Original Article

Spectrum of Cholelithiasis in Decompensated Cirrhotic

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Abstract

Background: Cirrhosis patients are up to twice as likely to develop cholelithiasis (gallstones) as the general population. This is primarily caused by gallbladder hypomotility (poor emptying), changes in bile acid composition, and altered cholesterol metabolism. The risk increases with liver disease severity. The impaired gallbladder motility is due to fibrotic and cirrhotic which liver alters systemic hormones and nerve signaling, preventing the gallbladder from emptying effectively and leading to stone formation. The diseased liver cannot properly transport and synthesize bile acids, creating cholesterol-supersaturated bile. Moreover, impaired liver metabolism of oestrogen further impairs gallbladder emptying and increases the risk of stone precipitation. Cirrhotic have a significantly higher risk of developing cholelithiasis. The stones formed are most often pigment stones rather than cholesterol stones. The majority remain asymptomatic, but when complications occur, morbidity is substantially higher. The liver's inability to process and secrete bile components normally is the primary driver. The failing liver has a reduced capacity to synthesize bile acids, changing the delicate balance of bile and making precipitation more likely. Autonomic dysfunction and altered hormonal signaling in cirrhosis impair the gallbladder's ability to contract, leading to bile stasis. Hypersplenism due to portal hypertension leads to increased red blood cell destruction, resulting in excess bilirubin, which forms pigment stones. The aim is to estimate prevalence of development of cholelithiasis in decompensated cirrhotic patients at tertiary care center of Northern India. **Material and Methods:** This study was conducted at Medical Gastroenterology Department at PGIMS, Rohtak. It was a prospective study done over five years, from 01.03.2021 to 28.02.2026, during which 400 confirmed decompensated cirrhotic patients were followed up for development of cholelithiasis. For better understanding 100 patients each of Alcoholic liver disease (ALD), Chronic Hepatitis C (HCV), Chronic Hepatitis B (HBV), and Metabolic dysfunction- associated steatotic liver disease (MASLD) were enrolled in the study after proper written consent. Patient were labelled decompensated cirrhotic on basis of ultrasonogram evidence of ascites with altered echotexture of liver, Pedal edema, Endoscopic proven variceal bleed, Serum bilirubin greater than 3 gm%. Cholelithiasis was also proven on ultrasonogram basis. All patients who were having cirrhosis for at least three years duration were enrolled in the study. All hepatitis B and C patients were confirmed on HbsAg and anti-HCV antibody on Enzyme linked immunosorbent assay (ELISA) test and HBV DNA and HCV RNA Quantitative on Polymerase chain reaction test (PCR). **Results:** Our department is Model treatment Center (MTC) under National Viral Hepatitis Control Program (NVHCP) and is one of the high flow centers in India. On daily basis, 2-3 new and 20 follow up patients of cirrhosis of different aetiologies come for consultation and till date approximately 10,000 cirrhotic patients have been treated at our department. On prospective analysis of 400 confirmed decompensated cirrhotic patients, out of which 100 each were ALD, HCV, HBV and MASLD related. In ALD subgroup, all were males, in HCV- 59% were males, in HBV-65% were male and in MASLD-55% were male. Thus, there was male predominance in all the groups. Moreover, in all four groups, maximum number patients were above 40 yrs of age and peak was between 50-70 yrs, with mean age of 59 yrs. The prevalence of cholelithiasis was maximum in MASLD subgroup (32%), followed by HBV (25%), HCV (21%) and ALD (20%). **Conclusion:** Our vision regarding complications of chronic liver disease, especially in decompensated stage, from hepatic encephalopathy, hepatorenal syndrome, gastro-intestinal bleed, recurrent infections, refractory ascites etc. has to become broader and should must include cholelithiasis and sexual dysfunction. Moreover, cholelithiasis has to be dealt with symptoms associated with it and stage of liver disease, according to Child-Pugh score.

Keywords: Cholelithiasis, HBV, HCV, ALD, MASLD, Decompensated, Cirrhotic.

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Introduction

Patients with chronic liver disease (CLD), particularly cirrhosis, have a significantly higher risk of developing cholelithiasis.[1,2] The stones formed are most often pigment stones rather than cholesterol stones. The majority remain asymptomatic, but when complications occur, morbidity is substantially higher. The liver's inability to process and secrete bile components normally is the primary driver. The failing liver has a reduced capacity to synthesize bile acids, changing the delicate balance of bile and making precipitation more likely. Autonomic dysfunction and altered hormonal signaling in cirrhosis impair the gallbladder's ability to contract, leading to bile stasis. Hypersplenism due to portal hypertension leads to increased red blood cell destruction, resulting in excess bilirubin, which forms pigment stones. Treatment depends on severity of the liver

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disease, which is typically measured using the Child-Pugh classification or MELD (Model for End-Stage Liver Disease) score. Asymptomatic Gallstones are usually managed conservatively with close monitoring, as the risk of surgery often outweighs the risk of the stones. Symptomatic gallstones (Class A and B) are treated with laparoscopic cholecystectomy in well-compensated or moderately compensated patients. However, surgery carries increased bleeding risks and must be performed by specialized hepatobiliary teams. In decompensated cirrhosis (Class C) patients, surgery carries very high mortality rates. In these cases, conservative management or minimally invasive procedures like endoscopic stenting are strongly preferred over open surgery. When gallstones cause choledocholithiasis or cholecystitis, they require immediate attention. In cirrhotic patients, these conditions can precipitate hepatic decompensation, including jaundice, ascites, and encephalopathy.

Aim of Study: To estimate prevalence of development of cholelithiasis in decompensated cirrhotic patients at tertiary care center of Northern India.

Materials and Methods

This study was conducted at Medical Gastroenterology Department at PGIMS, Rohtak. It was a prospective study done over five years, from 01.03.2021 to 28.02.2026, during which 400 confirmed decompensated cirrhotic patients were followed up for development of cholelithiasis. For better understanding 100 patients each of Alcoholic liver disease (ALD), Chronic Hepatitis C (HCV), Chronic Hepatitis B (HBV), and Metabolic dysfunction- associated steatotic liver disease (MASLD) were enrolled in the study after proper written consent. Patient were labelled decompensated cirrhotic on basis of ultrasonogram evidence of ascites with altered echotexture of liver, Pedal edema, Endoscopic proven variceal bleed, Serum bilirubin greater than 3 gm%. Cholelithiasis was also proven on ultrasonogram basis. All patients who were having cirrhosis for at least three years duration were enrolled in the study. All hepatitis B and C patients were confirmed on HbsAg and anti-HCV antibody on Enzyme linked immunosorbent assay (ELISA) test and HBV DNA and HCV RNA Quantitative on Polymerase chain reaction test (PCR).

Results

Our department is Model treatment Center (MTC) under National Viral Hepatitis Control Program (NVHCP) and is one of the high flow centers in India. On daily basis, 2-3 new and 20 follow up patients of cirrhosis of different aetiologies come for consultation and till date approximately 10,000 cirrhotic patients have been treated at our department. On prospective analysis of 400 confirmed decompensated cirrhotic patients, out of which 100 each were ALD, HCV, HBV and MASLD related. In ALD subgroup, all were males, in HCV- 59% were males, in HBV-65% were male and in MASLD-55% were male. Thus, there was male predominance in all the groups. Moreover, in all four groups, maximum number patients were above 40 yrs of age and peak was between 50-70 yrs, with mean age of 59 yrs. The same pattern of above 40 years of age was seen in all groups of patients who developed cholelithiasis. Overall survival in ALD groups was lesser in ALD group, as no patient was seen above 70 yrs of age group. The prevalence of cholelithiasis was maximum in MASLD subgroup (32%), followed by HBV (25%), HCV (21%) and ALD (20%).

Table 1: Showing Distribution of Different Aetiologies and Prevalence of Cholelithiasis

Total Decompensated Cirrhotic Patients	ALD	HCV	HBV	MASLD
400	100	100	100	100
Cholelithiasis	20 (20%)	21 (21%)	25 (25%)	32 (32%)

Table 2: Showing Age Distribution in Total Pool of Decompensated Cirrhotic Patients

Total Patients (400)	ALD (100)	HCV (100)	HBV (100)	MASLD (100)
20-30 Yrs	0 (0%)	0 (0%)	5 (0%)	7 (7%)
31-40 Yrs	0 (0%)	10 (10%)	10 (10%)	7 (7%)
41-50 Yrs	36 (36%)	10 (10%)	30 (30%)	12 (12%)
51-60 Yrs	36 (36%)	31 (31%)	25 (25%)	26 (26%)
61-70 Yrs	28 (28%)	35 (35%)	20 (20%)	42 (42%)
71-80 Yrs	0 (0%)	14 (14%)	10 (10%)	6 (6%)

Table 3: Showing Age Distribution in Decompensated Cirrhotic Who Developed Cholelithiasis

Total Patients Developing Cholelithiasis (98)	ALD (20)	HCV (21)	HBV (25)	MASLD (32)
20-30 Yrs	0 (0%)	0 (0%)	0 (0%)	0 (0%)
31-40 Yrs	0 (0%)	3 (14.28%)	0 (0%)	0 (0%)
41-50 Yrs	15 (75%)	3 (14.28%)	19 (75%)	0 (0%)
51-60 Yrs	5 (25%)	8 (38.09%)	0 (0%)	12 (37.5%)
61-70 Yrs	0 (0%)	4 (19.04%)	6 (25%)	20 (62.5%)
71-80 Yrs	0 (0%)	3 (14.28%)	0 (0%)	0 (0%)

Discussion

Patients with liver cirrhosis are more likely than the general population to develop cholelithiasis. There are several pathogenic mechanisms that improve the formation of gallbladder stones (GS): hypersplenism that causes chronic hemolysis, hypomotility of the gallbladder, reduced secretion of cholesterol, reduced synthesis of bile acids and their transportation. Any of these factors can influence the

pathogenesis of gallstones and the onset of biliary colic, acute cholecystitis and biliary pancreatitis. The prevalence of GS in patients with CLD is 20-40%, while it is 10-15% among the general population.[3] Moreover, the incidence of gallstones increased significantly with the progression of liver disease.[4] A study included 131 patients with CLD; Their mean age was 52.9, SD (±11.7) years. There were 55 (42%) males and 76 (58%) females. Chronic HCV infection was found in 101 (77%) patients and 21 (16%) had chronic HBV infection.[5] The prevalence of cholelithiasis in the examined patients was 50.4% (66 of 131 patients) and cholelithiasis was more associated with decompensated than compensated liver diseases (54.1% Vs 30.4%; p value <0.05). In general, the frequency of GS in patients with CLD ranges from 3.6% to 46%, with a 1.2- to 5-fold increase compared with the general population.[6] There appears to be no definitive epidemiological evidence that alcohol affects GS formation. Froutan et al.[7] observed that GSD disease was not significantly related to alcohol consumption. In Friedman et al,[8] study, there was no association between chronic alcoholism and lithogenesis. Trotman and Soloway,[9] observed that the type of GS in patients undergoing cholecystectomy was not influenced by alcohol consumption. Bouchier,[10] concluded that there were no good reasons why alcoholics should be more prone to develop gallstones. Several other studies have confirmed that alcohol consumption reduces the risk of GS.[11] Grodstein et al,[12] found a protective effect of alcohol consumption in women without cirrhosis, suggesting that a decreased risk of symptomatic GS was associated with increased alcohol intake. MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease) and gallstones frequently occur together because they share the same root metabolic issues. In one study, Gallstone prevalence was higher in the MASLD group (14.2%) compared to the non-MASLD group (8.5%).[13] HCV infection significantly increases the risk of developing cholelithiasis, with a pooled meta-analysis showing that HCV-infected individuals have nearly double the risk of gallstone disease compared to uninfected controls. The prevalence is elevated across both sexes and is especially pronounced in patients with advanced liver fibrosis or cirrhosis. Chronic HCV infection is frequently associated with hepatic steatosis and abdominal obesity, which are major independent drivers of gallstone formation in these patients. Systemic and local gallbladder inflammation induced by the virus may contribute to altered gallbladder emptying. Systematic reviews reveal that HCV-infected patients are about 1.83 times more likely to develop gallstones than the general population, with men showing an Odds Ratio (OR) of 2.07 and women an OR of 3.00. While non-cirrhotic HCV patients do show an elevated risk, but the prevalence significantly increases to 50% in decompensated HCV-cirrhosis (e.g., Child-Pugh B and C stages). Younger males with HCV show a particularly high susceptibility to gallstones, while the classic female-to-male gallstone disparity tends to diminish in older HCV patients. One study conclude that chronic HCV infection was strongly associated with GBD among men but not women and GBD was more common in adults with severe liver disease.[14] HBV infection is considered a risk factor for cholelithiasis, particularly in individuals with chronic HBV or those who have developed HBV-associated liver cirrhosis. The risk increases due to prolonged hepatic inflammation, altered bile salt metabolism, and impaired gallbladder function over time. Patients with chronic hepatitis B (CHB) and HBV-related cirrhosis have a significantly higher incidence of gallstones (ranging from 18% to nearly 30%) compared to the general population. HBV X protein is known to interfere with lipid aggregation and cholesterol transport, leading to cholesterol accumulation in the liver, which can promote cholesterol gallstone formation. HBV can cause functional and structural changes to the gallbladder (cholecystopathy), impairing normal motility and increasing the likelihood of biliary sludge and stone formation. Older age, higher GGT level and alcohol use are independent risk factors for gallstone in CHB. Shao etal showed that older age, higher total serum bilirubin level (TBIL) and ascites are independent risk factors for gallstone in liver cirrhosis. Higher TBIL level is an independent risk factor for gallstone in the liver cirrhosis complicated by HCC.[15] In the general population, most studies see hepatitis C as a risk factor for GSD,[16] whereas hepatitis B is not shown to be associated with GSD.[17,18] The findings in our study are in alignment with previous studies which also highlight the fact that cirrhotic patients have more chances of developing cholelithiasis than general population. We also agree to previous proven fact that as stage of cirrhosis advance, then chances of developing cholelithiasis increase in direct proportion. Our finding that cholelithiasis is most common in MASLD related CLD, has already been testified by available literature. The mean age of developing cholelithiasis is usually above 50 years and same has been seen in our study pool. The two-contrast fact which appeared in our analysis, in comparison to available data are male preponderance and increased chances of cholelithiasis in ALD group and this is area for further research.

Conclusion

Our vision regarding complications of chronic liver disease, especially in decompensated stage, from hepatic encephalopathy, hepatorenal syndrome, gastro-intestinal bleed, recurrent infections, refractory ascites etc. has to become broader and should must include cholelithiasis and sexual dysfunction. Moreover, cholelithiasis has to be dealt with symptoms associated with it and stage of liver disease, according to Child-Pugh score.

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Conflicts of interest

There are no conflicts of interest.

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