AN OBSERVATIONAL STUDY ON THE RELATION BETWEEN CHRONIC LIVER DISEASE AND IT'S PRIMARY COMPLICATION PORTAL HYPERTENSION

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ABSTRACT

Aim: An observational study on the relation between chronic liver disease and its primary complication portal hypertension.

Objectives:

- To estimate the incidence of chronic liver disease in hospital patients.
- To compare the ration of thecomplications of chronic liver disease (portal hypertension).
- To distinguish the etiologies of chronic liver disease (Alcoholic liver disease, non-alcoholic fatty liver disease).

Methods: The sample population (n=60) was chosen from one of the well reputed hospitals of Hyderabad.All the patients were of different age group, sex, socio-economic status with different co-morbidities.A format consisting of patient profile, anthropometric measurements, bio chemical data, clinical data, 24 hour dietary recall, dietary habits ,social habits and past medical history

Result &conclusion: Poor knowledge and practices regarding chronic liver disease in the community are important weaknesses. 75% of males and 25% of females suffer from chronic liver disease in which 40-60 age group suffers more with this life threatening disease (36.6%) followed by 20 to 40 age group[21.6%], greater than 60 age group[16.6%], infants[13.3%], less than 20 age group [11.6%] respectively. People having sedentary lifestyle were more in number [71.6%] followed by moderate [28.3%] with zero percentage of heavy lifestyle suffering with this disease. Percentage of overweight people were 28.3%, 23% were obese, 13.3% were healthy. Annual income for 50% patients was below 2 lakhs, chronic liver disease was found to be more in overweight people. The percentage of people consuming alcohol and are non – smoker was 36.6% and 16.6% were alcoholic and smoker. The co-morbidities associated with chronic liver disease was diabetes mellitus (75%), hypertension(58.3%), coronary artery disease(25%), hepatitis B (25%), hepatitis C (16.6%), chronic kidney disease(15%). Patients with ascites (83.3%), people with portal hypertension was (78.3%), followed by 60% of people suffered from esophageal varices, 43.3 % of people were having hepatic encephalopathy, 28.3% of people were having hepato renal syndrome. The data obtained shows us about the prevalence and the complications of chronic liver disease.

Key words: chronic liver diseases, its primary complications and portal hypertension.

INTRODUCTION

Liver is the metabolic capital of the body. It is the largest of the body's internal organs weighing about 1.1-1.5kgs in adulthood. It is cone shaped reddish brown consisting of two major lobes.It accomplishes multiple metabolic tasks through its vast specialized network of cells and circulating channel within each of its thousands of microscopic functional units [10].

- Functions:

The liver has many metabolic functions related to the three energy yielding macronutrients [carbohydrate,protein and fat]:

- Carbohydrate metabolism:

-Formation and storage of glycogen in glycogenesis.

-Conversion of amino acids residues to glucose in gluconeogenesis. [10]

- Protein metabolism:

-Deamination of amino acids.

-Urea formation for removal of ammonia from body fluids. [10]

- Fat metabolism:

-Formation of cholesterol and phospholipids.

-Formation of bile salts.

-Conversion of carbohydrate and protein intermediates to fat through lipogenesis. [10]

Chronic liver disease: In the clinical context a disease of the liver that involves a process of progressive destruction and regeneration of the liver cells which leads to fibrosis and cirrhosis is known as chronic liver disease. It is a persistent inflammatory condition of the liver in which the biochemical and histopatholigical abnormalities are present over a long period of time.

Non-alcoholic fatty liver disease, a common cause of chronic liver disease in adults, is incompletely characterized in children [42]. Hepatic and hepatobiliary diseases are a common cause of morbidity and mortality in children.[43]

- Etiology:

Non-alcoholic fatty liver disease [NAFLD]/non-alcoholic steatohepatitis[NASH]:

NAFLD is manifested with abnormal aminotransferases or incidental radiographic findings of fatty liver.It is associated with co-morbidities such as obesity,diabetes mellitus and dyslipidemia.

NASH is now a part of a spectrum of NAFLD,it occurs in individuals who do not consume excessive amount of alcohol.

Alcoholic liver disease: Alcohol has a direct toxic effect on liver.The 'world health organization' has identified more than 60% alcohol related diseases on consumption of ethanol. Alcohol has a direct action on lipid metabolism in liver by decreasing fatty acid oxidation, enhancing fatty acid synthesis, stimulation to triglyceride formation leading to fatty liver.

- Complications:

1. Portal hypertension
2. Ascites
3. Hypersplenism
4. Esophageal varices
5. Hypoalbuminaemia
6. Hepatopulmonary syndrome
7. Hepatorenal syndrome

- Risk factors :
    1. -Alcohol
    2. -Obesity
    3. -Blood borne viruses
    4. -Metabolic syndrome including raised blood lipids

Chronic liver disease (CLD) may be accompanied by portal hypertension (PHT)[4].Portal hypertension is most frequently associated with cirrhosis and is also associated with complications such as variceal bleeding, ascites or hepatic encephalopathy. As such, clinically significant portal hypertension forms the prelude to decompensation and impacts significantly on the prognosis of patients with liver cirrhosis [1].

❖ Prevention:

Current PHT treatment strategies orientate on the existence and characterization of esophageal varices, which strongly correlate with the hepatic venous pressure gradient (HVPG)—the gold standard for quantification of PHT. For prevention of variceal bleeding, oral non-selective beta blockers (NSBBs) are used, while, in acute bleeding situations, intravenous somatostatin, octreotide or terlipressin are available [1].

These drugs aim to decrease portal pressure; however, not all patients achieve a hemodynamic response, which is defined by a HVPG decrease >10% of baseline. Thus, current research intensively seeks new treatment options for PHT. Most experimental strategies aim at structural (liver fibrosis) and/or dynamic (endothelial dysfunction, hyper dynamic circulation) factors, which contribute to the severity of PHT [1].

The term "Portal Hypertension" (PHT) is to describe a condition characterized by increment in portal pressures, at least 5 mm of Hg above inferior vena cava (IVC) pressure, which was associated with portal circulatory structural changes and gastrointestinal bleeding [2]. Patients with chronic bleeding usually present with chronic iron deficiency anemia [3].

Although they are found in up to 70% of patients with PH [pulmonary hypertension] and are more common in patients with EV and PHG, they rarely cause bleeding [3]. The pathogenesis of portal hypertension is increasingly understood and emerging knowledge of the vascular processes that underpin portal hypertension has paved the way for exploring novel biomarkers of vascular injury, angiogenesis, and endothelial dysfunction [5].

In cirrhosis, PHT is initiated by an increase in intrahepatic vascular resistance (IHVR) and then exacerbated by changes in the systemic and splanchnic circulation that increase the portal inflow [6].

❖ Treatment/management :

Treatment should include non specific therapy such as blood volume replacement and antibiotic prophylaxis as well as specific treatment such as pharmacology therapy and endoscopy therapy.Trans jugular intrahepatic Porto systemic shunt [TIPS] through a jugular route connects the hepatic and portal vein in the liver to reduce portal pressure and thus prevent variceal bleeding [11].It is very important to know that there is treatment for CLD with PHT such as to include high protein food in the diet,do proper exercise,exclude oily food,junk food etc. instead include food which help in diminishing cirrhosis and hence decreasing the effect of PHT [7].

Try to exclude alcohol consumption as much as possible because alcohol is directly absorbed by the stomach lining and then passed to liver and other organs thereby it is very important to reduce the intake of alcoholic beverages [8].

It is very important to include protein in the diet because in many CLD with PHT cases there is a lot of albumin loss so hence in order to prevent this loss good amount of balanced diet should be included [9].

REVIEW OF LITERATURE

According to the global disease burden study, it is the 12th cause of death, the 17th cause of years of life-lost, and the 23rd cause of disability adjusted life year in the world [12, 13].

The total number of global deaths attributed to liver cirrhosis is increased from 777, 800 in 1990 to 1030,800 in 2010[12]

The natural history of liver cirrhosis Is largely influenced by the occurrence of variceal bleeding, ascites and infection. [14-17]

Child-Pugh score, model for end stage liver disease [MELD] score, and their components (i.e. bilirubin,albumin,prothrombin time or international normalized ratio, creatinine, encephalopathy, and ascites) are considered as the major predictors for the survival of liver cirrhosis [14-18]

The investigators found that the prophylactic anticoagulation could not only decrease the incidence of PVT, but also reduce the development of hepatic decompensation events and improve the survival. Recently it has been also proposed that the identification of thrombotic risk factors forPVT should be helpful to stratify the benefit of prophylactic anticoagulant in liver cirrhosis [19, 20]

Portal hypertension is a severe and frequent complication of chronic liver disease. The primary factor in the development of portal hypertension is a marked increase in hepatic vascular resistance to portal blood flow, which was classically attributed to distortion of liver architecture inherent to cirrhosis [21]

Hepatic venous pressure gradient [HVPG] has been shown to be an accurate prognosis index in patients in cirrhosis. In several reports liver stiffness proved as effective as Hepatic venous pressure gradient [HVPG] in predicting clinical decompensation and portal hypertension related complications in patients with chronic liver disease [22]

In patients with cirrhosis due to HBeAg-negative CHB, lamivudine mono-therapy reduces HVPG especially when virological suppression and biochemical remission is achieved.[23]

Non-cirrhotic portal hypertension has recently been reported as a liver complication in humanwho are found to be associated withexposure to didonosine with portal hypertensionwho initially presented with massive ascites and portal vein thrombosis. On reviewing the literature on didanosine-related non cirrhotic portal hypertension and analyzed the findings of 61 similar previously reported cases. [24]

Spleen stiffness and liver stiffness were more accurate than other non –invasive parameters in identifying patients with esophageal varices and degrees of portal hypertension .A linear model that included spleen stiffness and liver stiffness accurately predicted hepatic vein pressure gradient [HVPG] values. [25]

Portal hypertension is a surrogate of advanced liver disease. Reduction of portal pressure is the most efficient step to prevent intestinal bleeding and treat ascites. But this has a limited impact on survival. Interruption or modulation of inflammatory stimuli leading to liver damage and dysfunction of other organs is keyin order to prevent death or liver transplantation as ultimate rescue. [26]

Many questions remain with regards to the exact therapeutic parameters of beta-blockers in patients with cirrhosis in general, and in patients with refractory ascites in particular, and additional studies on the optimal timing and dosage of beta-blockers are certainly needed. Perhaps the most appropriate timing for beta-blocker therapy was outlined in the “window hypothesis” from Krag and colleagues which suggests that beta-blockers are beneficial only in a narrow clinical window in the course of cirrhosis. This dilemma will be resolved only by a prospective randomized controlled trial, but until that evidence becomes available, the Baveno VI Consensus Workshop provides practical clues to guide physicians’ decision about NSBB therapy in patients with decompensated cirrhosis. [27]

A survey in which stated that about 48% of people who consume alcohol on regular bases are prone to develop cirrhosis, and hence this is the emerging reason for mortality. [28]

Patients with portal hypertension are primarily directed at controlling complications such ascites, hepatic encephalopathy and gastric ulceration. In cases, in which these clinical syndromes are refractory to treatment, pharmacologic intervention to lower portal venous pressure may be indicated. [29]

Prolonged exposure to didanosineis one of the main risk factors for developing non-cirrhotic portal-hypertension. Since the therapeutic options are not yet clear, symptomatic treatment of portal hypertension and discontinuation of didanosine should be considered as the primary options for treatment. [30]

Ascites is initiated by portal hypertension through leakage of excessive lymph from a congested liver, causing effective plasma volume to contract and renal sodium retention to follow as a consequence of this contraction .an observation showed the rate of ascites formation does not increase falling paracentesis nor does the plasma volume fall, both of which should occur if traditional concept of ascites formation is correct. [31]

Ascites could be made to reform in a patient with cirrhosis by administering a sodium retaining hormone. This observation indicates that it is possible for ascites to form as a consequence of plasma volume expansion in cirrhosis [31]

For the cirrhotic patients undergoing surgical or interventional shunts, the overall mortality was not significantly associated with the presence of portal vein thrombosis in previous studies. The presence of portal vein thrombosis might be associated with the long term mortality in non liver transplant patients with liver cirrhosis, but not with short term mortality. [32]

Quality of life is variably impaired in cirrhosis. All domains of health related quality of life, exceptpain were altered in cirrhosis [9%-42%] mainly in younger patients. There were minor differences in relation to gender were as etiology had no effects [33]

It is reported that the impact of chronic hepatitis-C progresses to cirrhosis in about 20% of patients .interferon treatment leads to transient responses in about 40% of patients and apparent eradication of infection in 7% -40% of patients [34]

One hundred and four patients with primary biliary cirrhosis and primary sclerosing cholangitis participated ,of whom 73% were women , with an average age 55+/-12 years. Of these patients 61% had cirrhosis (37% child's A ,23%child's B & 2% child's C)[35]

Portal hypertension accounts for the majority of morbidity and mortality that is encountered in patients with cirrhosis .recent data suggests that intrahepatic angiogenesis and sinusoidal remodeling could also been involved in sinusoidal resistance ,fibrosis and portal hypertension .[36,37]

METHODOLOGY

Description: The present study was conducted among 60 patients in a multi-specialtyhospital. A structured questionnaire was administered along with 24 hour dietary recall.

Participants: An observational study was done on 60 patients were various etiological factors were taken under consideration, patientprofile, anthropometricmeasurements, bio chemical data, clinical data,24 hour dietary recall, eatinghabits, social habits and past medical history.

Material and methods: A questionnaire was designed for 60 patients were different parameters were taken into account.

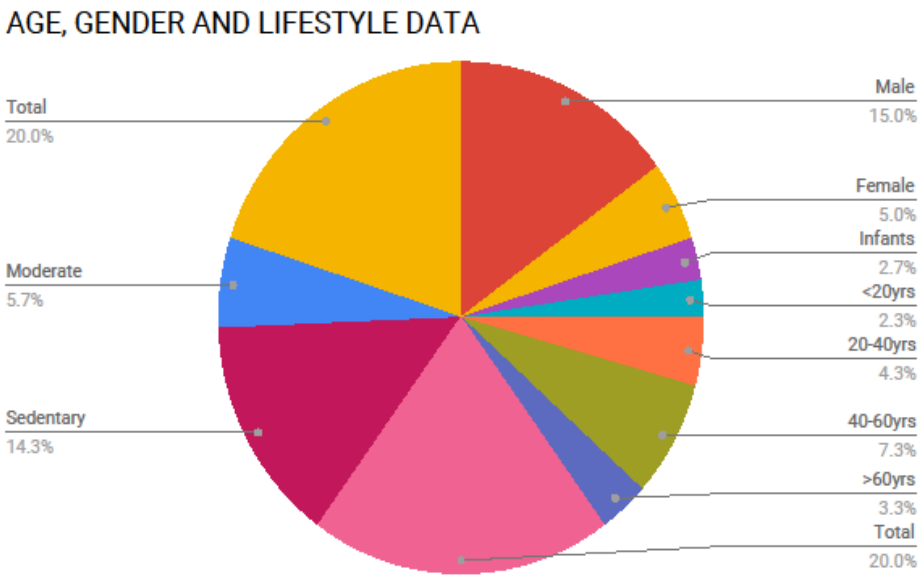
Firstly a one on one interaction was done with the patient were he/she was asked to tell the entire history of the disease outcome.

Secondly out of the above details the questionnaire was filled. Later on data analysis was done.

RESULTS AND DISCUSSION

Table 1:Socio economic profile of the respondents

Gender of respondents	Frequency number	Percentage
Male	45	75%
Female	15	25%
Age of respondents		
Infants	8	13.3%
<20	7	11.6%
20-40	13	21.6%
40-60	22	36.6%
>60	10	16.6%
Total	60	100%
Lifestyle		
Sedentary	43	71.6%
Moderate	17	28.3%
Heavy	0	0%
Total	60	100%

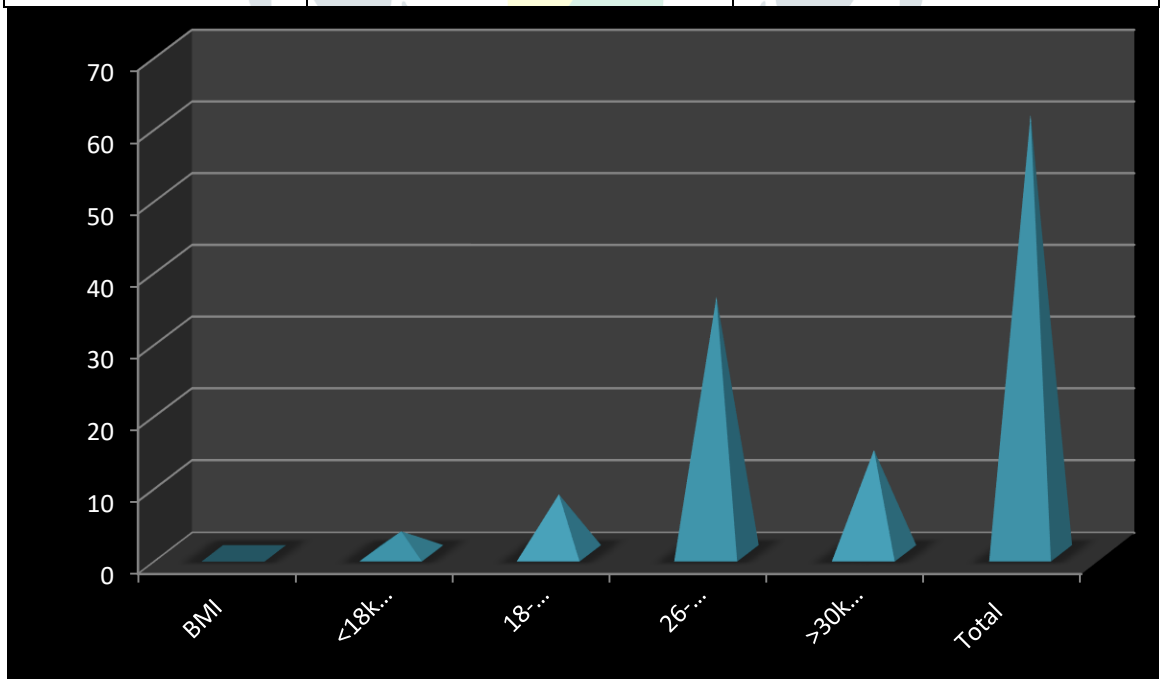


Total sample size of this study was 60 respondents out of total 60 respondents, 45(75%) were male and 15(25%) were female.

Lifestyle modifications leading to weight reduction and/or increased physical activity consistently reduced liver fat and improve glucose control/insulin sensitivity.Limited data also suggest that lifestyle interventions may hold benefits for histopathology. (41)

Table 2: Body mass index data

BMI	Frequency	Percentage
<18kgs/m2	3	5%
18-25kgs/m2	8	13.3%
26-30kgs/m2	35	58.3%
>30kgs/m2	14	23.3%
Total	60	100%

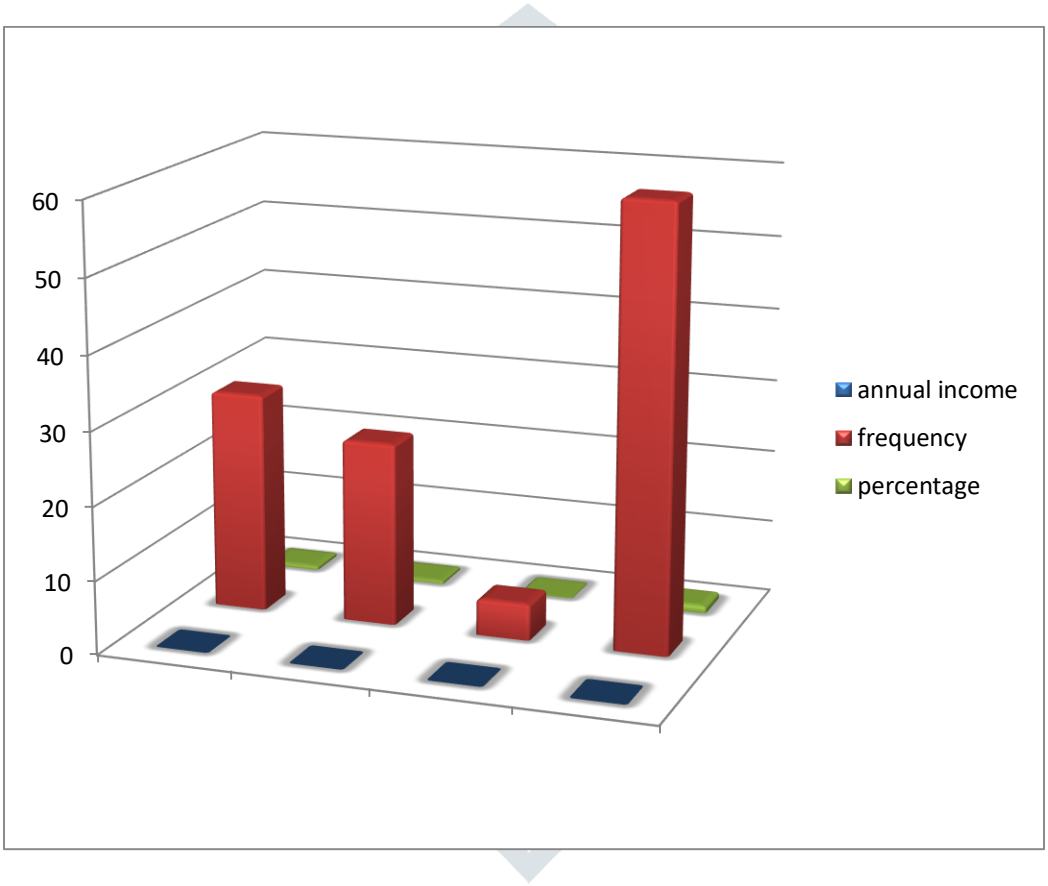


Out of 60 respondents 58% of the people were overweight, 23% were obese, 13.3% were healthy and 5% were underweight which shows that most of the CLD cases were present in overweight people.

Fifty eight of eighteen patients enrolled had varying degrees of non-alcoholic steatohepatitis, of these 26 had fibrosis and 8 had silent cirrhosis. The association of metabolic syndrome, female-sex, a long history of obesity and body mass index>45 were considered to be independent risk-factors for fibrosis. (40)

Table 3: Annual income data

Annual income	Frequency	Percentage
< 2 lakhs	30	50%
2-6 lakhs	25	41.6%
>6 lakhs	5	8.3%
TOTAL	60	100%

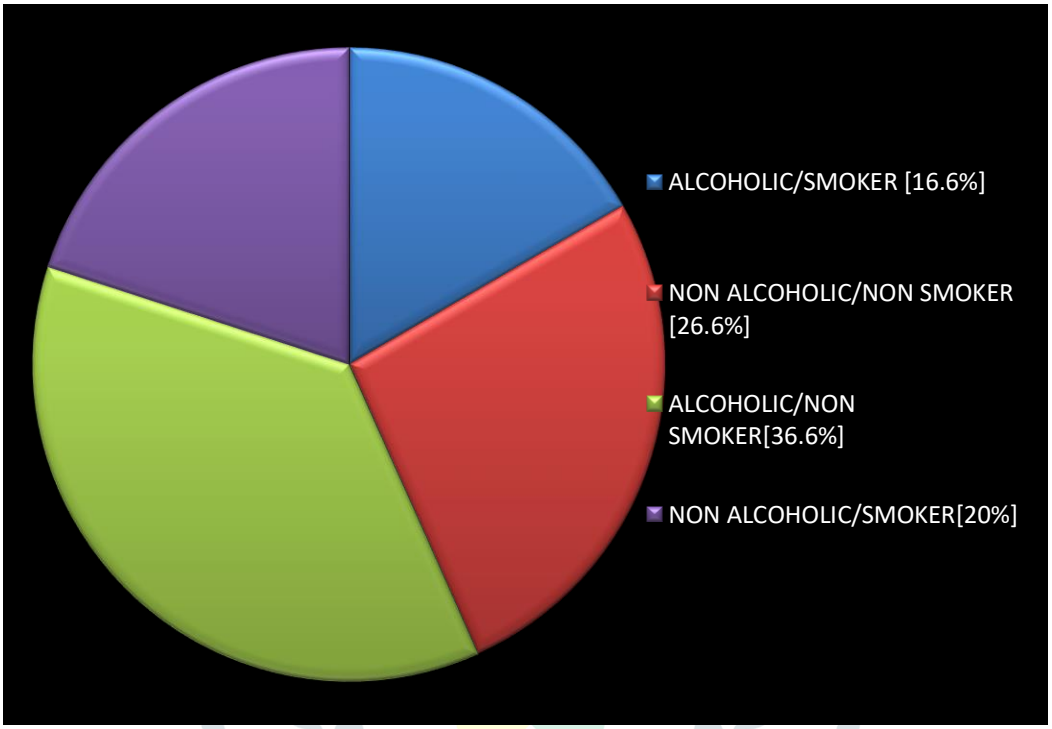


Out of 60 respondents 50% people annual income is below 2 lakhs, above 41.6 % people annual income between 2-6 lakhs , and about 8.3 % people annual income is greater than 6 lakhs .

According to National health interview survey 2017 about 792 people who were poor suffered from cld and about 809 nearly poor people also suffered from cld and about 2677 people who were rich suffered the most due to unhealthy social habits.[41]

Table 4: Social habits data

Social habits	Frequency	Percentage
Alcoholic / Smoker	10	16.6%
Non alcoholic / Non smoker	16	26.6%
Alcoholic / Non smoker	22	36.6%
Non alcoholic /Smoker	12	20%

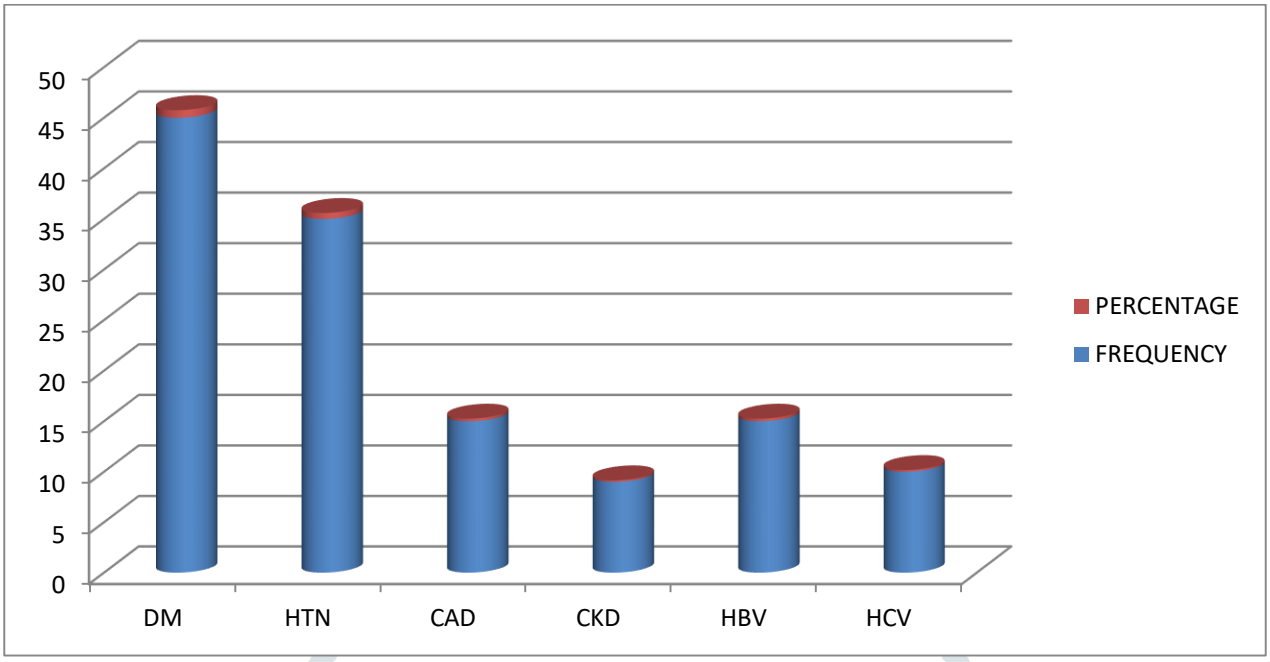


Out of 60respondents, 36.6% were alcoholic and non-smoker, 26.6% were non-alcoholic non-smoker, 20% were non-alcoholic smoker and 16.6% were alcoholic and smoker.

The prevalence rates for chronic liver disease were 11.78% (1998-1994), 15.66% (1999-2004), and 14.78 %(2005-2008).During the same period, alcoholic liver disease (1.38%, 2.21% and 2.05%) remain generally stable. In contrast, the prevalence of non alcoholic fatty liver disease increases from 5.51% to 9.84% to 11.0%. [42]

Table 5:Past medical history data

Diabetes mellitus	Hypertension	Coronary artery disease	Chronic kidney disease	Hepatitis b	Hepatitis c
45[75%]	35[58.3%]	15[25%]	9[15%]	15[25%]	10[16.6%]

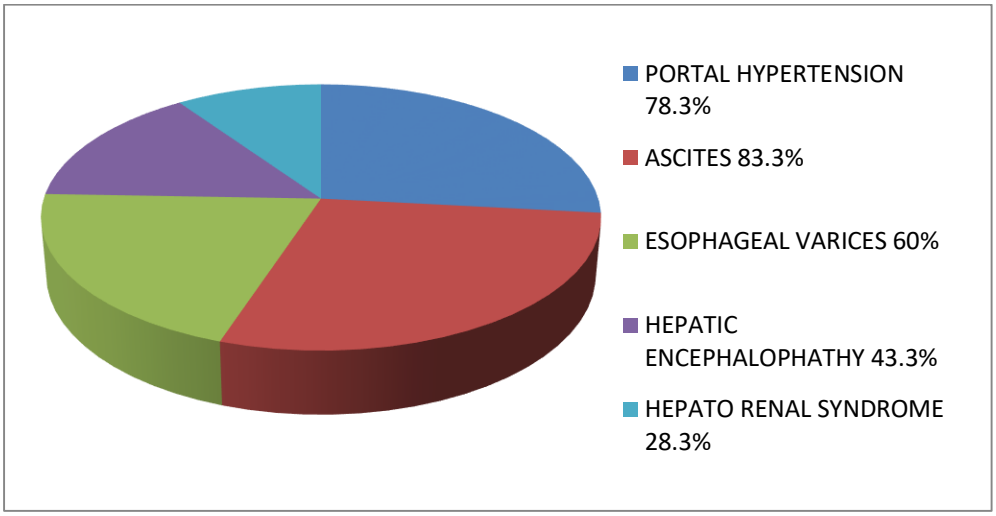


Out of the study 75% of people were having diabetes mellitus, 58.3 % of people were having hypertension, 25% of people were having coronary artery disease, 25% of people were having hepatitis B, 16.6 % of people were having hepatitis C,and 15% of people were having chronic kidney disease.

The prevalence of hepatitis B virus is 0.36%, 0.33%, 0.34% whereas the prevalence of hepatitis C is 1.95%, 1.97% , 1.68%,diabetes mellitus is 5.55%, 7.88%, and 9.11%,hypertension is 22.68%, 33.11%, 34.08% [44].

Table 6: Complications of chronic liver disease

Complication	Frequency	Percentage
Portal hypertension	47	78.3%
Ascites	50	83.3%
Esophageal varices	36	60%
Hepatic encephalopathy	26	43.3%
Hepato renal syndrome	17	28.3%



Out of the study 83.3% of people were having ascites,78.3% of people were having portal hypertension,60% of people were having esophageal varices,43.3% of people were having hepatic encephalopathy and 28.3% of people were having complication of hepato renal syndrome.

Sinusoidal intrahepatic portal hypertension most often is the result of fibrotic hepatopathies[45,46].Concurrent hypoalbuminemia secondary to hepatic synthetic failure lowers vascular colloid osmotic pressure that further aggravate ascites formation[47].In humans the hepatic renal syndrome is always accompanied by a state of refractory ascites and end stage liver failure[48,49].

Global study on figures of portal vein thrombosis [38]

First author [year]	Country	Number of total patients	Portal vein thrombosis number (%)
Amitrano (2012)	Italy	185	32 (17%)
Attili(2012)	Italy	129	25(19%)
Chen(2012)	Taiwan	101	25(25%)
D'Amico (2003)	Italy	291	37(13%)
Doumit (2009)	Canada	398	44(11%)
Hung(2012)	Taiwan	95	13(14%)
Lee (2012)	Taiwan	97	19(20%)

International incidence of chronic liver disease [39]

Frequency in 1000's:

Total number of liver disease	4497
Male	2261
Female	2236
18-44yrs	1247
45-64yrs	2258
65-74yrs	704
75 and 0ver	288
Total number of morbidity rate=4.5million Total number of mortality rate=40,545	

RECOMMENDATIONS

Chronic liver disease is a major public health problem throughout the world affecting hundreds of millions of people. It is a cause of considerable illness and death in human population from the acute infection or its effects, which may include chronic acute hepatitis, cirrhosis and primary liver cancer.

Avoiding alcohol after getting diagnosed with chronic liver disease helps reducing its complications.

Fatty changes in the liver are common whenever there is a high proportion of fat in the metabolic mixture, for example in uncontrolled diabetes , in starvation ,in some cases of obesity and when too much carbohydrate has been infused during intravenous feeding .

Modern dietary management of chronic liver disease essentially involves modification of the quality and quantity of food to be taken by the liver patients .the following guidelines are applicable to chronic liver disease irrespective of type, weight status, age, gender or occupation.

Dietary management in chronic liver disease

- Energy: consumption of food is difficult because of anorexia and ascites. The patient are usually emaciated by the time cirrhosis of the liver is diagnosed .the patient requires highly nutritious food i.e. high calorie diet is necessary for prolonged undernourishment.
- Protein: The serum albumin which is exclusively synthesized by the liver cells , is low in cirrhosis and aggravated by the loss of considerable amount of albumin into ascitic fluid
- Fats: even if fatty changes are present in the liver, fat should be given provided adequate amounts of proteins
- Salt restricted diet advised 0 gm
- Wide variety of cereals can be included liberally in their refined form
- Pulses to be included daily. cooked form is better tolerated
- Double toned milk and their products are preferred
- Cooking oil about 3 teaspoons per day inclusive of ghee, butter
- Whole fruit to be preferred , avoid fruit juices

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CASE STUDY FORMAT

I. NUTRITIONAL ASSESSMENT

1. Patient profile:
   a. Patients name-
   b. Age-
   c. Sex-
   d. Life style-
   e. Date of admission-
   f. Date of discharge-
2. Anthropometric measurement:
   a. Height-
   b. Weight-
   c. BMI-
   d. IBW-
   e. DBW-
      - Assessment: (modified MUST tool is used for assessment)

Nutritional screening:-

☐ Patient has been identified to be at minimal risk and does not need further assessment.
☐ Patient has been at nutritional risk and must undergo detailed nutritional assessment.

Nutritional Risk Factor:

- ○ Inadequate PO intake
- ○ Chewing/swallowing problem
- ○ Significant weight loss
- ○ Mouth ulcer
- ○ Poor skin integrity
- ○ Abnormal lab values
- ○ Dxof GI tract
- ○ Increased nutritional need
- ○ Nausea/vomiting
- ○ Other

Short term goals/recommendations:

- ○ Loose/gain weight
- ○ Maintain present nutritional status
- ○ To improve nutritional status
- ○ Maintain blood sugars within limit
- ○ Increase physical activity

3. Biochemical data:
   a. Diagnosis-
   b. Investigations-

4.Clinical data:

   a. Present complaint-
   b. Past medical history-
   c. Family history-
   d. Medications-

   e. Treatment/plan-

4. Dietary history(24hrs recall-

QUESTIONNAIRE TO ELICIT THE INFORMATION ON CHRONIC

LIVER DISEASE WITH PORTAL HYPERTENSION

General information:

1. Name:

2. Age and gender:

3. Marital status:

4. Profession:

5. Lifestyle:

(A) Sedentary (b) moderate(c) heavy

6. What is your annual income?

(a) BelowRs.2lakhs (b) Rs.2lakhs-4lakhs (c) Rs.4lakhs-6lakhs (d)> 6l

Anthropometric measurements:

1. Height:

2. Weight:

3. BMI:

4. IBW:

5. Dry body weight:

Socio economic data:

1. Family type:

a. Nuclear ( ) b. Joint ( ) c. Single ( )

2. Total number of family members: 1, 2, 3, 4 or more ( )

Eating habits:

1. How is your appetite?

(a) good

(b) Fair

(c) Poor

2. What is your food choice?

(a) Vegetarian

(b) Non vegetarian

(c) Jain

3. What is your bowel moment frequency?

(a) Once

(b) Twice

(c) Thrice

4. What is your total oil consumption per month per head?

(a) 1-2lt

(b) 3-4lt

(c) >5lt.

5. What is your salt consumption per day?

(a) 1tsp

(b) 1 ½ tsp

(c) 2tsp or more

6. What is your fruit consumption per day?

(a) Everyday

(b) Twice

(c) Thrice

7. What is your number of meals per day?

1( ) 2( ) 3 or more ( )

8. What is your fluid intake per day?

(a)1-2lt (b) 3-4lt(c)>5lt

9. Do you have any food allergies?

If YES then _____NO ( )

10. Do you consume any?

(a) Bakery products like (bread, pastries, biscuits etc.)

(b) Savoury products like (pickles, papads etc.)

(c) None of the above

11. Do you consume junk food in a week [yes/no?]

a) Onceb) twice c) more than two days

12. Do you consume fatty products like [yes/no?]

a) Ghee b) butter c) cream d) any other_____

Social habits:

(a)Smoking (b) alcoholic(c) tobaccochewing (d) anyother (e) none

13. How many numbers of cigarettes do you consume per day?

(a)2 (b)3 (c)more than 3

Physical activity:

1. Do you do physical activity [yes/no], if yes then how much then how much time do you spend

(a)1 ½ hr. (b)1hr (c)more than 1hr

2. What are your sleeping habits/no. of hours you sleep (a) 2-4hrs (b) 5-6hrs (c) 6-8hrs

3. Do you had any weight loss or weight gain if yes then

(Intentional or unintentional)

Medical history:

1. Do you have any past medical history?

(DM, HTN, CLD, CKD, ANY OTHER_____)

2. Do you consume any supplements like [yes/no?]

(a)Vitamin d (b) iron (c) multivitamins

If yes then since from how long_____

3. Do you consume any herbal medicine yes/no if yes then_____

4. Are you consuming any ayurvedic or herbal medicine (if yes_____/no?)

5. Do you had any GI complications (yes____/no)

6. Do you have any family history of?

Diabetes mellitus (), Hypertension () Coronary artery disease ( ) Hepatitis b ( ) or any other_____

7. Do you have any past surgical history (if yes_____/no?)

IS LIFE WORTH LIVING? IT ALL DEPENDS ON LIVER!!