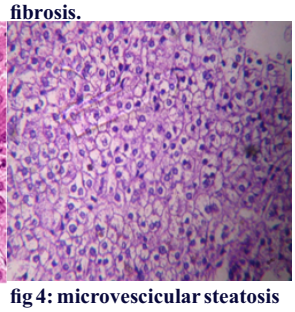


fig 4: microvescicular steatosis

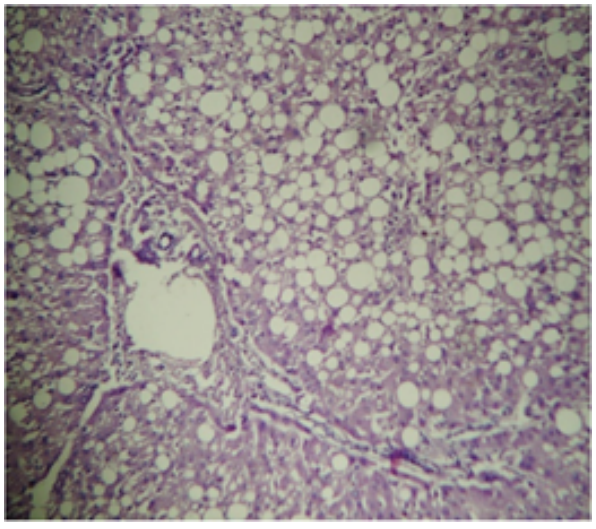
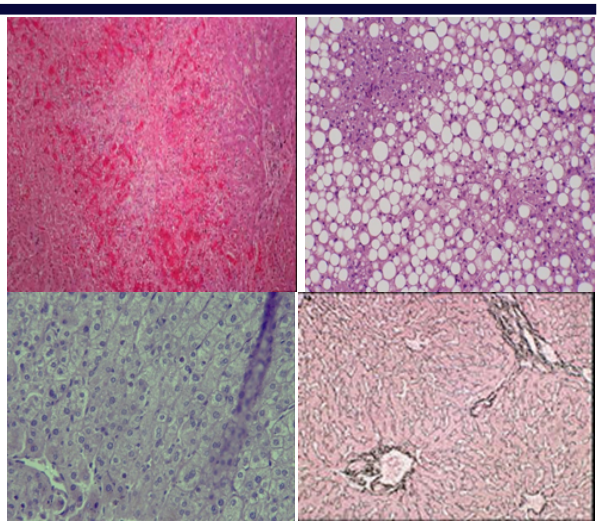


Fig 5: portal inflammation(100x)



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