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PREVALENCE OF RISK FACTORS ASSOSCIATED WITH LIVER DISORDERS IN A TERITIARY CARE HOSPITAL: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Aim: To compare the Prevalence of liver disorders and risk factors in a representative sample of people living in Karimnagar region. **Methods:** The current study is Prospective Observational Study conducted over a period of six months at Chalmeda Anandarao Institute Of Medical Sciences, Abhinav Gastro and Liver Centre, SRR Gastro and Liver Centre. The focus of this study is on the risk factors associated with Liver Disorders. Data was collected from 236 subjects with Liver Disorders through structured questionnaire. Results were analysed using Microsoft Excel 2010. **Results:** Results revealed that in our study of Liver Disorders highest impact was seen in Males of age group 41-60 with (35.16%) of frequency and infrequent in young age group 12-20 with (0.84%). Out of 236 cases 152 were attributed to Fatty Liver, 25 to Hepatitis, 23 to Jaundice, 16 to Hepatomegaly, 14 to ALD and 6 to Liver Abscess. The major risk factor observed was consumption of Alcohol (65.25%), Exposure to Toxins (11.01%), Consumption of Contaminated Food (10.16%), Viral Infections (6.77%) Blood transfusions (3.38%), Drug Abuse (3.38%). **Conclusion:** Alcohol is the major risk factor of Liver Disorder that was observed in our study period in Karimnagar region**.**

KEYWORDS: Prevalence, Liver disorders, Observational study, Alcohol consumption.

INTRODUCTION

The liver is the largest organ, accounting for approximately 2% to 3% of average body weight.It is protected by the rib cage and maintains its position through peritoneal reflections, referred to as ligamentous attachments.

FUNCTIONS OF THE LIVER

•Production of bile, which helps carry away waste and break down fats in the small intestine during digestion and also certain proteins for blood plasma, and cholesterol, special proteins to help carry fats through the body.

•Conversion of excess glucose into glycogen for storage (glycogen can later be converted back to glucose for energy) and to balance and make glucose as needed.

•Regulation of blood levels of amino acids, which form the building blocks of proteins.

•Processing of hemoglobin for use of its iron content (the liver stores iron).

•Conversion of poisonous ammonia to urea & clearing the blood of drugs and other poisonous substances.

•Regulating blood clotting & resisting infections by making immune factors and removing bacteria from the blood stream.[1]

CLASSIFICATION OF LIVER DISEASES

I. Hepatitis - This is the name for any condition involving inflammation of your liver. There Sometimes, excessive alcohol use, drugs or toxins cause hepatitis.

A). Acute: - Acute viral hepatitis, type A,B,C.

- Hepatitis associated with systemic viral infection.
- Infectious mononucleosis hepatitis, Cytomegalic virus hepatitis.
- Alcoholic hepatitis, Drug-induced hepatitis, Chemical hepatitis.

B). Chronic: - Chronic active hepatitis & Chronic persistent hepatitis.

II). Alcoholic liver disease: Alcoholic liver disease is a result of alcohol abuse.

Those who continue to consume alcohol excessively may cause injury to their liver.

III). Cholestasis: This happens when the flow of bile from your liver is limited or blocked. Cholestasis can be caused by certain drugs, genetic factors or even pregnancy. It can also happen from a blockage caused by a tumor or a gallstone stuck in the body's digestive system.

IV).Cirrhosis: This is a hardening of your liver due to scar tissue. Heavy alcohol use and viruses like hepatitis are common causes of cirrhosis. Diabetes, immune problems and genetic diseases can also cause the disease.

V). **Non alcoholic liver disease:** This happens when there are fat deposits in the liver. The deposits prevent your liver from functioning properly and removing toxins from your body.

VI). Alcoholic Liver Disease: Alcoholic liver disease is liver injury that is due to alcohol abuse. However, those who consume more than the daily "threshold" level of alcohol will have some evidence of liver injury. Daily ethanol consumption exceeding 40–80 g/day for males and 20–40 g/day for females for 10–12 years will almost certainly lead to ALD.

VII). CIRRHOSIS: Although "cirrhosis" is the most commonly used term to describe liver damage, there are actually three stages of liver damage:

- **Fatty liver** is an abnormal accumulation of fat in the liver. This can cause your liver to become enlarged. It is usually asymptomatic and is completely reversible once you stop drinking alcohol.
- **Alcoholic hepatitis** occurs when your liver becomes inflamed, destroying liver cells, commonly patients experienced abdominal pain, jaundice and feeling very sick.
- **Cirrhosis (scar formation)** occurs when the normal liver tissue is destroyed and replaced with scar tissue. Blood flow through your liver becomes difficult and fluid accumulates in the abdominal cavity. This could lead to liver failure and liver cancer.[2]

RISK FACTORS

1. **Gender -** Much more common in males than in females.
2. **Chronic viral hepatitis (Hep-B or Hep-C) -** The most common risk factor for liver cancer is chronic (long-term) infection with hepatitis B virus (HBV) or hepatitis C virus (HCV). These infections lead to cirrhosis of the liver.
3. **Cirrhosis -** Most cases occur in people who abuse alcohol or have chronic HBV or HCV infections.
4. **Non-alcoholic fatty liver disease -** A condition in which people who consume little or no alcohol develop a fatty liver, is common in obese people. People with a type of this disease known as non-alcoholic steatohepatitis (NASH) might go on to develop cirrhosis.
5. **Primary biliary cirrhosis -** In Primary biliary cirrhosis (PBC), the bile ducts in the liver are damaged and even destroyed which can lead to cirrhosis. People with advanced PBC have a high risk of liver cancer.
6. **Inherited metabolic diseases-** Certain inherited metabolic diseases can lead to cirrhosis. People with hereditary hemochromatosis absorb too much iron from their food. If enough iron builds up in the liver, it can lead to cirrhosis and liver cancer.
7. **Heavy alcohol use -** Alcohol abuse is a leading cause of cirrhosis, which in turn is linked with an increased risk of liver cancer. Individuals consuming excess alcohol can develop alcoholic liver disease (ALD).
8. **Obesity**- Being obese (very overweight) increases the risk of developing liver cancer. This is probably because it can result in fatty liver disease and cirrhosis.
9. **Type 2 diabetes -** Type 2 diabetes has been linked with an increased risk of liver cancer, usually in patients who also have other risk factors such as heavy alcohol use and/or chronic viral hepatitis.
10. **Certain rare diseases:** Diseases that increase the risk of liver cancer include:
    - Tyrosinemia, Alpha1-antitrypsin deficiency, Porphyria cutanea tarda, Glycogen storage diseases, Wilson disease.
11. **Vinyl chloride and thorium dioxide (Thorotrast)-** Exposure to these chemicals raises the risk of angiosarcoma of the liver. It also increases the risk of developing cholangio carcinoma and hepatocellular cancer, but to a far lesser degree.
12. **Anabolic steroids -** Long-term anabolic steroid use can slightly increase the risk of hepatocellular cancer. Cortisone-like steroids, such as hydrocortisone, prednisone, and dexamethasone, do not carry this same risk.
13. **Arsenic** - Drinking water contaminated with naturally occurring arsenic, such as that from some wells, over a long period of time increases the risk of some types of liver cancer.
14. **Tobacco use -** Smoking increases the risk of liver cancer. Former smokers have a lower risk than current smokers, but both groups have a higher risk than those who never smoked.
15. **Hepatotoxic Drugs:**Erythromycin estolate, Pyrazinamide, Tetracyclines, Nalidixic acid,Pefloxacin, Acetaminophen.[3]

SIGNS AND SYMPTOMS

I. NON-SPECIFIC SIGNS AND SYMPTOMS - Asthenia, Anorexia, Weight loss, Nausea are often present in patients with Hepato-cellular Carcinoma(HCC).

II. CLINICAL ASPECTS

1. Cirrhosis - HCC usually present at an advanced stage of the disease with clinical signs as - Jaundice, Ascites, Peripheral oedemas, Neurologic manifestations of hepatic encephalopathy, bleeding, or infections. Other signs of hepatic cirrhosis include - Gynecomastia, Palmar erythema, Spider angiomas, Axillary or chest hair loss, Hypogonadism (testicular atrophy, loss of libido).

2. Hepatomegaly - Hepatomegaly can be an expression of the tumor mass & is more often present in patients without advanced cirrhosis..

3. Abdominal pain, portal vein thrombosis, rupture of HCC

- Other clinical manifestations of portal vein thrombosis and/or portal hypertension are - Hematemesis from rupture of esophageal varices, Nausea,Vomiting, Anorexia & weight loss
Diarrhea, Splenomegaly.

4. Gastrointestinal bleeding - A Variceal bleeding can present with melena or hematemesis.
5. Jaundice - Clinical manifestation are those of typical cholestatic syndrome. In these cases jaundice is usually accompanied by - Itchiness, caused by elevation of serum level of bile acids, Hypocolic stool and dark urine.
6. Fever - It can be intermittent, and usually is accompanied by leukocytosis.
7. **Caval invasion -** In this case relevant pitting edema can appear, usually bilaterally, affecting both inferior limbs, from the inguinal region. The invasion of the venous district, can worsen ascites and hepatomegaly.
8. Extrahepatic metastases - Lung metastases (dyspnea, cough, hemoptysis, chest pain, Fatal respiratory failure), Brain metastases (Paralysis), Peritoneal metastases (ascites and abdominal pain).
9. Paraneoplastic syndromes - Hypercholesterolemia, Erythrocytosis, Hypoglycemia, Hypercalcemia, Thrombocytosis.
10. Arterial hypertension - Overproduction of angiotensinogen and renin could both play a role in causing paraneoplastic hypertension.
11. Diarrhea - Overproduction of intestinal peptides as gastrin and vasoactive intestinal peptide (VIP) is one possible explanation of the onset of diarrhea in these patients.
12. Cutaneous manifestation – Dermatomyositis, Polymyositis, Rhabdomyolysis, Porphyria cutanea tarda, Pityriasis rotunda.
13. Neurological manifestations - Multifocal necrotizing leukoencephalopathy, Peripheral polyneuropathy.
14. Other paraneoplastic syndromes - Dysfibrinogennemia, Cryofibrinogenemia, Carcinoid syndrome, Myasthenia gravis, Membranous glomerulonephritis.[4]

DIAGNOSIS

1. ANALYTES TO EVALUATE LIVER FUNCTION INCLUDES -

- Alanine aminotransferase (ALT) (serum glutamate pyruvate transaminase [SGPT]}
- Alkaline phosphatatase (ALP), Ammonia
- Aspartate ammotransferase (AST) (serum glutamate oxaloacetate transaminase [SGOT]}
- Total bilirubin, Unconjugated (indirect), Conjugated (direct)
- Glutamyl transferase (GGT), Lactate dehydrogenase (LD), 5' Nucleotidase (5' NT)
- Pseudocholinesterase, Ceruloplasmin, Alpha feto protein, Bile acids
- Ornithme carbamoyltransferase, Carbohydrate deficient transferrin, Fatty acid ethyl esters.

In general, only four serum biochemical tests are necessary to assess hepatic abnormalities (1). These analytes are – ALT, AST, ALP, Prealbumin or Prothrombin.

2. NON INVASIVE TOOLS FOR THE DIAGNOSIS OF LIVER CIRRHOSIS

- **Serum markers**

a). Direct serum markers – Hyaluronan, Procollagen Type Ⅰ & III Carboxy-amino terminal peptide.
- Metalloproteinases (MMP) - MMP-2 (gelatinase-A), MMP-3 (stromelysin) and MMP-9 (gelatinase-B).
- Tissue inhibitors of matrix metalloproteinases, Laminin
- Transforming growth factor-beta1 (TGF-β1)
- Connective tissue growth factor (CTGF), also known as CCN2
- YKL-40, also called human cartilage glycoprotein-39 (HC gp-39)
- Microfibril-associated glycoprotein 4, Cytokeratin-18 fragments.

b). Indirect serum markers: AST/ALT ratio.

3. IMAGING INSTRUMENTS

- Ultrasound (US), Doppler US, Contrast-enhanced ultrasonography
- Elastography, Strain imaging,- Shear wave technique, TE (Fibro scan)
- Acoustic radiation force impulse, ARFI and transaminases
- Magnetic resonance elastography.[5]

TREATMENT

I). HEPATITIS

A. DRUGS

1. Pegylated interferon alpha: Consider in young, non cirrhotic pts. With low viral load and high ALT. Dose: 180 microgram/wk/ s/c for 48wks.
2. Nucleoside analogues: High antiviral potency; negligible adverse effects, oral administration, safe and effective for all ages, suitable for cirrhotic and HIV coinfected pts.
i. Lamivudine: 100mg/day; high rate of resistance. (specific point mutation YMDD motify of the HBV polymerase).
ii. Adefovir dipivoxil: 10mg/day; potentially nephrotoxic.
iii. Entecavir: 0.5-1mg/day; low rate of resistance.
iv. Tenfovir: 300mg/day; low rate of resistance.
v. Others: Emtricitabine, Clevudine.

B. PREVENTION

The patient should avoid salivary transmission to others by avoiding kissing, spitting, sharing food, cigarettes or utensils and sexual contact. Infected stools and urine in Hepatitis A and E infections must be disposed of carefully.

1. **Immunization**

a. **Hepatitis A:** All Immune globulin (IG) Preparations contain ant-HAV. It should be given within 2weeks of contact in the dose of 0.02ml/kg. For travelers a dose 0.06ml/kg should be given every 6months.
b. **Hepatitis B:** A vaccine for active immunization has been prepared from HBsAg carrier serum as well as by genetic engineering from recombinant yeast.

For pre- exposure prophylaxis, Hepatitis B vaccine must be given at 0, 1 and 6-month interval. The dose is 20mcg I.M for immuno-compressed adults and 10mcg I.M for infants and children under 10years**.**

For post-exposure cases, a combination of HBIG(for rapid achievement of high circulating litre of anti-HBs) and hepatitis B vaccine (for long lasting immunity) is given.

For perinatal exposure of infants, of HBsAg positive mothers, HBIG 0.5ml should be given in thigh at birth followed by Hepatitis B vaccine 10mcg started with a week of birth and repeated at 1 and 6 months**.**

For percutaneous exposure a single dose of 0.06ml/kg of HBIG is given followed by three doses of hepatitis B vaccine.

c. Hepatitis D: There is no immune prophylaxis to prevent delta super infection. Susceptible persons are vaccinated with Hepatitis B vaccine**.**

d. Hepatitis C and E: IG prophylaxis in this hepatitis is not known to be beneficial and is not recommended**.**

II). PORTAL HYPERTENSION

A. TREATMENT

1. Treatment of Variceal bleeding

a).Local measures – Sclerotherapy, Banding, Balloon tamponade, Shunt surgery.

b).**Drugs**: Splanchnic flow can be reduced by vasopressin, somatostatin, and terlipressin.

-Vasopressin: It is given as an IV infusion, 0.4units/min until bleeding sops or for 24hrs and then 0.2units /min for a further 24hrs. It can cause angina, MI, arrhythmias. ECG monitoring is necessary.

- Somatostatin and Octreotide: Synthetic form of somatostatin is given in the dose of 50mcg IV followed by an infusion of 50mcg hourly. It is expensive but the drug of choice.

- Terlipressin: It is given in the dose of 2mg IV every 6hrs until bleeding stops and then 1mg IV for further 24hrs.

c). Prophylaxis: In patients with variceal bleed, recurrent bleeding is very common. This could be prevented by regular sclerotherapy, banding and drugs like beta- blockers or nitrates. Propranolol 80-160mg/day reduces portal venous pressure and can be used to prevent recurrent variceal bleed**.**

d). TIPS (Transjugular Intrahepatic Portal Shunt) used for patients awaiting for liver transplant.

e). Liver Transplant.

2.Treatment of Enlarged Spleen: Massive splenomegaly, hypersplenism and splenic infarction may require splenectomy and shunt surgery.

3. Treatment of Ascites: Salt restricted diet with diuretics spironolactone and of furosemide. Massive ascites require therapeutic tapping**.**

III). CIRRHOSIS OF LIVER

TREATMENT

1. Bed rest till improvement. Diet: 2000calories with about 100gm proteins. Fats and carbohydrates as much as the patient tolerates. Salt is restricted if edema or ascites is present. Supplemental Vitamin B complex should be given.
2. Diuretics: Spironolactone is an aldosterone antagonizes the effects of excess aldosterone present in cirrhotics due to inadequate elimination of aldosterone by liver. 100mg/day may be given. Maximum dose 400mg. Furosemide can also be added up to 80mg.
3. Removal of cause: Withdrawal of alcohol, D-penicillamine for Wilson's disease, etc.
4. Corticosteroids and immunosuppressants may be helpful in active post-hepatitis cirrhosis.
5. Antifibrotic agents like colchicine and propylthiouracil are still experimental.
6. Interferons utility is limited once cirrhosis sets in. It is useful only if there is actively replicating virus B or C and patient is awaiting transplant.
7. TIPS (Transjugular Intrahepatic Porto-systemic Shunts).
8. Artificial Liver support.
9. Hepatocyte transplant and orthoptic liver transplant.[6]

METHODOLOGY

Study Site: Chalmeda Anandarao Institute Of Medical Sciences, Karimnagar. Abhinav Gastro and Liver Center, SRR Gastro and Liver Center Karimnagar.

Study Design: A Prospective Observational Study.

Study Period: This study was carried out for a period of 6 months.

Study Criteria

a) Inclusion criteria: Participating patients that must have been diagnosed with Liver Disorders with the age of above 11years, and are presented with any or all of the following symptoms such as abdominal pain, Vomitings, icterus, ascites, edema.

b) Exclusion criteria: Participants with below 12years of age and are not diagnosed with any liver disorders.

Source of Data - Patient prescriptions and medical records were studied to obtain demographic details. Other information was asked verbally which includes occupation, locality of the subject, histories of medical and medication (name, dose, frequency, route), family history, allergies, and any blood transfusions, amount of alcohol intake (occasional/regular) and any previous histories of alcohol Intake(occasional/regular). We also included the data that whether the subject was exposed to any contaminated Food, water, toxins and synthetic chemicals and pesticides**.**

- Data from USG, CT-Abdomen were taken.

Ethical Committee Approval - The protocol of the study including the introduction, objectives, data collection form and methodology was submitted for approval of ethical committee members, the study was approved by Institutional Ethical Committee of Vaageswari College Of Pharmacy.

Study Procedure - All the patients who are diagnosed with LIVER DISORDERS.

- **Patient:** Data that can be collected includes demographic details (age, gender, and occupation), weight, height, social history (Alcohol consumption), past medical history, causes & pre-disposing factors, diagnostic reports (USG,CT), Non-pharmacological, Pharmacological, Life-style modifications.

Data Collection Form - Appropriate data collection form was designed to collect, document, analyse the data. Data collection form include the provision for collection of information related to all study parameters mentioned above like demographic details of patient (name, age, sex,), social history, Comorbid conditions like Hypertension, Diabetes- Mellitus, any other Neurologic –conditions.

RESULTS

Total of 236 patients fulfilled the inclusion criteria and provided the information regarding their demographic details.

DEMOGRAPHIC CHARACTERISTICS

1. GENDER WISE DISTRIBUTION: Out of 236 patients, 190(80.5%) were males and 46(19.49%) were females and the data was tabulated as shown below. Male patients (80.5%) were almost quadrapled in number as that of female patients (19.49%) who are suffering with various Liver Disorders.

Table 1: Gender Distribution.

Gender	Number of patients	Frequency (%)
Males	190	80.5
Females	46	19.49

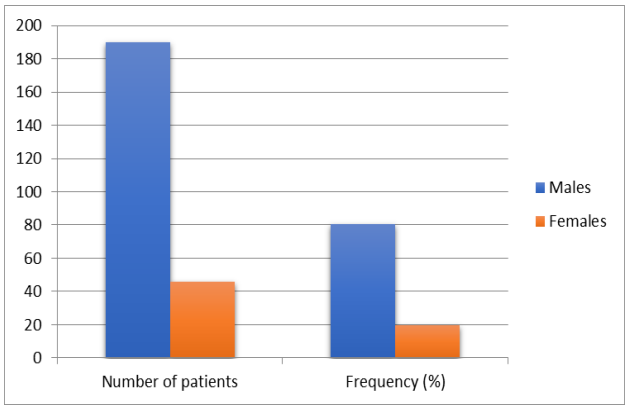

Fig 1: Gender wise Distribution.

2. AGE-WISE DATA DISTRIBUTION: The age of patients diagnosed with Liver Disorders was classified into different groups as follows: Among 236 patients, majority of patients i.e. 83 patients (35.16%) were found to be in the age group of 41-60 yrs, while 76(32.2%) were found to be in the age group of 21-40 yrs followed by 75 (31.77%) were in 61-80yrs and least patients of 02(0.84%) were in the age group of 12-20 yrs. A maximum prevalence is seen in the age group of 41-60yrs and least prevalence is seen in the age group of 12-20 yrs.

Table 2: Age- Wise Data Distribution.

Age (yrs)	Number of patients	Frequency in percentage
12-20	2	0.84
21-40	76	32.2
41-60	83	35.16
61-80	75	31.77

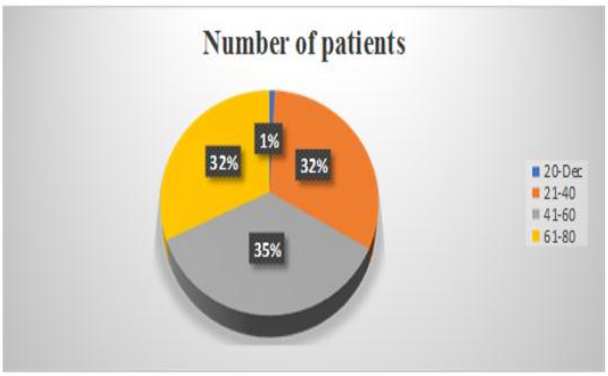

Fig 2: Age- wise data analysis.

3. LIVER DISORDERS OBSERVED DURING STUDY: In our study, the various Liver Disorders observed are Fatty Liver 152(64.4%) followed by Alcohol Liver disease 14 (5.93%), Hepatitis 25(10.59%), Liver Abscess 6(2.54%) and other diseases like Jaundice 23(9.74%), Hepatomegaly16 (6.77%).

Tab 3: Liver Disorders During Stuudy.

Liver disorders	No.Of Patients	Frequency
Fatty liver	152	64.40%
ALD	14	5.93%
Hepatitis	25	10.59%
Liver abscess	6	2.54%
Hepatomegaly	16	6.77%
Jaundice	23	9.74%

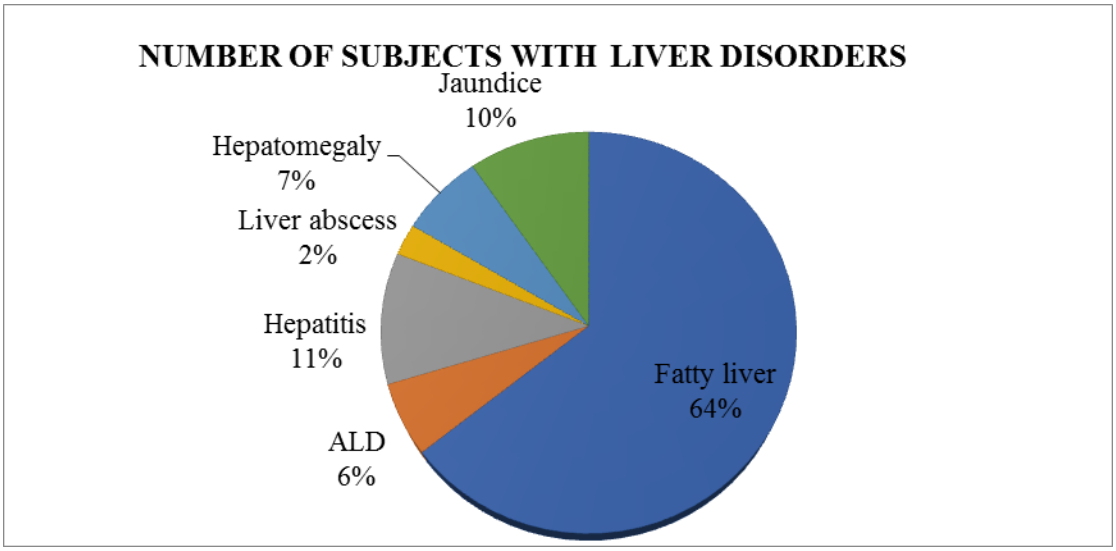

Fig 3: Liver Disorders During Study.

4. GENDER WISE DISTRIBUTION OF LIVER DISORDERS: In our study among 236 subjects majority of them were suffering with Fatty liver (152), among them 138 were male and 14 were female & least were affected with Liver abscess.

Tab 4: Gender wise distribution of Liver Disorders.

Disease	Total	Male	Female
Fatty liver	152	138	14
ALD	14	14	0
Liver abscess	06	06	0
Jaundice	23	17	6
Hepatomegaly	17	9	8
Hepatitiis	25	16	9

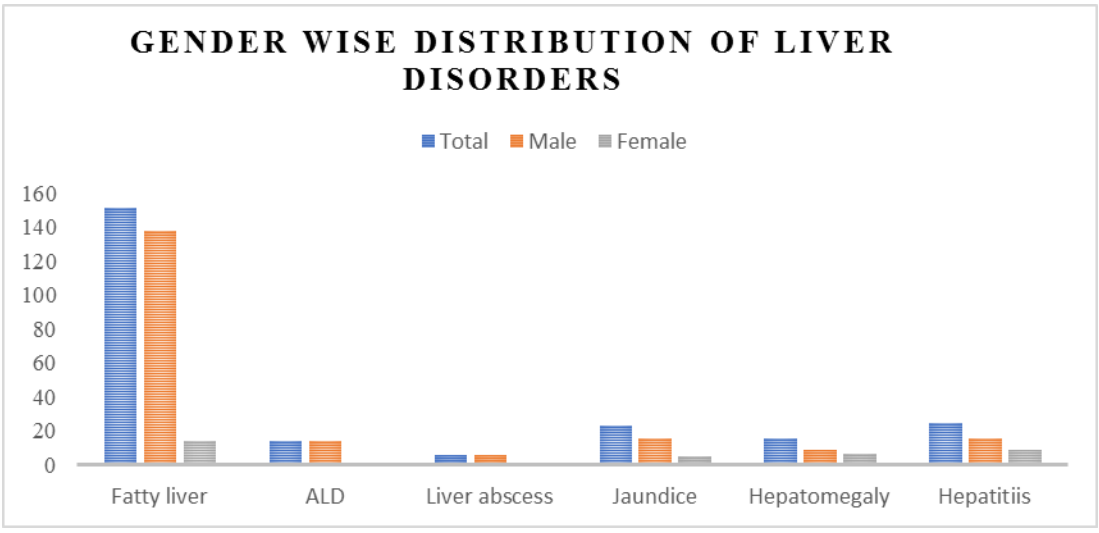

Fig 4: Gender wise distribution of Liver Disorders.

Tab 5: Frequency wise distribution of Liver Disorders in both genders.

Disease	Male	Frequency	Female	Frequency
Fatty liver	138	90.78%	14	10.14%
ALD	14	100%	0	0
Liver abscess	06	100%	0	0
Jaundice	16	69.56%	5	21.73%
Hepatomegaly	9	56.25%	7	43.75%
Hepatitis	16	64%	9	36%

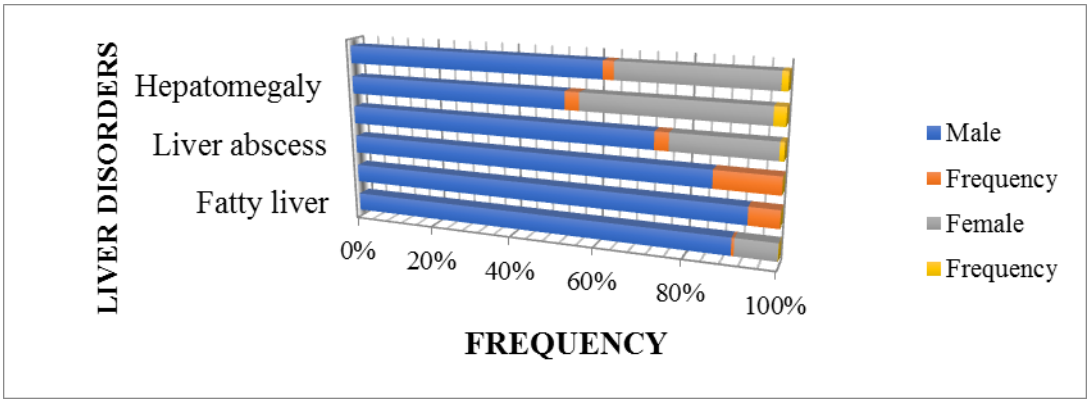

Fig 5: Frequency wise distribution of Liver Disorders in both genders.

5. RISK FACTOR WISE DISTRIBUTION: The frequency of distribution among total no of subjects(236) with different risk factors include Alcohol consumption(154) of which frequency is (65.25%), Blood transfusions(8) frequency is (3.38%), Toxins exposure(26) frequency is (11.01%), Viral infections(16) frequency is (6.77%), Contaminated food(24) of which frequency is (10.16%) and Drugs Abuse(8) frequency is (3.38%).

Tab 6: Risk factor wise distribution.

Risk factor	No. of subjects	Frequency of distribution
Alcohol consumption	154	65.25%
Blood transfuions	8	3.38%
Toxins exposure	26	11.01%
Viral infections	16	6.77%
Contaminated food	24	10.16%
Drugs abuse	8	3.38%

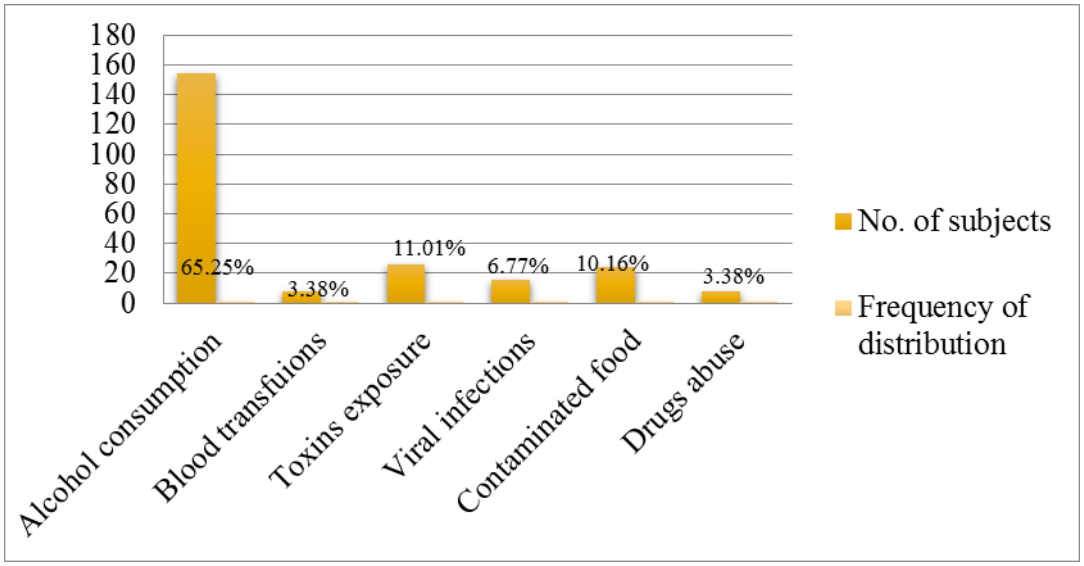

Fig 6: Distribution Of Subjects Based on Risk factors.

6. VARIOUS COMORBIDITIES OBSERVED DURING THE STUDY: Various comorbidities observed in our study are HTN of 42 subjects with 17.7% frequency, followed by DM with 37members (15.67%).

Tab 7: Various Comorbidities Observed During The Study.

Comorbidities	No. Of subjects	Frequency
HTN	42	17.7
DM	37	15.67
HTN+DM	26	11.01
NEUROLOGIC CONDITIONS	09	3.81
NILL SIGNIFICANT	122	51.69

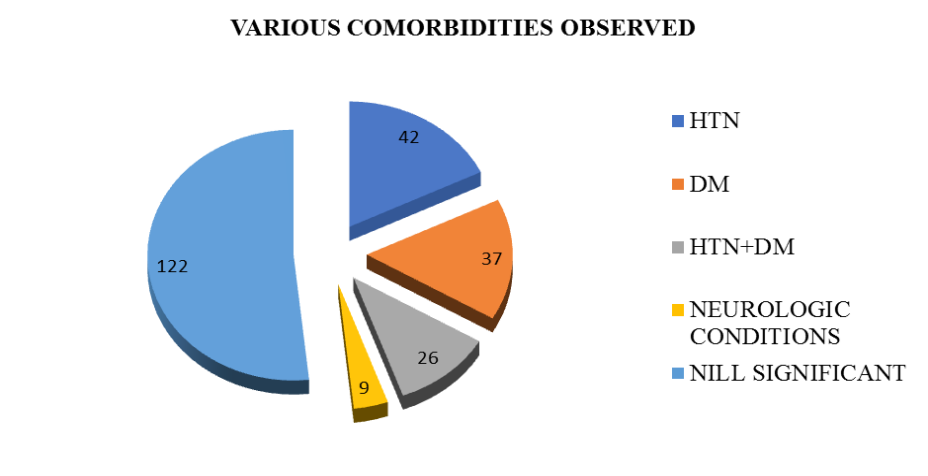

Fig 7: Various Comorbidities Observed During The Study

DISCUSSION

This epidemiological study is to compare the risk factors of LIVER DISORDERS in a cohort of People living in Karimnagar region. In this present study total no. of cases collected are 236 among these 190 are male subjects and remaining 46 are female. So, in this study prevalence of risk factors of Liver Disorders was found to be more in Male subjects compared to Female. Our results are nearly comparable to the **Yvonne N Flores** et.al**,** states that male subjects are more prone to the Liver Disorders. In our study, population including both Male and Female out of 236 subjects, 83 are within the age group of (41-60) i.e (35.16%). Maximum prevalence is seen in the (41-60) years age group subjects. A low prevalence is seen in extreme age groups of (12-20).

In this 236 subjects the major risk factor observed was consumption of Alcohol (65.25%), Exposure to Toxins (11.01%), Consumption of Contaminated Food (10.16%), Viral Infections (6.77%) Blood transfusions (3.38%), Drug Abuse (3.38%).Our results are consistent to the **Yvonne N Flores** et.al**,** with other studies that report high rate of excessive alcohol consumption.[7]

As per one of the findings of this study, advancing liver disorder or as a result of the complications of liver disorder ,alcohol is a risk factor for liver disorder irrespective of the many are nonspecific and may occur in other diseases patients above the age of 45 years. In total of 236 subjects 152 were diagnosed with Fatty Liver which was the major disorder we have observed in our study majority of the subjects were males compared to females. Our results are comparable to Monica Rodrigues de Araujo et al.[8]

CONCLUSION

Based on the results of this study, it is concluded that, various risk factors are responsible for liver disorders such as alcohol consumption, blood transfusions, exposure to toxins, viral infections, consumption of contaminated food and water, drug abuse. Among these, alcohol is the major risk factor having highest impact on symptomatic liver disorder. Second major risk factor with high impact on toxins exposure is hepatitis. Third major risk factor with high impact on consumption of contaminated food and water is Jaundice. More ever in the presence of more than one risk factors the progression to liver disorder is rapid.

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