ORIGINAL RESEARCH PAPER

General Medicine

DECOMPENSATED CHRONIC LIVER DISEASE WITH ALCOHOL ABUSE IN TERTIARY HEALTH CENTER

KEY WORDS:

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INTRODUCTION

Decompensated alcohol related liver disease occurs, when there is deterioration in liver function in a patient with cirrhosis, which presents with jaundice, ascites and hepatic encephalopathy.Alcoholic liver disease (ALD) describes a spectrum of conditions ranging from reversible fatty liver to alcoholic hepatitis (AH), cirrhosis, and hepatocellular carcinoma (HCC). AH is a distinct clinical syndrome caused by chronic alcohol abuse and carries a particularly poor prognosis with a 28-day mortality ranging from 30% to 50%. Although AH is an acute condition, nearly 50% of patients with AH have established cirrhosis at the time of clinical presentation.AH typically occurs in an individual with long-standing history of alcohol intake although abstinence for several weeks prior to admission is not uncommon. However, clinical presentation after abstinence of more than 3 months should raise suspicion of advanced underlying alcoholic cirrhosis or chronic liver disease.Several pro-inflammatory cytokines have been detected in AH patients. In uncomplicated cases, histology of AH is characterized by neutrophilic infiltration (a marker of alcohol-induced hepatitis), ballooning degeneration of hepatocytes, spotty necrosis and fibrosis in the perivenular and perisinusoidal space of Disse (''chicken wire'' fibrosis), and Mallory hyaline inclusions.

INCIDENCE

Globally, alcohol consumption is leading risk factor for both death and burden of disease and injury. Alcohol use accounts for 6.8% standardized deaths in men and 2.2% in women, with a disproportionate effect on young people. Alcohol is major cause of alcohol related morbidity and mortality. In tertiary care center most of them present with abdominal distension, jaundice, and hepatic encephalopathy in most of the cases.

The precise incidence of AH is unknown, although a prevalence of approximately 90% was noted in a cohort of 84 patients with alcohol abuse in tertiary care center during two months of my period. The true prevalence of AH is difficult to assess because AH may be completely asymptomatic and often remains undiagnosed.

ETIOLOGY AND PATHOPHYSIOLOGY

The aetiology of chronic liver disease covers a wide range of congenital or acquired abnormalities of the hepatocellular biochemical network. Although our knowledge has considerably increased in recent years, the aetiology of chronic liver disease often remains obscure. Acquired irreversible disturbances of normal liver function can be mediated by hepatotrophic viruses, chemicals, chronic oxygen depletion, or interference with the immune system. Considerable progress has been made in the detection and characterisation of hepatitis B, C, and D viruses as causative agents of chronic active hepatitis. Alcohol abuse remains the predominant cause of chronic liver disease in the Western world. The targets of autoantibodies used to diagnose autoimmune diseases of the liver and primary biliary cirrhosis continue to be biochemically defined. Their significance for the aetiology of the disease, however, remains to be established. Nonparenchymal cells play an important role in the sequence of events following hepatocellular injury and ultimately leading to liver cirrhosis. They release vasoactive compounds, cytokines, and other important mediators, and participate in the modulation of the extracellular matrix that is characteristic of liver fibrosis and cirrhosis. The biochemical basis of liver cell necrosis remains poorly defined. In spite of recent progress, and the detection of some new pathogenic principles that help in the understanding of the complications of chronic liver disease such as portal hypertension, oesophagogastric variceal bleeding, portosystemic encephalopathy, ascites, and other metabolic disturbances, many questions concerning the aetiology and pathophysiology of chronic liver disease and its complications remain to be answered. Viruses such as hepatitis B and hepatitis C, non alcoholic fatty liver disease, Hemochromatosis (iron overload), cystic fibrosis, wilsons's disease(copper accumulation), biliary atresia, galactosemia, glycogen storage diseases, genetic digestive disorders, primary biliary cholangitis, primary sclerosing cholangitis, medications such as methotrexate, amiodarane, methyldopa are non alcoholic causes.

PATHOPHYSIOLOGY

Non parenchymal cells play an important role in sequence of events following in hepatocellular injury and ultimately lead to liver cirrhosis. They release vasoactive compounds, cytokines and important mediators, participate in modulation of extracellular matrix that is characteristic of liver fibrosis and cirrhosis. The biochemical basis of liver cell necrosis remains poorly defined. Inspite of recent progress, detection of some new pathogenic principles that helps in understanding of complication of chronic liver disease such as portal hypertension, oesophagogastric variceal bleeding, hepatic encephalopathy, ascites, other metabolic disturbances, many questions concerning aetiology and pathophysiology of chronic liver disease and its complications remain to be answered. In my study I have seen so many of them with presentation of ascites and jaundice.

	Compensated Cirrhosis		Decompensated Cirrhosis	
Stage	Stage 1	Stage 2	Stage 3	Stage 4
Clinical	No Varices No Ascites	Varices No Ascites	Ascites +/- Varices	Bleeding +/- Ascites
Death (at 1 Year)	1%	3%	20%	57%

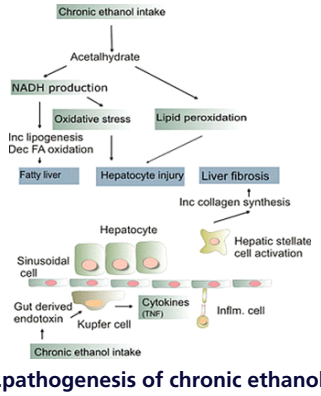

Fig.2.pathogenesis of chronic ethanol intake.

LIVER DISEASE IN WOMEN

Women more commonly present with acute liver failure, autoimmune hepatitis, benign liver lesions, primary biliary cirrhosis, and toxin-mediated hepatotoxicity. Women less commonly have malignant liver tumors, primary sclerosing cholangitis, and viral hepatitis. There is a decreased rate of decompensated cirrhosis in women with hepatitis C virus infection, no survival difference in alcohol-related liver disease, and improved survival from hepatocellular carcinoma. In general, men are 2-fold more likely to die from chronic liver disease and cirrhosis than are women. Liver transplant occurs less commonly in women than in men, with variable disease outcomes based on etiology. Women are more commonly affected by toxin-mediated liver disease, such as alcohol- and drug-induced liver disease, and have an increased prevalence of acute liver failure.Women are more susceptible than men to the toxic effects of alcohol on the liver for any given dose of alcohol, even though men abuse or depend on alcohol more than women, at a ratio of 2:1 in persons over the age of 26 years. Women are 10 times more likely to have primary biliary cirrhosis (PBC) than men and 4 times as likely to have autoimmune hepatitis. Benign liver lesions that predominantly occur in women include cavernous hemangioma, focal nodular hyperplasia (FNH), hepatic adenoma, biliary cystadenoma, and solitary hepatic cysts. NAFLD is most frequently recognized in men and postmenopausal women who have not received hormone replacement therapy (HRT). This suggests that sex hormones, particularly estrogen, play a role in the pathogenesis of NAFLD. Women clear acute hepatitis C virus (HCV) infection at a higher rate than do men. Hepatitis B virus (HBV) affects men and women similarly. However, male sex is a risk factor for reactivation of HBV infection after seroconversion from hepatitis B e antigen—positive to —negative and for the development of cirrhosis and HCC. In general, men are 2-fold more likely to die from chronic liver disease and cirrhosis than are women, according to an analysis by the National Center for Health Statistics that was reported in 2005. Women represent approximately 30% of liver transplant recipients.74 Women appear more likely than men to die on the waiting list in the Model for End-Stage Liver Disease (MELD) era vs the pre-MELD era. In fact, the disparity in transplant rates for women has increased in the MELD era as waiting-list mortality risk has risen, in particular for MELD scores of 15 or greater. The natural history of liver disease in women varies according to etiology. Although women have slower progression of fibrosis and decreased incidence of cirrhosis pretransplantation, after liver transplantation, women have a higher risk of advanced fibrosis and graft loss in HCV-related disease.In the most recent investigation of the role of sex and transplant outcomes in HCV-related disease, women had a 31% increased risk of advanced recurrent disease compared with men in multivariate models.In patients transplanted for HCV-related disease, women have a 14% increased risk of death at 5 years compared with men.

CLINICAL MANIFESTATIONS

When compensated liver disease progresses to decompensated liver disease, typical symptoms can include:

Fatigue, easy brusing/bleeding, itching, jaundice(yellowish discolouration of skin and eyes), ascites, pedal edema, abdominal pain, nausea,fever, brownish/orange colour urine, loss of appetite, weight loss, hepatic encephalopathy. Most of the cases are presented with ascites, jaundice, hepatic encephalopathy.

Diagnosis

Diagnosis can be made by routine blood investigations sucha s complete blood count, complete hemogram, in which most of the common blood picture is hypochromic microcytic anemia and series of special blood tests can often determine whether or not the liver is functioning properly. These tests can also distinguish between acute and chronic liver disorders and between hepatitis and cholestasis.

The most commonly performed blood tests include the following:
Serum bilirubin test: This test measures the levels of bilirubin in the blood. Bilirubin is produced by the liver and is excreted in the bile. Elevated levels of bilirubin may indicate an obstruction of bile flow or a problem in the processing of bile by the liver.

Serum albumin test: This test is used to measure the level of albumin (a protein in the blood) and aides in the diagnosis of liver disease.

Serum alkaline phosphatase test: This test is used to measure the level of alkaline phosphatase (an enzyme) in the blood. Alkaline phosphatase is found in many tissues, with the highest concentrations in the liver, biliary tract, and bone. This test may be performed to assess liver functioning and to detect liver lesions that may cause biliary obstruction, such as tumors or abscesses.

Serum aminotransferases (transaminases): This enzyme is released from damaged liver cells.

- Prothrombin time (PTT) test: The prothrombin time test measures how long it takes for blood to clot. Blood clotting requires vitamin K and a protein that is made by the liver. Prolonged clotting may indicate liver disease or other deficiencies in specific clotting factors.
- Alanine transaminase (ALT) test: This test measures the level of alanine aminotransferase (an enzyme found predominantly in the liver) that is released into the bloodstream after acute liver cell damage. This test may be performed to assess liver function, and/or to evaluate treatment of acute liver disease, such as hepatitis.
- Aspartate transaminase (AST) test: This test measures the level of aspartate transaminase (an enzyme that is found in the liver, kidneys, pancreas, heart, skeletal muscle, and red blood cells) that is released into the bloodstream after liver or heart problems.
- Gamma-glutamyl transpeptidase test: This test measures the level of gamma-glutamyl transpeptidase (an enzyme that is produced in the liver, pancreas, and biliary tract). This test is often performed to assess liver function, to provide information about liver diseases, and to detect alcohol ingestion.
- Lactic dehydrogenase test,5'-nucleotidase test,Alpha-fetoprotein test, Mitochondrial antibodies tests etc.

The national institute on alcohol abuse and alcoholism states that if AST is two times more than ALT then it is considered as ALCOHOL RELATED LIVER DISEASE.

Liver biopsy, percutaneous transhepatic cholangiography(PTC), CT scan ,Ultrasound are other investigations.

AIMS AND OBJECTIVES OF THE STUDY

To determine incidence of decompensated chronic liver disease with alcohol abuse in tertiary health center.
All the patients admitted in tertiary care center with chronic liver disease.

MATERIALS AND METHODS:

Source of the data- 60 patients who admitted with chronic liver disease with ethanol abuse in tertiary health center during two months of my internship period.

METHOD: Child Pugh Score.

DISCUSSION

Out of 60 patients who got admitted into tertiary care center in view of chronic liver disease with alcohol abuse, percentage of patients who are around 40-50 years is 46.66%, 20-30 years is 15.11%, and 30-40 years is 38.33%.

AGE		PERCENT
20-30 Years	9	15.11%

30-40 Years	23	38.33%
>40 Years	28	46.66%

SEX		PERCENT
Male	55	91.66%
Female	5	8.33%

Out of 60, 91.66% patients were men. About 8.33% were women.

TIME PERIOD OF ALCOHOL DRINKING		PERCENT
2-5 YEARS	7	11.66%
5-10 YEARS	15	25%
>10 YEARS	28	63.33%

Patients who had alcoholic history more than 10 years are of 63.33%, around 2-5 years are of 11.66%, and around 5-10years are 25%.

NON ALCOHOLIC RISK FACTORS		PERCENT
Hepatitis B	8	13.33%
Hepatitis C	5	8.33%
HIV	2	3.3%

Out of 60 patients, 8 were associated with Hepatits B around 13.33%, 5 were associated with Hepatitis C which is around 8.33%.

CHILD PUGH SCORE:

The Child-Pugh score consists of five clinical features and is used to assess the prognosis of chronic liver disease and cirrhosis.

Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3
Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points) Class A = 5 to 6 points (least severe liver disease) Class B = 7 to 9 points (moderately severe liver disease) Class C = 10 to 15 points (most severe liver disease)			

Chronic liver disease is classified into Child–Pugh class A to C,

Points	Class	One year survival	Two year survival
5–6	A	100%	85%
7–9	B	80%	60%
10–15	C	45%	35%

Fig3. Child pugh score

CHILD PUGH SCORE		
5 to 6 points	6	10%
7 to 9 points	12	20%
10-15 points	42	70%

Out of 60, 42 members had score of 10-15 points around 70%, and who had 7 to 9 points were 12 members around 20%, those who had 5 to 6 were 6 members around 10 %.

CONCLUSION

In my study I conclude that, out of 60 patients who got admitted with all the clinical manifestations, 90% of them presented with abdominal distension, icterus, hepatic encephalopathy. Around 75% were with alcohol abuse, in those who are chronic alcoholics were around 63%, those who are associated with other risk factors such as hepatitis b, hepatitis c, were about around 25%. Around 96% were men. Out of 60, 42 members had child pugh score of 10-15 points around 70%, and in them one year survival rate is 45% and two year survival rate is 35%, and who had 7 to 9 points were 12 members around 20%,in them one year survival rate is 80% and two year survival rate is 60%, those who had 5 to 6 were 6 members around 10 %, in them one year survival rate is 100% and two year survival rate is 85%.

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