"CLINICAL PROFILE OF INFECTIONS IN CIRRHOTIC PATIENTS- ONE YEAR HOSPITAL BASED CROSS - SECTIONAL STUDY"

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ABBREVIATIONS

Glossary	Abbreviations
ACLF	Acute-on-chronic liver failure
ALT	Alanine aminotransferase
BT	Bacterial translocation
CA	Community-acquired
CAIDS	Cirrhosis associated immune dysfunction syndrome
CI	Confidence interval
CT	Computed tomography
DAILYs	Disability adjusted life years
GGT	Gamma-glutamyl transferase
GNB	Gram-negative bacilli
HA	Hospital-acquired
HBsAg	Hepatitis b surface antigen
HCA	Healthcare-associated
HCV	Hepatitis c virus
HRS	Hepatorenal syndrome
MDR	Multi-drug resistant
MDRA	Multidrug-resistant agents
MRI	Magnetic resonance imaging
NAFLD	Nonalcoholic fatty liver disease
PPI	Proton pump inhibitor
SBP	Spontaneous bacterial peritonitis
UTI	Urinary tract infection
XDR	Extensively drug-resistant

ABSTRACT

Introduction:

Considering the rapidly changing profile of infections in liver cirrhosis patients periodic studies documenting the etiological profile of organisms in these patients are extremely vital in this regard.

Objective of the study:

To study the incidence and clinical profile of infections in cirrhotic patients.

Materials and methods:

The current study was a prospective observational study conducted in the department of General medicine, KLES Dr. Prabhakar Kore Hospital, Belgaum between 1st January June 2015 to June 2016, for a period of 1 year. The study has included 100 patients admitted in the wards and ICU with decompensated cirrhosis of liver. The profile of infection was assesed using culture and sensitivity of various body fluids including ascitic fluid, urine and blood. The analysis was done by mean and standard deviation for quantitative varibales, frequerncy and proportion fro categorical variables using IBM SPSS statistical software version 21.

Results:

The mean age of study population was 52.85 ± 9.49 with high male preponderance (75%) in study populations. The proportion of participants with HCV positivity was 22% and HBsAg positivity was seen in 32% of the study population.

The other common etiology of cirrhosis was alcoholism in 53% of the study population. Ascitic fluid culture was positive in 48% of the subjects, with E. coli being the most common organism (26%). The urine culture was positive in 16% of the subjects with E. coli (12%) as the common organism. Blood culture was positive in 39% of the subjects, with Klebsiella Pneumoniae (15%) as most common organism. The most common infection was SBP in 53%, followed by UTI in 16%, pneumonia in 26% and cellulitis in 5% of the subjects.

Conclusion:

Infection is very common in liver cirrhosis patients. Liver cirrhosis patients are susceptible to wide range of infections ranging from spontaneous bacterial peritonitis, urinary tract infections and sepsis. The profile of organisms is also quite varied in these patients.

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INTRODUCTION

Bacterial infection is one of the most important clinical problems in patients with decompensated cirrhosis. It is present at admission or develops during hospitalization in 20% to 60%odf the patients with cirrhosis. It is also reported to be one of the common causes of mortality in liver cirrhosis patients. [1]The most common causes of infection reported has been constantly changing with time due to a multitude of reasons. Most studies assessing the aetiology and clinical types of bacterial infections in cirrhosis were performed in the 1980s have reported urinary tract infections, pneumonia, and spontaneous bacterial peritonitis (SBP) as the most common infections. Majority of infections were community acquired and approximately 70% to 80% of the isolated organisms were gram-negative bacilli (GNB).[2]

However, during the last decade practice in hepatology has considerably changed and this may have influenced the epidemiology of bacterial infections in liver diseases. Treatment of cirrhotic patients with severe complications in intensive care units has been on the rise. The rapid extension of the liver transplantation programs and the invasive procedures used in this setting have resulted in the emergence of hospital acquired infections as an important cause replacing the community acquired infection pattern. [3] Development and extensive use of various new invasive diagnostic and treatments modalities to treat various complications of cirrhosis, like variceal ligation, transjugular intrahepatic portosystemic shunt, and arterial embolization or percutaneous ablation of hepatocellular carcinoma. [4] These treatments may be also associated with infections. Extensive use of norfloxacin for selective decontamination of intestines and prevention of spontaneous bacterial peritonitis also may have

changed the type and aetiology has also resulted in development of norfloxacin resistant infections and also emergence of gram positive bacterial infection as one of the common causes of infection these patients.[5]

Bacterial infection is a major cause of decompensation.[6] Patients with cirrhosis are at an increased risk of developing bacterial infections, sepsis, severe sepsis, and death develops during hospitalization in about 25%-30% of patients. Bacterial infection is not only more frequent but also more severe in cirrhosis, causing a four-fold increase in the probability of death, reaching 38% at 1 month. [7, 8] Infection can accentuate circulatory dysfunction leading to the development of hepatorenal syndrome (HRS) and can also induce an excessive pro-inflammatory response that could contribute to the development of sepsis-related organ failure and septic shock.[9] Therefore, it is important to identify determinants of poor prognosis in patients with bacterial infections and cirrhosis in order to be alert to the group of patients with the highest risk of death and, if possible, reverse the deleterious effect of these determinants by modifying the standard clinical practice performed in this major disease.[10]

Need for the study

In cirrhosis, decreased bacterial clearance, as well as structural and functional alterations in the intestinal mucosa, leading to an increased permeability to bacteria. The most common and life threatening infections in cirrhosis are spontaneous bacterial peritonitis, urinary tract infections, pneumonia, endocarditis and skin and soft tissue infections. Patients with decompensated cirrhosis have increased the risk of developing sepsis, multi-organ failure. The etiological profile, factors associated with infection and the impact of it on prognosis is quite variable across different settings. Also as pointed out the etiological profile is constantly changing and emerging as a

challenge for clinicians to choose appropriate antimicrobial therapy. Hence knowledge of common organisms causing infection in liver cirrhosis patients in a given setting is vital for clinicians treating the patients with cirrhosis. But the studies conducted on the subject are relatively scarce from India. The current study is an attempt to fill this vital gap in knowledge.

AIMS AND OBJECTIVES

1. To study the incidence and clinical profile of infections in cirrhotic patients.

REVIEW OF LITERATURE

Cirrhosis of Liver-Back ground:

Liver cirrhosis is defined in histology as a bridging fibrosis- a late stage of hepatic fibrosis-leading to deranged liver architecture and regenerative nodules. Liver cirrhosis is considered the end stage of a variety of chronic liver diseases and is irreversible in its advanced stages. Cirrhosis is characterized by poor life expectancy and is a leading cause of morbidity and mortality.[11] The natural history of cirrhosis is characterized by a silent course until decompensation when the progressive deterioration of liver function causes a rapid decline in life expectancy. The early stage of the disease is usually referred to as 'compensated cirrhosis', while the late one, defined by the appearance of ascites, bleeding, encephalopathy or jaundice, is termed 'decompensated cirrhosis'. Due to the strikingly different survival,[12] compensated and decompensated cirrhosis are considered two distinct clinical entities.[13]long-lasting initial phase denominated compensated cirrhosis, characterized by the absence of symptoms and excellent survival, followed by an advanced stage (i.e. decompensated cirrhosis), marked by the appearance of complications related to the presence of portal hypertension or liver dysfunction and associated with an elevated mortality rate. [14] Recently, a new clinical entity denominated acute-on-chronic liver failure (ACLF) has been identified as an alternative path in cirrhosis progression, characterized by its acute onset and poor prognosis. [15]Liver cirrhosis carries the risk of life-threatening complications, partly due to several co-morbidities. Medical treatments that may halt the progression of compensated cirrhosis to decompensated cirrhosis are currently being developed. [11]Recent advances in the understanding of the natural history and pathophysiology of cirrhosis, and in the

treatment of its complications, resulting in improved management, quality of life and life expectancy of cirrhotic patients. At present, liver transplantation remains the only curative option for a selected group of patients, but pharmacological therapies that can halt progression to decompensated cirrhosis or even reverse cirrhosis are currently being developed. Because of the increasing prevalence of chronic viral hepatitis and (alcoholic nonalcoholic) steatohepatitis and their high-risk evolution toward liver cirrhosis and end-stage liver disease, preventive programs and early management of these conditions are considered an emerging health issue. Cirrhosis is an immunocompromised state which predisposes the patient to a variety of infections. Once infection occurs, the pro-inflammatory cytokines and hemodynamic circulation derangement further facilitate the development of serious consequences of infections such as septic shock, multiple organ failure, and death. [9]

The burden of liver cirrhosis:

Global burden:

Liver cirrhosis has emerged as a major cause of global health burden. According to the Global Burden of Disease 2010 study, in 2010 liver cirrhosis ranked as the 23rd cause of disease burden worldwide causing31 million Disability Adjusted Life Years (DALYs), or 1.2% of global DALYs and one million deaths, or 2% of all deaths worldwide in that year. [16, 17] with nearly equal proportions attributable to hepatitis B, hepatitis C and alcohol consumption. [16]Regionally, liver cirrhosis is a health priority in Central Asia (ranked 9th among the leading causes of disease burden in 2010), Central Europe (rank 10), Eastern Europe (rank 11) and Central Latin America (rank 12). Global liver cirrhosis deaths increased monotonically from just over 676,000 (676,079:95% uncertainty interval: 452,863 to 1,004,530) deaths in 1980, or 1.54% of global deaths, to more than one million (1,029,042: 670,216 to

1,554,530) deaths in 2010, or 1.95% of the global total. On average, there were twice as many liver cirrhosis male deaths as female deaths. In terms of age-standardized mortality rates, liver cirrhosis decreased globally from 20.0 (95% uncertainty interval; 13.5 to 29.4) deaths per 100,000 person-years in 1980 to 15.8 (10.2 to 23.6) deaths per 100,000 person-years in 2010, a 21.6% reduction. This was largely driven by countries in East Asia b (21.2 to 8.2 deaths per 100,000; a 61.3% reduction), North Africa/Middle East (28.7 to 20.2 deaths per 100,000; a 29.6% reduction) and high-income Asia Pacific (21.2 to 10.3 deaths per 100,000; a 51.5% reduction). This trend was offset by an increase in the age-standardized liver cirrhosis mortality rate in South Asia (18.8 to 21.3 deaths per 100,000; a 12.8% increase), Central Asia (26.3 to 33.7 deaths per 100,000; a 28.4% increase) and Eastern Europe (12.6 to 20.0 deaths per 100,000; a 58.5% increase. [18]

Liver cirrhosis-Burden in India:

South Asia, particularly India, is another region where priority attention to improving prevention and control of liver cirrhosis risk factors is needed, with almost one fifth (18.3%) of global liver cirrhosis deaths in 2010 occurring in India alone. Cirrhosis mortality has been steadily increasing in India since 1980, as has alcohol consumption, a prevalence of hepatitis B and C and diabetes (a major risk factor for nonalcoholic fatty liver disease (NAFLD)). In 2010, there were an estimated 188,575 (109,748 to 303,989) liver cirrhosis deaths in India, accounting for almost one-fifth (18.3%) of the global liver cirrhosis death toll.[19]

Immune dysfunction in Cirrhosis:

Cirrhosis is the final stage of chronic liver diseases from any cause and is associated with various levels of immune dysfunction, which are referred to as cirrhosis associated immune dysfunction syndrome (CAIDS). [20] Acquired alterations

of both the innate and the adaptive immune functions are diverse, encompassing recognition, effector, and regulatory mechanisms. [8] Paradoxically, depression and overstimulation exist concurrently in the system, and result in an enhanced susceptibility to acute inflammatory processes and their exaggerated courses, both locally and far from the portal of entry of the microbes or the non-microbial toxic agents. The worst consequence of the imbalance in the pro- and anti-inflammatory processes is the development of acute-on-chronic liver failure (ACLF). Subtle immune dysfunction, however, also favors a shift towards persistence of inflammation leading to progression of liver fibrosis and development of different complications (portal hypertension and hepatic encephalopathy). From a pathogenetic point of view, the predominant mechanisms are different during acute and chronic worsening of liver function in cirrhosis.[21] Enhanced bacterial translocation (BT) [22] associated with systemic endotoxemia and increased occurrence of systemic bacterial infections have substantial impacts on both clinical situations. [23] The other important feature is that the immune status of patients is not constant during the illness, and the extent of the acquired immune dysfunction is related to the severity and etiology of the liver disease. The more severe the liver disease, the more subtle is the immune dysfunction. [9]In the case of an alcoholic etiology, more profound alterations are generally expected.[24]Lastly, in cirrhosis, the clinical effect of functional variations of innate immunity-related genes are more pronounced compared to non-cirrhotic cases because of a pre-existing acquired immune dysfunction with limited compensatory mechanisms. [24]

Systemic inflammation and an impaired immune response may not be mutually exclusive but operate simultaneously in decompensated cirrhosis and ACLF. Recently, a theory encompassing both features denominated cirrhosis-associated

immune dysfunction (CAID) has been proposed. This syndrome describes excessive systemic inflammation as an initial phenotype in compensated cirrhosis, caused by persistent immune cell stimulation by bacterial and bacterial products translocation; under this constant stimulus, the immune response system eventually becomes exhausted and switches to an ‘‘immunodeficient” phenotype in late stages of decompensated cirrhosis, such as ACLF. [25] Even though there is plentiful evidence of the role of immune dysfunction in the pathogenesis in cirrhosis, the identification of objective, reproducible and readily-available surrogate biomarkers of this syndrome is still a work in progress. Several features have been analyzed, such as leukocyte count, procalcitonin, and C-reactive protein; however, no clear cut-off points or extensive validation has been achieved so far. [26] Cytokines have also been proposed as prognostic tools in various stages of cirrhosis. In stable cirrhotic patients, they have been evaluated as tools in the detection of clinically-significant portal hypertension and even as independent factors related to mortality in decompensated cirrhosis. [27]

Innate immune dysfunction

Cirrhosis affects innate immunity by impairing the synthesis and function of PRRs and various proteins, thus reducing the bactericidal capacity of that body. [28]Different PRRs recognize different PAMPs. Among the PRRs, TLRs are most extensively studied and have a major influence in CAID. [29, 30] TLRs have an important role to play in the pathogenesis of various hepatic disorders, such as non-alcoholic fatty liver disease, alcoholic liver disease, viral hepatitis, autoimmune liver disease, hepatic fibrosis and liver cancer. [31]Acquired alteration of TLRs and their signaling pathways are a major mechanism of innate immunity dysfunction in cirrhosis. [30] This phenomenon may be due to prolonged exposure to bacteria, its products, and PAMPs, because of the loss of barrier function related to loss of tight

junctions, widening of intercellular spaces (which increases the gut translocation), the presence of toxic agents (i.e. ethanol) and by their production by damaged hepatocytes. [32] Gut flora can promote alcoholic

liver disease by activating TLRs but this process can be reduced by altering the gut microbiota by administration of antibiotics and probiotics.[33]

Adaptive immune dysfunction

Adaptive immune dysfunction is also common in cirrhotic patients. The various defects in B and T cell functions in alcoholic liver disease have been known for a long time.[34]

Risk factors for infection in Cirrhosis:

Infection is present at admission or develops during hospitalization in around 25%-30%. Bacterial infection is not only more frequent but also more severe in cirrhosis, causing a four-fold increase in the probability of death, reaching 38% at 1 mo.[35] Infection can accentuate circulatory dysfunction leading to the development of hepatorenal syndrome (HRS) and can also induce an excessive pro-inflammatory response that could contribute to the development of sepsis-related organ failure (acute-on-chronic liver failure) and septic shock. [36]

Bacterial infection is responsible for approximately 30%-50% of deaths in cirrhotic patients. [9]Compared to a 5%-7% infection rate reported in hospitalized patients in general, those hospitalized with cirrhosis have an infection rate of 32%-34% [37] and which may be up to 45% in those with gastrointestinal bleeding. The most common bacterial infections are spontaneous bacterial peritonitis (SBP) (25%-31%), urinary tract infection (UTI) (20%-25%), pneumonia (15%-21%), bacteremia (12%) and soft tissue infection (11%). [38] Approximately 75% of bacterial infections in patients with cirrhosis is caused by gram-negative bacteria, e.g. *Escherichia coli,*

Klebsiella spp., *Enterobacter spp.*, *P. aeruginosa, Vibrio spp.*, *Aeromonas spp.*, whereas gram-positive comprise 20.2% and anaerobes only 3.2%. [39]

Bacterial infection in cirrhotic patients is associated with poor clinical outcomes (up to 4-fold mortality). The mortality rate of sepsis in cirrhotic patients is approximately 26%-44%. A recent analytical review of 11987 cirrhotic patients suggested several clinical predictors of death after infection, such as advanced liver disease, the presence of shock and/or organ failure (particularly kidneys), gastrointestinal bleeding, encephalopathy, hepatocellular carcinoma and nosocomial acquisition. Patients who survived a significant episode of infection are still at high risk of death (up to 30%) within 1 year. [35] Acute renal dysfunction following infections has been observed in 27%-34% of patients with advanced cirrhosis. Thus, it is a strong independent risk of death in these patients with a 40%-50% mortality rate.[35] Several risk factors for the development of renal failure in cirrhotic patients with bacterial infections include advanced liver disease, pre-existing renal insuffi-ciency, inadequate circulatory volume, low baseline cardiac output, lack of resolution of infection and not receiving early albumin infusion. Renal failure that does not respond to albumin infusion in the setting of bacterial infection without septic shock was recently considered hepatorenal syndrome (HRS). Sepsis-related renal failure and HRS can persistently progress despite the resolution of infection, thus needing further special interventions.[40, 41]

Bacterial infections can precipitate a rapid deterioration of liver functions and encephalopathy which is associated with poor short-term prognosis.[9]Pulmonary complications are increasingly common in cirrhotic patients. Acute respiratory distress syndrome may develop because of exaggerated systemic inflammatory response syndrome in severe sepsis which leads to higher mortality. Aspiration is

common in encephalopathic patients. Prognosis of cirrhotic patients who were intubated was dismal, with a 33%-60% mortality rate.[42]

The effects of sepsis on coagulation cascades are more complex in cirrhosis. Patients with advanced cirrhosis are associated with thrombocytopenia and low clotting factors (e.g. factor , , , and prothrombin). The consumption of coagulation factors and the enhanced fibrinolytic activity by sepsis-induced inflammatory cytokines leads to a further worsening of pre-existing coagulation and platelet abnormalities. [43] Presence of bacterial infection in patients with variceal bleeding is independently associated with failure to control and early recurrent bleeding. Antibiotic prophylaxis in cirrhotic patients with variceal hemorrhage decreases infections, rebleeding and mortality.[44, 45]

UTI is the second most common bacterial infection in cirrhosis after SBP.[38] In cirrhotic patients, the prevalence of bacteriuria is 16%-18%, which is twice as frequent as matched controls.[46] Compared to non-cirrhotic, cirrhotic patients with community-acquired pneumonia are more frequently associated with bacteremia, multi-lobar involvement, impaired consciousness, renal failure, septic shock, and death (overall mortality 7.4% *vs* 14.4%, P <0.024). [47] Bacteremia without particular organ-specific source is increasingly common in cirrhosis and can be arbitrarily divided into 2 entities

(1) primary or spontaneous bacteremiaand

(2) secondary bacteremia.

True primary bacteremia shares the same initial step of pathogenesis as SBP, whereby bacteria flora in the gut lumen translocate into the bloodstream

Spontaneous bacterial empyema, Skin, and soft tissue infection, Endocarditis, Meningitis are other common infections in cirrhotic patients.[48]

The diagnosis of liver cirrhosis

Cirrhosis is histologically characterized by fibrous septa between the portal fields; it comes in micro- and macronodular forms.[11, 49] The condition is diagnosed by its characteristic findings on clinical examination, laboratory tests. The typical findings in cirrhosis include

- cutaneous signs of liver disease,
- a firm liver on palpation, and
- certain risk constellations such as:
  - metabolic syndrome
  - heavy alcohol consumption
  - exposure to hepatotoxic substances
  - use of hepatotoxic medications.[11, 49]

The physical examination of a patient with cirrhosis may reveal abdominal swelling, muscle atrophy, spider angiomas, palmar erythema, and pinpoint bleeding in the skin (petechiae). The liver may feel lumpy (nodular) and an enlarged spleen may be present. Males can have testicular atrophy and excessive breast tissue (gynecomastia), while women may experience an absence of menstruation (amenorrhea).

Laboratory tests can be unremarkable or show an elevated bilirubin level, low levels of a protein called albumin, thrombocytopenia, and impaired clotting as evidenced by an elevated prothrombin time. Further testing with ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) detects the presence of a cirrhotic liver; however, a liver biopsy remains the diagnostic gold standard.[11, 49]

Prevention and Treatment:

Cirrhosis is the end stage of chronic liver diseases that progress over years or decades. It can thus be prevented by appropriate screening for chronic liver diseases so that they can be treated in time. Chronic liver diseases usually asymptomatic. Measuring the serum concentrations of alanine aminotransferase (ALT—an indicator of hepatic inflammation) and gamma-glutamyl transferase (GGT—an indicator of cholestasis and impaired hepatic metabolism) is indicated as a screening measure for patients initially presenting to a primary care physician, even if asymptomatic.

Treatment of the underlying disease can often halt or even reverse the progression of early-stage cirrhosis. Some examples of beneficial treatment approaches are:

- antiviral therapy in cirrhosis due to hepatitis B or C (24, 25, 33);
- immune suppression in autoimmune hepatitis (1);
- treatment of iron overload in hemochromatosis and copper overload in Wilson disease (34, 35);
- abstinence from alcohol in alcoholic cirrhosis (36).

In cirrhosis, the areas of scar tissue are permanently damaged and cannot be reversed, so treatment is aimed at correcting the underlying liver disease and avoiding liver toxins to prevent further damage. Alcohol should be avoided, and if hepatitis is present, an antiviral medication may be prescribed. Ascites is initially controlled with diuretic medications, which increase urination, such as spironolactone and furosemide. When diuretic medications fail, ascitic fluid can be directly withdrawn through a needle inserted into the peritoneal cavity (paracentesis).

Clearly, the etiology of cirrhosis in any particular case is highly relevant to the prognosis. Thus, appropriate screening should be performed (37).

Relevant studies:

In the year 1990 Aloy Duch, A., et al. have conducted a prospective study on Bacteremia in the patient with liver cirrhosis. “Totally 54 hepatic cirrhosis patients were involved in the study and evaluation of 61 episodes of bacteremia was done, Spontaneous bacteremia represented 46% of all episodes whereas the proportion of the urinary origin was 30%. In 71% of episodes, gram negative organisms were isolated, among them 43% of were hospital-acquired and 25% of patients had spontaneous peritonitis. Shock (28%), renal failure (24%), and disseminated intravascular coagulation (6%) were the reported other complications of bacteremia. The mortality rate was 28% due to sepsis, 20% was due to complications of cirrhosis by itself and that of nonrelated diseases was 8%. Shock and renal failure secondary to bacteremia were independent predictors of a poor prognosis”.[50]

Fernandez, J., et al.in 2002 have conducted a study on Bacterial infections in cirrhosis patients and the influence of invasive procedures and norfloxacin prophylaxis on epidemiological changes. “A total of 405 patients presented 572 bacterial infections in 507 admissions were selected as the study population. Spontaneous bacterial peritonitis was the most frequent infection (138 cases). Gram-positive cocci were responsible for 53% of total bacterial infections in the study, being the main bacteria isolated in nosocomial infections (59%). Patients requiring treatment in an intensive care unit and those submitted to invasive procedures presented a higher rate of infections caused by gram-positive cocci (77% vs. 48%, P <.001 and 58% vs. 40%, P <.02, respectively). 50% of culture-positive spontaneous bacterial peritonitis in patients on long-term norfloxacin administration (n = 93) and 16% in patients not receiving this therapy (n = 414) were caused by quinolone-resistant gram-negative bacilli, P =.01. The rate of culture-positive spontaneous

bacterial peritonitis caused by trimethoprim-sulfamethoxazole-resistant gram-negative bacilli was also very high in patients on long-term norfloxacin administration (44% vs. 18%, P =.09). Authors concluded that infections caused by gram-positive cocci have markedly increased in cirrhosis. This phenomenon may be related to the current high degree of the instrumentation of cirrhotic patients. Quinolone-resistant spontaneous bacterial peritonitis constitutes an emergent problem in patients on long-term norfloxacin prophylaxis, with trimethoprim-sulfamethoxazole not being a valid alternative."[38]

In 2004 Jarcuska, P., et al. studied on the incidence of infectious complications in liver cirrhosis, and also trying to find out the relation among stage of liver cirrhosis, a number of infectious complications and mortality in cirrhotic patients. "93 hospitalized cirrhotic patients were selected for the study . 6 patients were in class B, 87 in class C of Child-Pugh classification. Ascites was found in 81 patients; pleural effusion was found in 14 patients. Chest X-ray, examination of ascites and pleural effusion a urine culture were performed by admission, the other infections were actively screened for clinical signs. Spontaneous bacterial peritonitis was found in 17 patients (18,28 %), secondary bacterial peritonitis in 5 patients (5,38 %), spontaneous bacteremia in 3 patients (3,23 %), spontaneous bacterial pleuritis in 3 patients (3,23 %), bronchopneumonia and infections of respiratory tract in 22 patients (23,66 %), uro-infection in 69 patients (74,19 %) and the other kinds of infection in 14 patients (15,05 %). Mortality of patients correlates with stage of liver cirrhosis and number of infectious complications. As per study findings that Infections are common complications in hospitalized cirrhotics. Infectious complications are the most common cause of mortality of cirrhosis, patients with bronchopneumonia, secondary bacterial peritonitis or spontaneous bacterial pleuritis had the bad prognosis. Early

antibiotic treatment at the base of culture and sensitivity is an optimal therapeutic approach in cirrhotics with infections".[51]

In 2010, Arvaniti, V., et al. have demonstrated that in cirrhotic patients, infections increase mortality four-fold and should be used in determining prognosis. "Overall median mortality of infected patients was 38%: 30.3% at 1 month and 63% at 12 months. Pooled odds ratio for death of infected versus noninfected patients was 3.75 (95% confidence interval, 2.12-4.23). In 101 studies that reported spontaneous bacterial peritonitis (7062 patients), the median mortality was 43.7%: 31.5% at 1 month and 66.2% at 12 months. In 30 studies that reported bacteremia (1437 patients), the median mortality rate was 42.2%. Mortality before 2000 was 47.7% and after 2000 was 32.3% (P = .023); mortality was reduced only at 30 days after spontaneous bacterial peritonitis (49% vs 31.5%; P = .005). As per the study findings that in patients with cirrhosis, infections increase mortality 4-fold; 30% of patients die within 1 month after infection and another 30% die within 1 year."[35]

Shizuma, T. et al in 2012 have assessed the microorganisms involved and the outcome among liver cirrhosis patients with bacteremia, including spontaneous bacterial peritonitis. Totally 236 cirrhotic patients were recruited for the study and 30 patients diagnosed with spontaneous bacterial peritonitis. "The rate of positive blood culture was 37.1% (140/377), and the isolated microorganisms were predominantly Gram-negative bacteria. In patients with confirmed bacteremia, the Child-Pugh score and serum blood urea nitrogen and creatinine levels were significantly higher than in non-bacteremia cases. Moreover, short-term mortality (within 1 month) was 48.2% (53/110), being significantly higher than that among non-bacteremia cases (18.8%; 22/117). Among spontaneous bacterial peritonitis cases, mortality within one month was 33.3% (10/30). Again, the Child-Pugh score and serum blood urea nitrogen and

creatinine levels were significantly higher among the fatalities than among survivors. Conclusions of the study findings were stating that severity of liver dysfunction and severity of renal dysfunction are both important determinants of short-term mortality among liver cirrhosis patients with bacteremia and spontaneous bacterial peritonitis."[52]

Tandon, P., et al. (2012). have conducted a study on the greater prevalence of antibiotic-resistant bacterial infections among patients with cirrhosis. "Thirty percent of infections were nosocomial. Urinary tract infections (32%) and spontaneous bacterial peritonitis (24%) were most common. Of the 70 culture-positive infections, 33 (47%) were found to be antibiotic resistant (12 were vancomycin-resistant Enterococci, 9 were extended-spectrum-beta-lactamase-producing Enterobacteriaceae, 7 were quinolone-resistant gram-negative rods, and 5 were methicillin-resistant Staphylococcus aureus). Exposure to systemic antibiotics within 30 days before infection was associated independently with AR-BI, with an odds ratio (OR) of 13.5 (95% confidence interval [CI], 2.6-71.6). Exposure to only non-absorbed antibiotics (rifaximin) was not associated with AR-BI (OR, 0.4; 95% CI, 0.04-2.8). In a sensitivity analysis, exposure to systemic antibiotics within 30 days before infection and nosocomial infection was associated with AR-BI (OR, 5.2; 95% CI, 1.5-17.7; and OR, 4.2; 95% CI, 1.4-12.5, respectively). Study findings suggested that the prevalence of AR-BI is high. Exposure to systemic antibiotics within 30 days before infection (including those used for prophylaxis of spontaneous bacterial peritonitis), but not oral non-absorbed antibiotics, is associated with the development of an AR-BI."[53]

Bartoletti, M., et al.in 2014 have studied on epidemiology and outcomes of bloodstream infection in patients with cirrhosis. Authors evaluated retrospectively 162 BSI episodes in cirrhotic patients to describe the etiology and risk factors for 30-day

mortality. “BSI episodes were identified in 162 patients, including 29 mixed infections. Most of the episodes were hospital acquired or healthcare-associated (93%). Gram-negative bacteria (GNB), Gram-positive bacteria and Candida spp. caused 64%, 38%, and 10% of episodes, respectively. GNB were classified as multi-drug resistant (MDR) and extensively drug-resistant (XDR) in 25% and 21% of cases, respectively. The overall crude 30-day mortality rate was 29%. Four risk factors were independently associated with 30-day crude mortality: worsening of MELD score from baseline (the last MELD score available in the 2 weeks prior BSI) to that at BSI onset (HR 1.11 per point increase, 95% CI 1.07-1.15, p<0.0001), spontaneous bacterial peritonitis as BSI source (HR 4.42, 2.04-9.54, p=0.002), sepsis grading (HR 2.18, 1.39-3.43, p=0.0007), and inappropriate antibiotic therapy within 24h from blood cultures (HR 2.82, 1.50-5.41, p=0.002). Study findings confirm that an increasing proportion of BSIs in cirrhotic patients is caused by resistant GNB and Candida spp., accurate evaluation of risk factors for mortality may improve early appropriate therapeutic management.”[7]

In 2015 Merli, M., et al. have demonstrated the epidemiology, prevalence and risk factors of multi-resistant infections, and also failure rate of empirical antibiotic therapy in cirrhotic patients. “Among 111 patients, 124 (15% CA, 52% HA, 33% HCA) were reported. More common infections were urinary tract infections, pneumonia and spontaneous bacterial peritonitis. Gram-negative bacteria cause 47% of infections. 51% of the isolates were multi-resistant to antibiotic therapy (76% MDR, 21% XDR, 3% PDR): the use of antibiotic prophylaxis (OR = 8.4; 95%CI = 1.03-76; P = 0,05) and current/recent contact with the healthcare-system (OR = 3.7; 95%CI = 1.05-13; P = 0.04) were selected as independent predictors. The failure of the empirical antibiotic therapy was progressively more frequent according to the

degree of resistance. The therapy was inappropriate in the majority of HA and HCA infections. Authors concluded that in hospitalized cirrhotic patient's Multi-resistant infections are an increasing trend. To improve the efficacy of empirical antibiotic therapy needs A better knowledge of the epidemiological characteristics. The use of preventive measures aimed at reducing the spread of multi-resistant bacteria is also essential." [4]

Preveden, T. (2015). Have assessed the prevalence, localization, and etiology of bacterial infections in hospitalized patients with liver cirrhosis. The study population includes 401hospitalized liver cirrhosis patients. "The prevalence of bacterial infection was 38.15% (153/401). The most common infections were pneumonia (21.56%), urinary tract infection (20.91%), and spontaneous bacterial peritonitis (18.95%). Localization of infection remained undetermined in as many as 37 patients (24.18%). Bacterial cultures were positive in 32 patients (20.91%), Gram-negative bacteria were commonly isolated, mostly Escherichia coli (71.87%). The mortality rate among patients with bacterial infections was 31.37% (48/153). Study findings suggested that Bacterial infections are often found in patients with liver cirrhosis, the most frequent being pneumonia, urinary tract infection and spontaneous bacterial peritonitis. Gram-negative bacteria, especially Escherichia coli were predominant in the etiology." [3]

The population-based retrospective study was conducted by Sargent, K., et al. in 2015 on the impact of bacterial infections on the course of compensated and decompensated cirrhosis as well as the occurrence, predictors of infection-related acute-on-chronic liver failure (ACLF) and its fatal outcome are limited. "Totally, 398 serious bacterial infections were reported in 241/633 (38%) patients (106/332 diagnosed with compensated and 135/301 with decompensated disease; follow-up

time was 2276 patient-years). ACLF occurred in 95/398 (24%) serious infections with an in-hospital mortality of 50%. In logistic regression analysis, the model for end-stage liver disease score, active alcohol misuse, and healthcare-associated infections were predictors of infection-related ACLF ($p < 0.05$ for all). In-hospital mortality in infections with ACLF was related to albumin levels, Charlson comorbidity index >1 and occurrence of one or more organ failures ($p > 0.05$ for all). In Cox regression analysis, infection-related ACLF was an independent negative predictor of transplant-free survival in decompensated patients ($p = 0.049$). Population-based cirrhotic cohort concludes that infection-related ACLF was a negative predictor of survival in decompensated disease. Infection-related ACLF was frequent and related to cirrhosis severity and infection acquisition type, as well as to high inpatient mortality, particularly in patients with significant comorbidity."[54]

In 2015 Sargenti, K., et al. assessed the occurrence of healthcare-associated (HCA) and hospital-acquired (HA) bacterial infections in cirrhosis, their predictors, and their impact on outcome are limited. "A total of 398 serious infections occurred in 241/633 (38%) patients. Forty-seven percent were HCA and 21% HA. Proton pump inhibitor (PPI) use was more common in HA (80%) vs. HCA (64%) vs. community-acquired (44%) infections ($P < 0.001$). In regression analysis, decompensated status, use of antibiotics and PPIs at infection diagnosis were independent predictors of HCA/HA infections ($P < 0.05$). After adjustment for confounders, HCA/HA infections were significantly related to infection-related ACLF ($P < 0.05$), but not severe sepsis, AKI or infection-related mortality ($P > 0.05$). Antibiotic-resistant infections were more frequent among HA (17%) than HCA (6%) or community-acquired (8%) infections ($P < 0.05$). Antibiotic-resistant HCA/HA infections were independently related to severe sepsis ($P < 0.05$). Findings of the study confirm that

two-thirds of serious bacterial infections were HCA or HA among cirrhotic patients. Predictors of serious HCA/HA infections were decompensated liver disease, antibiotics, and PPIs and were associated with the development of ACLF. HA infections were mostly resistant to antibiotics, and contributed to the risk of severe sepsis."[55]

Klimova, K., et al .in 2016 have evaluated the proportion of patients who were infected by multi-resistant bacteria and their epidemiology, risk factors, and clinical impact. 294 cirrhotic patients were considered as study population and retrospectively evaluated. "310 microorganisms were isolated from 294 patients; 109 (35.2%) were Gram-positive, 167 (53.9%), Gram-negative, and 34, fungi (11%). The most frequently existing microbial agent was Escherichia coli (98 isolations). In 22.9% of cases, infections were community-acquired, healthcare-associated infections were seen in 38.1% cases whereas nosocomial in 39%. In multiresistant isolates (p=0.05) worse liver infections and septic shock were more predominant (p=0.05), and also higher intrahospital mortality was found (p=0.017). Previous hospital admission, antibiotic treatment 60 days before, nosocomial or healthcare-associated acquisition and bacterial isolation in control cultures were identified as possible risk factors for the development of multiresistant infection. As per the study findings that important changes have occurred in the microbiological spectrum of bacterial infections in patients with liver cirrhosis. Multi-resistant bacteria are associated with high morbidity and mortality, as well as the failure of traditional antibiotic treatment. Successful control of the infection requires an early identification of patients at risk."[56]

Recently in 2017, Salerno, F., et al. have conducted a multicenter prospective study to demonstrate the antibiotic susceptibility of bacteria isolated from infections

in cirrhotic patients. "308 cirrhotic patients were recruited as study participants and among them, 313 culture-positive infections (173 community-acquired [CA] and 140 hospital-acquired [HA]) were identified. Urinary tract infections, spontaneous bacterial peritonitis, and bacteremias were the most frequent.48% were Quinolone-resistant Gram-negative isolates,44% were extended-spectrum beta-lactamase producers and 9% were carbapenem-resistant. Among 83 culture-positive infections (27%), multidrug-resistant agents (MDRA) were isolated and prevalence was same between CA and HA infections. MDRA were identified in 17 of 37 patients on quinolone prophylaxis, and in 46 of 166 not on prophylaxis (45% vs 27%; P<.03). In 287 cases an empiric antibiotic therapy was undertaken, in 37 (12.9%) this therapy failed. The in-hospital mortality rate of this subset of patients was significantly higher compared to patients who received an effective broad(er)-spectrum therapy (P=.038). During a 3-month follow-up, 56/203 culture-positive patients (27.6%) died, 24/63 who have had MDRA-related infections (38%) and 32/140 who have had antibiotic-susceptible infections (22.8%) (P=.025). Multivariate analysis disclosed MDRA infection, age, hepatocellular carcinoma, bilirubin, international normalized ratio and the occurrence of portal hypertension-related complications independent predictors of death. As per the study findings cirrhotic patients were frequently infected by MDRA with severe prognosis, mainly in patients unresponsive to empiric antibiotic therapy.[57]

Studies from INDIA:

Bajaj, J. S., et al.in 2012 assessed the factors predisposing to infection-related mortality in hospitalized patients with cirrhosis. Study was a prospective, cohort study of cirrhosis patients who ever infected. "Totally 207 patients (55 years, 60% men, MELD 20) were included. Most first infections were HCA (71%), then nosocomial

(15%) and community-acquired (14%). Urinary tract infections (52%), spontaneous bacterial peritonitis (SBP, 23%) and spontaneous bacteremia (21%) formed the majority of the first infections. Second infections were seen in 50 (24%) patients and were largely preventable: respiratory, including aspiration (28%), urinary, including catheter-related (26%), fungal (14%), and Clostridium difficile (12%) infections. Forty-nine patients (23.6%) who died within 30 days had higher admission MELD (25 versus 18, P < 0.0001), lower serum albumin (2.4 g/dL versus 2.8 g/dL, P = 0.002), and second infections (49% versus 16%, P < 0.0001) but equivalent SOFA scores (9.2 versus 9.9, P = 0.86). The case-fatality rate was highest for C. difficile (40%), respiratory (37.5%), and spontaneous bacteremia (37%), and lowest for SBP (17%) and urinary infections (15%). The model for mortality included admission MELD (odds ratio [OR]: 1.12), heart rate (OR: 1.03) albumin (OR: 0.5), and second infection (OR: 4.42) as significant variables. Conclusions of the study were second infections which can be potentially preventable were acts as predictors of mortality independent of liver disease severity."[58]

A multicenter prospective study was conducted by Baijal, R., et al.in 2014 to evaluate epidemiology, risk factors, and clinical consequences of bacterial infections in cirrhotic patients. "Among 420 cirrhotic patients 106 (25 %) patients had infection. Infection rate among indoor patients was 37.5 % (92/245) and among outdoor patients was 8 % (14/175). Out of 106 patients, CA, HCA, and HA were seen in 19.8 %, 50 %, and 30.2 %, respectively. Spontaneous bacterial peritonitis (31.1 %), urinary tract infections (22.6 %), and pneumonia and cellulitis (11.3 % each) were common infections. Gram-negative bacteria (54 %) were more common than Gram-positive cocci (46 %). Multidrug-resistant (MDR) organisms were seen in 41.7 % of patients. Most of the MDR organisms were seen in HCA and HA patients. The degree of liver

impairment was significantly more severe in patients with infection. Independent predictor of infection was high Child-Turcott-Pugh (CTP) class (p = 0.006, Child B vs. A (odds ratio (OR) 3.04 95 % CI = 1.63 to 5.68) and Child C vs. A (OR 4.17 95 % CI = 2.12 to 8.19). Overall in-hospital mortality was 7.6 %. Patients with infection had increased mortality at 30-day follow up compared to those without infection (23.5 % vs. 2.2 %; p<0.001). Study findings concluded that In patients of liver cirrhosis mortality and morbidity were mainly caused by infections. The most frequent infections are HCA and HA. Infection predisposes to deterioration of liver function and increases mortality. Cirrhotic patients should be monitored closely for infections especially those with Child class B and C."[59]

MATERIALS & METHODS

Study design: The current study was a prospective observational study

Study setting: The study was conducted in the department of General medicine, KLES Dr. Prabhakar Kore Hospital, Belgaum

Study population: The study population was included Patients admitted in the wards and ICU of the study setting, with decompensated cirrhosis of liver

Inclusion criteria:

- All cirrhotic patients above 18 years
- Patients admitted to the wards and ICU

Exclusion criteria:

- Diabetes Mellitus.
- HIV patients
- Patients on immunosuppressive drugs.
- Patients on steroids.
- Patients with malignancy

STUDY PERIOD: The data collection for the study was done between 1st January 2016 to January 2017, for a period of 1 year.

SAMPLE SIZE: The sample size was calculated assuming the expected proportion any culture positive infection in the study population as 25% as per the study by

Baijal, R., et al.[59]. To detect this proportion with 9% precision and 95% confidence level, a total of 89 subjects will be required as per the below mentioned calculation.

$$n = \frac{Z^2 P(1-P)}{d^2}$$

Where n = Sample size

Z = Z statistic for a level of confidence= 1.96

P = Expected prevalence of proportion

(If the expected prevalence is 20%, then P = 0.25), and

d = Precision (If the precision is 5%, then d=0.09)

To account for a non-participation/ loss to follow up/ incomplete data on outcome parameters another 10% of the subjects were added to the calculated sample size, making the required sample size as 98. Hence it was decided to include not less than 98 subjects. The final study has included 100 subjects in the analysis.

Sampling method: All the study subjects satisfying the inclusion and exclusion criteria were included in the study consecutively by purposive sampling.

Study procedure: After obtaining the informed written consent, each participant was evaluated thoroughly by clinical history, physical examination. Basing on the clinical findings, appropriate body fluid (Serum, ascetic fluid, urine etc) were collected under aseptic precautions and sent to the laboratory under proper transport conditions. The patient was started on appropriate antibiotic therapy as per the hospital protocol, along with comprehensive management of the underlying disease. Antibiotic therapy was titrated basing on the culture and sensitivity report. The final diagnosis of infection was made based on the culture positivity.

Data collection tools: all the relevant parameters for the study were documented in a structured questionnaire, containing the following details

- Socio-demographic details
- Clinical history including risk factors for cirrhosis, clinical presentation
- Clinical examination findings
- Details of the culture status and the organism profile

Ethical considerations

Clearance was obtained from the institutional ethical committee. Written and informed consent was sought from the patients or their attendants. They were given the option of quitting from the study if so desired by them. No element of compulsion was exerted. All personal data was kept confidential.

Statistical Analysis:

Presence of any culture positive infection was considered as the primary outcome. The profile of infections, the culture status of the relevant specimens, the organism profile etc was considered as relevant variables for analysis.

Descriptive statistics: Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram, and box plots. IBM SPSS version 22 was used for statistical analysis.[60]

RESULTS

A total of 100 subjects were included in the analysis.

Table 1: Descriptive analysis for AGE in study population (N=100)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
AGE	52.85 ± 9.49	54.00	32.00	76.00	50.97	54.73

The mean age of study population was 52.85 ± 9.49 with the youngest persons aged 32 years and eldest persons were 76 years. (Table 1)

Table 2: Descriptive analysis of Age group in study population (N=100)

Age group	Frequency	Percentages
up to 39	10	10.00%
40-49	21	21.00%
50-59	47	47.00%
60-69	17	17.00%
70 and above	5	5.00%

Among the study population, the age group was up to 39 in 10(10%),40-49 years in 21(21%), 50-59 years in 47(47%), 60-69 years in 17(17%) and 70 and above years in 5(5%). (Table 2)

Figure 1: Pie chart of Age group distribution in study population (N=100)

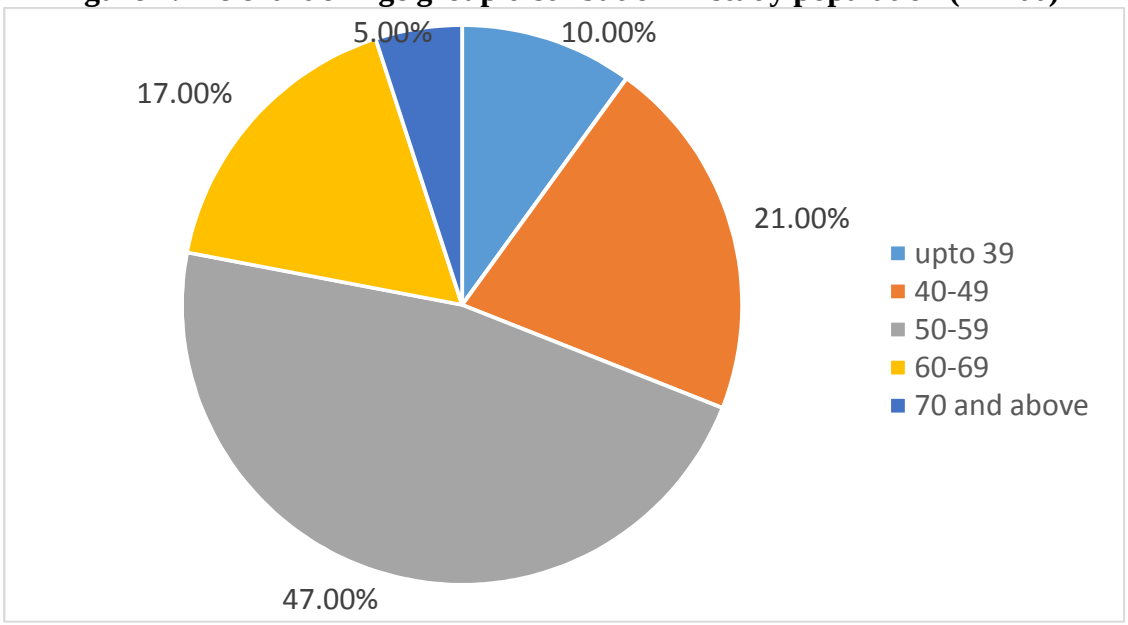

Table 3: Descriptive analysis of Gender in study population (N=100)

Gender	Frequency	Percentage
Male	75	75.00%
Female	25	25.00%

Among the study population, the number of males 75 (75%) was higher than females 25 (25%). (Table 2)

Figure 2: Bar chart of Gender distribution in study population (N=100)

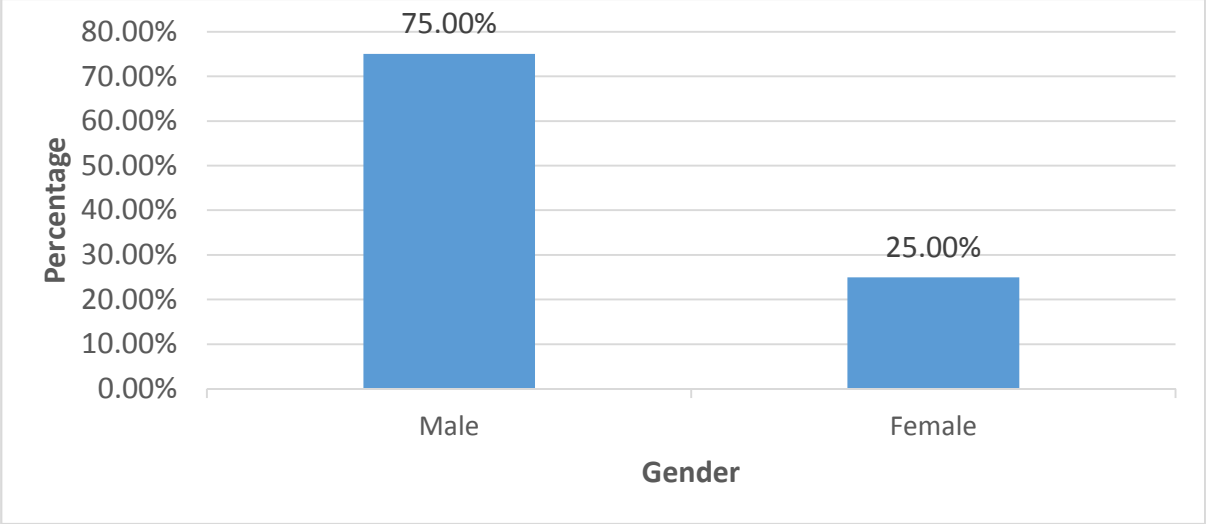

Table 4: Descriptive analysis for HB, TOTAL COUNT in study population (N= 100)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
HB	9.84 ± 2.26	9.90	4.80	15.60	9.39	10.29
Total count	10360 ± 8685.67	7300	1500	49900	8636.58	12083.42

The mean hemoglobin level was 9.84 ± 2.26, which was ranged between 4.80 and 15.60, The mean Total count was 10360±8685.67, which was ranging from 1500 to 49900, in the study population. (Table4).

Table 5: Summary of hemoglobin and WBC categories in the study population. (N=100)

Haemoglobin category	Frequency	Percentage
Below 13 gm/dl	93	93%
13and above	7	7%
TOTAL COUNT category		
up to 3999	16	16%
4000-9999	47	47%
10000 and above	37	37%

Among the study population, the HB was below in 93(93%), 13 and above in 7(7%) people. Among the study population, the total count was up to 3999 in 16(16%), 4000-9999 in 47(47%) and 10000 and above 37(37%). (Table 5)

Table 6: Descriptive analysis of Liver function parameters in study population (N=100)

Parameter	Mean ±STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
TOTAL BILIRUBIN	5.73 ± 4.94	4.64	0.46	30.63	4.75	6.72
DIRECT BILIRUBIN	3.74 ± 4.01	2.22	0.19	21.12	2.94	4.54
SGOT	117.73±118.58	76.00	18.00	626.00	94.08	141.38
SGPT	74.77 ± 65.67	45.00	10.00	343.00	61.67	87.87
ALKPHOSPHATE	125.89 ±50.25	111.00	19.00	291.00	115.87	135.91

The mean total bilirubin was 5.73 ± 4.94, with a minimum value of 0.46 and the maximum value of 30.63, in the study population. The mean direct bilirubin was 3.74 ± 4.01, with the minimum value of 0.19 and the maximum value of 21.12. The mean SGOT was 117.73±118.58 with a minimum value of 18 and the maximum value of 626. The mean SGPT was 74.77 ± 65.67. The minimum was 10 and maximum was 343 in the study population. The mean alkaline phosphatase was 125.89 ±50.25. The minimum was 19 and maximum was 291, in the study population. (Table6).

Table 7: Summary of elevated Liver function test parameters in study population (N=100)

TOTAL BILIRUBIN	Frequency	Percentage
0.00-0.29	19	19%
1.20 and above	81	81%
DIRECT BILIRUBIN		
0.00-0.29	1	1%
0.30 and above	99	99%
SGOT		
1-39	100	100%
SGPT		
1-40	43	43%
41 and above	57	57%
ALKPHOSPHATE		
Up to 39	1	1%
40-128	60	60%
129 and above	39	39%

Among the study population, total bilirubin was1.20 and above in 81(81%), direct bilirubin was 0.30 and above in 99(99%), SGPT was 41 and above in 57(57%) of the population. Alkaline phosphatase was 40-128 in 60(60%) participants and 129 or above in 39 939%) of participants. (table7)

Table 8: Descriptive analysis of HCV in study population (N=100)

HCV	Frequency	Percentage
Positive	22	22.00%
Negative	78	78.00%

Among the study population, the number of HCV positive people was 22(22%), and remaining 78(78%) were HCV negative. (Table 8)

Figure 3: Pie chart of HCV distribution in study population (N=100)

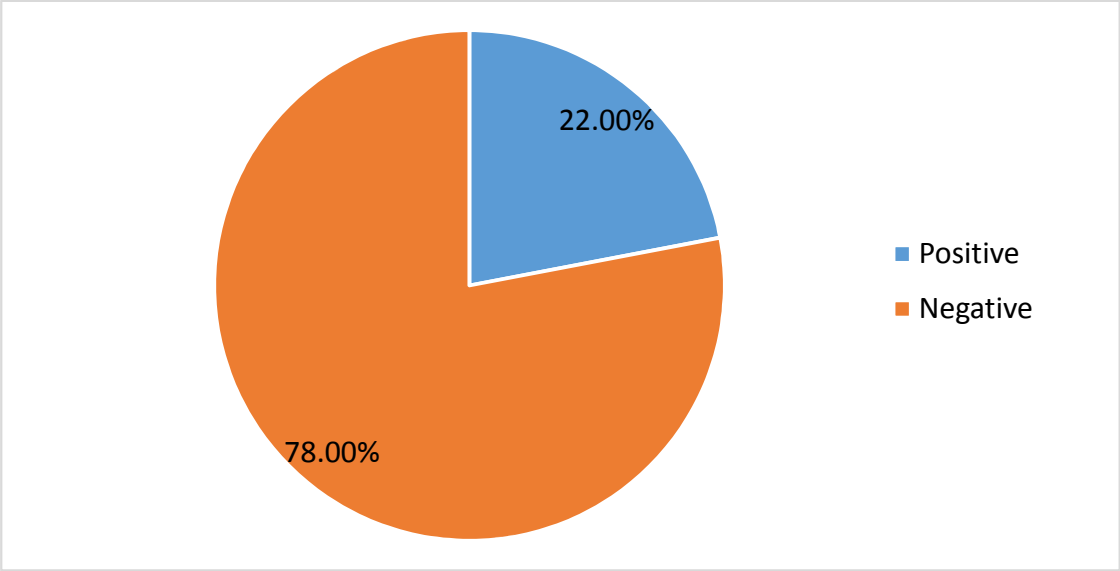

Table 9: Descriptive analysis of HBsAg in study population (N=100)

HBsAg	Frequency	Percentage
Positive	32	32.00%
Negative	68	68.00%

Among the study population, the number of people who were HBsAg positive was 32(32%), and 68(68%) were HBsAg negative. (Table9)

Figure 4: Bar chart of HBsAg distribution in study population (N=100)

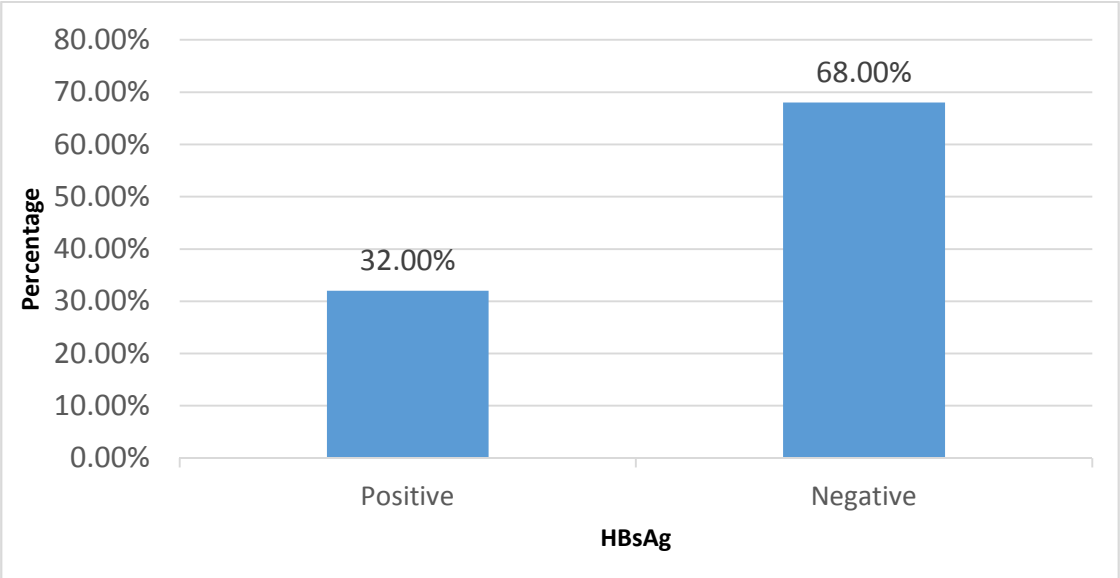

Table 10: Descriptive analysis of **alcohol history in study population (N=100)**

Alcohol history	Frequency	Percentages
NO	47	47%
Consuming for <15 YRS	4	4.0%
Consuming for >15 YRS	49	49.0%

Among the study population, 53% of the study population had reported alcoholism. Out of the 4 (4%), persons were consuming alcohol for the last 1o to 15 years and 49% were consuming it for more than 15 years. (Table 10)

Table 11: Descriptive analysis for Ascitic fluid Analysis in study population (N=100)

Ascetic fluid analysis	Mean ± STD	Median	Min	Max	95% C.I. for EXP(B)	
					Lower	Upper
Cell count	988.08 ± 86.14	800	120	11000	659.60	1316.56

The mean ascetic fluid cell count was 988.08±86.14 cells/ dl, with 120 minimum number of cells and 11,000 maximum number of cells. (Table 11)

Table 12: Categories of ascitic fluid cell count (N=100)

Ascetic fluid Analysis	Frequency	Percentage
upto250	8	8%
250and above	92	92%

Among the study population, the **ascitic** fluid cell count was up to 250 in 8(8%), 250and above in 92(92%) subjects. (Table 12)

Table 13: Descriptive analysis of ascitic fluid culture in study population (N=100)

Ascetic fluid culture	Frequency	Percentages
Positive	48	48.00%
No organism	52	52.00%

The ascites fluid culture was positive in 48% of the subjects.

Table 14: Organisms isolated in ascitic fluid culture (N=100)

organism isolated in ascetic fluid	Frequency	Percentages
E.coli	26	26.0%
Klebsiella pneumoniae	16	16.0%
Enterococcus gallinarum	4	4.0%
Acinetobacter iwofferi	2	2.0%
No organism	52	52.0%

E. coli was the most common organism identified in ascetic fluid culture in 26% of the subjects, followed by Klebsiella.Pneumoniae in 16% of the subjects. Enterococcus. Gallinarum was isolated in 4% of the subjects and Acinetobacter. Iwofferi was isolated in 2 % of the subjects.

Table 15: Urine culture status in study population (N=100)

URINE CULTURE	Frequency	Percentages
POSITIVE	16	16.0%
NO ORGANISM	84	84%

The urine culture was positive in 16% of the subjects**.** (table 15)

Table16: Organisms isolated in urine culture (N=100)

Urine culture positive	Frequency	Percentages
E.coli	12	12.0%
Enterobacter Species	2	2.0%
Klebsiella Pneumonia	2	2.0%
No Organism	84	84%

E. Coli was the most common organism identified in urine culture in 12% of the subjects. Enterobacter species and Klebsiella Pneumonia were isolated from 2% of the subjects each. (Table 16)

Table 17: Blood culture positivity in study population (N=100)

Blood Culture	Frequency	Percentages
Positive	39	39.0%
No organism	61	49.0%

Blood culture was positive in 39% of the subjects. (Table 17)

Table 18: Organisms isolated in blood culture in study population (N=100)

BLOODCULTUREPOSITIVE	Frequency	Percentages
Klebsiella Pneumoniae	15	15.0%
Staphylococcus Aureus	8	8.0%
E.coli	6	6.0%
Coagulase negative staphylococcus species	4	4.0%
Enterobacter species(pus)	2	2.0%
Enterococcus Gallinarum	2	2.0%
Staphylococcus Hemolyticus	2	2.0%
No organism	61	61%

The most common organisms identified in blood culture were Klebsiella Pneumoniae in 15%, followed by Staphylococcus Aureus in 8% subjects. coagulase negative staphylococcus species in 4% subjects, E. coli in 6% of the subjects. The other organisms isolated were Enterobacter Species (Pus), Enterococcus Gallinarum and Staphylococcus Hemolyticus in 2 subjects each. (Table 18)

Table 19: Descriptive analysis of profile of infection in study population (N=100)

Nature of infection	Frequency	Percentages
SBP	53	53%
Pneumonia	26	26%
UTI	16	16%
Cellulitis	5	5%

The most common infection identified was Spontaneous Bacterial Peritonitis (SBP) in 53% of the subjects, followed by UTI in 16% of the subjects, pneumonia in 26% of the subjects and cellulitis in 5% of the subjects. (Table 19)

DISCUSSION

Cirrhosis is an increasing cause of morbidity and mortality in more developed countries. It is the 14th most common cause of death in adults worldwide and it results in 1·03 million deaths per year worldwide.[18, 61] The main causes in more developed countries are infected with hepatitis C virus, alcohol misuse, and, increasingly, non-alcoholic liver disease; infection with hepatitis B virus is the most common cause in most parts of Asia. Bacterial infection is a severe and potentially fatal complication of the advanced liver disease. The immunological abnormalities occurring in cirrhosis, such as depressed reticuloendothelial system, neutrophil dysfunction, reduced serum complement and low bactericidal function, account for the increased susceptibility of cirrhotic patients to bacterial seeding and diffusion. In the last two decades, several studies carried out in cirrhosis have documented both a relevantly high prevalence of bacterial infection, ranging from 32 to 44%, as well as an increased mortality related to this complication. [6, 62, 63] The current study mainly aimed to demonstrate the clinical profile of infections in patients with liver cirrhosis.

In the current study totally 100 subjects were taken as study participants with mean age of 52.85 ± 9.49 years. Majority of study participants (47%) were distributed in 50-59 years age group. The proportion of males (75%) was higher than females (25%).Similar study settings were found in the studies conducted by Bajaj, J. S., et al. (2012)[58], Merli, M., et al. (2015)[4].In the study by Bajaj, J. S., et al. (2012)[58] recruited 207 study participants with mean age of 55± 9years and among 207, majority proportion were males (124). Fernandez, J., et al.[38] recruited totally 405 patients with mean age of 61±12 years. 251 patients were men. Mean HB level of our study population was 9.84±2.26 which is comparable with Hemang Suthar et al [64] which has 10.1 gm % and also with Sarin et al which has 10.2 gm% of mean haemoglobin level.

Mean total count reported in our study was 10360±8685.67 and is almost similar to other study findings by Hemang Suthar et al[64] which has 9521 mm3 mean total leukocyte count and also with Sarin et al which has reported as 9303.89 mm3. In 93% of study population HB was up to 13 and in 7% of subjects, it was reported above 13.In 47% of study participants, total count reported as 4000-9999, in 16% of subjects 3999 whereas in 37% of subjects reported total count was 10000.

In our study mean of TOTAL BILIRUBIN was 5.73 ± 4. The mean direct bilirubin was 3.74±4. Similar to our study mean total bilirubin was 3.17 ± 1.81 in Hemang Suthar et al.[64]

In current study results shows that The Mean **SGOT** was 117.73±118.58 which is comparable with Hemang Suthar et al [64], findings reported as 134.6 and also with Pathak et al (142.95 ± 159.85). Mean of SGPT in our study was 74.77 ± 65 and this value was almost similar to the findings of Hemang Suthar et al [64](56.1 IU/L) and also with Mendel hall et al (47-50 IU/L).

In present study mean alkaline phosphatase value was 125.89 ±50.25. Like current study findings, similar results were reported in Hemang Suthar et al,[64] (208 IU/L) and also comparable with Antonio Chedid study (163-219 IU/L)

In our study number of HCV positive cases were 22 (22%) and the HCV negative cases were 78 (78%). Like the current study Bajaj, J. S., et al. (2012)[58] reported 25% HCV positive cases.42% HCV positive cases were recorded in the study by Merli, M., et al. (2015).[4]. In the study of Fernandez, J., et al.[38] the cause of cirrhosis was hepatitis C virus (HCV) in 198 cases, HCV plus alcohol in 31% of patients. Kamani et al in 2008 [65] studied 187 cirrhotic patients with a most common etiological agent was HCV

139 (74.3%). Cruz Rde, C., et al. (2006).[66] conducted a study on 82 cirrhotic patients with a more predominant etiological agent was HCV in 45% of cases.

Among the study population, 53% of the study population had reported alcoholism. Out of them, 1 person was consuming it for less than 10 years, 3 (3%) persons were consuming alcohol for the last 10 to 15 years and 49% were consuming it for more than 15 years. In the study of Bajaj, J. S., et al. (2012)[58] 31% of participants were used to consume alcohol. Merli, M., et al. (2015).[4] the proportion of alcohol abusers were 23%.In the study of Fernandez, J., et al.(2002)[38] Alcoholism was found in 124 cases. Cruz Rde, C., et al. (2006).[66] reported 28% of alcohol abusers among 82 cirrhotic patients.

In the current study findings, in the study population, the proportion of subjects with positive ascitic fluid culture was 48%. The ascitic analysis was up to 250 in 8(8%), and 250and above in 92(92%).The proportion of subjects with positive ascitic fluid culture was 48%. Most common organism identified in Ascitic Fluid Culture was E. coli (26%).In 16% of the cases, Klebsiella Pneumoniae was identified. Enterococcus Gallinarum and Acinetobacter Iwofferi were positive in 4%, 2% of subjects respectively. A study conducted by Na, S. H., et al.[10] also documented similar to current study results among 533 patients 48.6% were ascitic fluid culture positive and comes under SBP group with the ascitic fluid PMN count was reported as >250/mm3. E. coli (39.4%, 102/259) was the most common pathogen isolated from ascitic fluid culture followed by K. pneumonia (17.8%, 46/259), and viridans streptococci (8.1%, 21/259). Kamani, L., et al.[65] reported results similar to current results as among 187 patients of AFI 44 (23.5%) had culture-positive ascitic fluid cultures i.e., SBP; Escherichia. Coli (E. Coli) being the most common organism isolated from 27 (61.3%) patients. Other organisms isolated were Streptococcus

pneumoniae in 5 (11.3%), Pseudomonas species in 4 (9%), Staphylococcus species in 3 (6.8%), Enterococcus species in 3 (6.8%), Bacillus species in 1 (2.2%), Group D Streptococcus in 1 (2.2%) person. Evans, L. T., et al. (2003) [67] have studied in 427 cirrhotic patients with ascitic fluid culture positive cases were 15 SBP patients. "Organisms grown from the ascitic fluid of patients with neutrocytic ascites included the following: Staphylococcus aureus (n = 1), Streptococcus viridans (n = 3), Staphylococcus saccharolyticus (n=1), and Bacteroides fragilis (n= 1). Organisms isolated from the ascitic fluid of patients diagnosed with bacterascites included the following: Staphylococcus aureus (n=2), Streptococcus viridans (n=1), coagulase-negative Staphylococcus (n =1), Propionibacterium (n = 3), and Pseudomonas luteola (n = 1)."

In our study, the proportion of Blood culture positive cases was 39%. Klebsiella Pneumoniae was present in 15% of subjects which is most common in blood culture. Other organisms identified were Staphylococcus Aureus in 8% subjects. coagulase negative Staphylococcus species in 4% subjects. E. coli in 6% of the subjects. Enterobacter Species (Pus), Enterococcus Gallinarum and Staphylococcus Hemolyticus were found in 2 subjects each. Like the current study Kamani, L., et al.[65] study findings were out of 187, 28 patients were blood culture positive(14.9%).E.coli was most commonly isolated from blood cultures 53.5%.A study by Na, S. H., et al. [10] recorded blood culture positive rate (38.1% versus 20.1%, p<.001) were higher in the SBP Group than CNNA.

E. Coli was the most common organism identified in urine culture in 12% of the subjects. Enterobacter species and Klebsiella Pneumoniae were isolated from 2% of the subjects each. Similar findings were found in Cruz Rde, C., et al. (2006).[66]'s study, in which among 82 study participants urine culture was positive in 4 (4.9%)

cases with increased leukocyte count. Among 4 cases E.Coli was found in 3 cases and Klebsiella pneumonia was isolated in 1 person. Similar to current study Borzio, M., et al. (2001)[37] have conducted a study on 132 patients and in 62 cases (41%) UTI was found. In 62 UTI cases, 41 (66%) were simple bacteriuria and 21 (34%) full urinary infections 43. Out of 62 UTI cases, the predominant isolated organism was E. coli followed by Staphylococcus Aureus was isolated from urine in 10 cases.

In our study, the most common infection identified was SBP in 53% of the subjects, followed by UTI in 16% of the subjects, pneumonia in 26% of the subjects and cellulitis in 5% of the subjects. Fernandez, J., et al.[38]'s study also reported like the current study the most common infection was SBP (138 infections), followed by urinary tract infection (111), pneumonia (78), and bacteremia associated with therapeutic invasive procedures and catheter sepsis (45). Other infections were culture-negative fever associated with leukocytosis (56), cellulitis (34), spontaneous bacteremia (28), cholangitis (21), secondary peritonitis (19), purulent bronchitis (16), spontaneous bacterial empyema (9), endocarditis (8), bacterascites in the setting of fever and leukocytosis(5), and gastroenteritis (4).In the study of Bajaj, J. S., et al. (2012)[58] The most common first infection was UTI (52 or 25%), followed by SBP (47 or 23%), spontaneous bacteremia (43 or 21%), skin (27 or 13%), and lower respiratory infections (16 or 8%).As per the study of Preveden, T.[3] The most common infections were pneumonia (21.56%), urinary tract infection (20.91%), and spontaneous bacterial peritonitis (18.95%). In the study of Tandon, P., et al. (2012)[53] 30%of infections were nosocomial. Urinary tract infections (32%) and spontaneous bacterial peritonitis (24%) were most common.

CONCLUSION

1. The mean age of study population was 52.85 ± 9.49 with a high male preponderance (75%) in study populations.
2. Elevated total bilirubin was seen in 81% in the study population. The proportion of the population with elevated direct bilirubin was seen 99%, elevated SGPT was seen in 57% and elevated alkaline phosphatase was seen in 39% of the population.
3. The proportion of participants with HCV positivity was 22% and HBsAg positivity was seen in 32% of the study population. The other common aetiology of cirrhosis was alcoholism in 53% of the study population.
4. The proportion of subjects with ascitic fluid cell count was > 250 in 92% of the subjects. The ascitic fluid culture was positive in 48% of the subjects. Among the organisms isolated in ascitic fluid E. coli was the most common one in 26% of the people. The other common organisms isolated in ascitic fluid were Klebsiella Pneumoniae, Enterococcus Gallinarum, and Acinetobacter Iwofferi.
5. The urine culture was positive in 16% of the subjects. E. coli was the most common organism isolated in urine culture in 12% of the subjects.
6. Blood culture was positive in 39% of the subjects. The most common organisms identified in blood culture was Klebsiella Pneumoniae in 15% of the subjects. The other common organisms isolated were Staphylococcus Aureus, Coagulase Negative Staphylococcus, E. coli.
7. The most common infection was Spontaneous Bacterial Peritonitis (SBP) in 53% of the subjects, followed by UTI in 16% of the subjects, pneumonia in 26% of the subjects and cellulitis in 5% of the subjects.

STRENGTHS:

The study has highlighted the profile of various infections in liver cirrhosis patients using objective diagnostic methods (Culture and sensitivity) of various body fluids.

LIMITATIONS:

Considering the descriptive nature of the study population, no analysis could be done to assess the factors associated with different infections in the study population.

RECOMMENDATIONS:

There is a need to conduct large-scale prospective studies on the subject to thoroughly understand the factors associated with the development of infections in liver cirrhosis patients. There is also a need to understand the impact of infection on the morbidity and mortality in these patients.

SUMMARY

Considering the rapidly changing profile of infections in liver cirrhosis, the current prospective observational study was planned to assess the incidence and clinical profile of infections in cirrhotic patients, in a tertiary care teaching hospital, between January 2016 to January 2017. The most common infection was SBP in 53%, followed by UTI in 16%, pneumonia in 26% and cellulitis in 5% of the subjects. Ascitic fluid culture was positive in 48%, urine culture was positive in 16%, blood culture was positive in 39% of the subjects. E. coli and Klebsiella Pneumoniae were the most common organisms isolated.

BIBLIOGRAPHY

1. Jalan R, Fernandez J, Wiest R, Schnabl B, Moreau R, Angeli P, et al. Bacterial infections in cirrhosis: a position statement based on the EASL Special Conference 2013. J Hepatol. 2014;60(6):1310-24.
2. Bunchorntavakul C, Chamroonkul N, Chavalitdhamrong D. Bacterial infections in cirrhosis: A critical review and practical guidance. World Journal of Hepatology. 2016;8(6):307-21.
3. Preveden T. BACTERIAL INFECTIONS IN PATIENTS WITH LIVER CIRRHOSIS. Med Pregl. 2015;68(5-6):187-91.
4. Merli M, Lucidi C, Di Gregorio V, Falcone M, Giannelli V, Lattanzi B, et al. The spread of multi drug resistant infections is leading to an increase in the empirical antibiotic treatment failure in cirrhosis: a prospective survey. PLoS One. 2015;10(5):e0127448.
5. Acevedo J. Multiresistant bacterial infections in liver cirrhosis: Clinical impact and new empirical antibiotic treatment policies. World J Hepatol. 2015;7(7):916-21.
6. Acevedo J, Fernandez J. New determinants of prognosis in bacterial infections in cirrhosis. World J Gastroenterol. 2014;20(23):7252-9.
7. Bartoletti M, Giannella M, Caraceni P, Domenicali M, Ambretti S, Tedeschi S, et al. Epidemiology and outcomes of bloodstream infection in patients with cirrhosis. J Hepatol. 2014;61(1):51-8.
8. Christou L, Pappas G, Falagas ME. Bacterial infection-related morbidity and mortality in cirrhosis. Am J Gastroenterol. 2007;102(7):1510-7.
9. Tandon P, Garcia-Tsao G. Bacterial infections, sepsis, and multiorgan failure in cirrhosis. Semin Liver Dis. 2008;28(1):26-42.

10. Na SH, Kim EJ, Nam EY, Song KH, Choe PG, Park WB, et al. Comparison of clinical characteristics and outcomes of spontaneous bacterial peritonitis and culture negative neutrocytic ascites. Scand J Gastroenterol. 2017;52(2):199-203.
11. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet. 2008;371(9615):838-51.
12. Gines P, Quintero E, Arroyo V, Teres J, Bruguera M, Rimola A, et al. Compensated cirrhosis: natural history and prognostic factors. Hepatology. 1987;7(1):122-8.
13. Garcia-Tsao G, Friedman S, Iredale J, Pinzani M. Now there are many (stages) where before there was one: In search of a pathophysiological classification of cirrhosis. Hepatology. 2010;51(4):1445-9.
14. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol. 2006;44(1):217-31.
15. Asrani SK, O'Leary JG. Acute-on-chronic liver failure. Clin Liver Dis. 2014;18(3):561-74.
16. Murray CJ, Vos T, Lozano R, Naghavi M, Flaxman AD, Michaud C, et al. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2197-223.
17. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2095-128.

18. Mokdad AA, Lopez AD, Shahraz S, Lozano R, Mokdad AH, Stanaway J, et al. Liver cirrhosis mortality in 187 countries between 1980 and 2010: a systematic analysis. BMC Medicine. 2014;12:145.
19. Tanaka H, Tsukuma H, Yamano H, Oshima A, Shibata H. Prospective study on the risk of hepatocellular carcinoma among hepatitis C virus-positive blood donors focusing on demographic factors, alanine aminotransferase level at donation and interaction with hepatitis B virus. Int J Cancer. 2004;112(6):1075-80.
20. Bonnel AR, Bunchorntavakul C, Reddy KR. Immune dysfunction and infections in patients with cirrhosis. Clin Gastroenterol Hepatol. 2011;9(9):727-38.
21. Jalan R, Gines P, Olson JC, Mookerjee RP, Moreau R, Garcia-Tsao G, et al. Acute-on chronic liver failure. J Hepatol. 2012;57(6):1336-48.
22. Wiest R, Lawson M, Geuking M. Pathological bacterial translocation in liver cirrhosis. J Hepatol. 2014;60(1):197-209.
23. Thalheimer U, Triantos CK, Samonakis DN, Patch D, Burroughs AK. Infection, coagulation, and variceal bleeding in cirrhosis. Gut. 2005;54(4):556-63.
24. Duddempudi AT. Immunology in alcoholic liver disease. Clin Liver Dis. 2012;16(4):687-98.
25. Albillos A, Lario M, Alvarez-Mon M. Cirrhosis-associated immune dysfunction: distinctive features and clinical relevance. J Hepatol. 2014;61(6):1385-96.
26. Dirchwolf M, Ruf AE. Role of systemic inflammation in cirrhosis: From pathogenesis to prognosis. World J Hepatol. 2015;7(16):1974-81.
27. Giron-Gonzalez JA, Martinez-Sierra C, Rodriguez-Ramos C, Macias MA, Rendon P, Diaz F, et al. Implication of inflammation-related cytokines in the natural history of liver cirrhosis. Liver Int. 2004;24(5):437-45.

28. Sipeki N, Antal-Szalmas P, Lakatos PL, Papp M. Immune dysfunction in cirrhosis. World J Gastroenterol. 2014;20(10):2564-77.
29. Broering R, Lu M, Schlaak JF. Role of Toll-like receptors in liver health and disease. Clin Sci (Lond). 2011;121(10):415-26.
30. Aoyama T, Paik YH, Seki E. Toll-like receptor signaling and liver fibrosis. Gastroenterol Res Pract. 2010;2010.
31. Szabo G, Dolganiuc A, Mandrekar P. Pattern recognition receptors: a contemporary view on liver diseases. Hepatology. 2006;44(2):287-98.
32. Li L, Chen L, Hu L, Liu Y, Sun HY, Tang J, et al. Nuclear factor high-mobility group box1 mediating the activation of Toll-like receptor 4 signaling in hepatocytes in the early stage of nonalcoholic fatty liver disease in mice. Hepatology. 2011;54(5):1620-30.
33. Llorente C, Schnabl B. The gut microbiota and liver disease. Cell Mol Gastroenterol Hepatol. 2015;1(3):275-84.
34. Noor MT, Manoria P. Immune Dysfunction in Cirrhosis. J Clin Transl Hepatol. 2017;5(1):50-8.
35. Arvaniti V, D'Amico G, Fede G, Manousou P, Tsochatzis E, Pleguezuelo M, et al. Infections in patients with cirrhosis increase mortality four-fold and should be used in determining prognosis. Gastroenterology. 2010;139(4):1246-56, 56.e1-5.
36. Foreman MG, Mannino DM, Moss M. Cirrhosis as a risk factor for sepsis and death: analysis of the National Hospital Discharge Survey. Chest. 2003;124(3):1016-20.
37. Borzio M, Salerno F, Piantoni L, Cazzaniga M, Angeli P, Bissoli F, et al. Bacterial infection in patients with advanced cirrhosis: a multicentre prospective study. Dig Liver Dis. 2001;33(1):41-8.

38. Fernandez J, Navasa M, Gomez J, Colmenero J, Vila J, Arroyo V, et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. Hepatology. 2002;35(1):140-8.
39. Brann OS. Infectious complications of cirrhosis. Curr Gastroenterol Rep. 2001;3(4):285-92.
40. Sort P, Navasa M, Arroyo V, Aldeguer X, Planas R, Ruiz-del-Arbol L, et al. Effect of intravenous albumin on renal impairment and mortality in patients with cirrhosis and spontaneous bacterial peritonitis. N Engl J Med. 1999;341(6):403-9.
41. Fasolato S, Angeli P, Dallagnese L, Maresio G, Zola E, Mazza E, et al. Renal failure and bacterial infections in patients with cirrhosis: epidemiology and clinical features. Hepatology. 2007;45(1):223-9.
42. Thomson SJ, Moran C, Cowan ML, Musa S, Beale R, Treacher D, et al. Outcomes of critically ill patients with cirrhosis admitted to intensive care: an important perspective from the non-transplant setting. Aliment Pharmacol Ther. 2010;32(2):233-43.
43. Plessier A, Denninger MH, Consigny Y, Pessione F, Francoz C, Durand F, et al. Coagulation disorders in patients with cirrhosis and severe sepsis. Liver Int. 2003;23(6):440-8.
44. Goulis J, Armonis A, Patch D, Sabin C, Greenslade L, Burroughs AK. Bacterial infection is independently associated with failure to control bleeding in cirrhotic patients with gastrointestinal hemorrhage. Hepatology. 1998;27(5):1207-12.
45. Chavez-Tapia NC, Barrientos-Gutierrez T, Tellez-Avila FI, Soares-Weiser K, Uribe M. Antibiotic prophylaxis for cirrhotic patients with upper gastrointestinal bleeding. Cochrane Database Syst Rev. 2010(9):Cd002907.

46. Cadranel JF, Denis J, Pauwels A, Barbare JC, Eugene C, di Martino V, et al. Prevalence and risk factors of bacteriuria in cirrhotic patients: a prospective case-control multicenter study in 244 patients. J Hepatol. 1999;31(3):464-8.
47. Viasus D, Garcia-Vidal C, Castellote J, Adamuz J, Verdaguer R, Dorca J, et al. Community-acquired pneumonia in patients with liver cirrhosis: clinical features, outcomes, and usefulness of severity scores. Medicine (Baltimore). 2011;90(2):110-8.
48. Bunchorntavakul C, Chavalitdhamrong D. Bacterial infections other than spontaneous bacterial peritonitis in cirrhosis. World J Hepatol. 2012;4(5):158-68.
49. Berg T. Diagnostik bei erhöhten Leberwerten. Der Gastroenterologe. 2009;4(6):557.
50. Aloy Duch A, Espejo Arenas E, Mauri Pont M, Garcia Restoy E, Simo Sanahuja M, Bella Cueto F. [Bacteremia in the patient with liver cirrhosis. Prospective study of 61 episodes]. Enferm Infecc Microbiol Clin. 1990;8(9):540-3.
51. Jarcuska P, Veseliny E, Orolin M, Takacova V, Hancova M. [Infectious complications in patients with liver cirrhosis]. Klin Mikrobiol Infekc Lek. 2004;10(4):176-80.
52. Shizuma T, Fukuyama N. Investigation into bacteremia and spontaneous bacterial peritonitis in patients with liver cirrhosis in Japan. Turk J Gastroenterol. 2012;23(2):122-6.
53. Tandon P, Delisle A, Topal JE, Garcia-Tsao G. High prevalence of antibiotic-resistant bacterial infections among patients with cirrhosis at a US liver center. Clin Gastroenterol Hepatol. 2012;10(11):1291-8.
54. Sargenti K, Prytz H, Nilsson E, Kalaitzakis E. Predictors of mortality among patients with compensated and decompensated liver cirrhosis: the role of bacterial

infections and infection-related acute-on-chronic liver failure. Scand J Gastroenterol. 2015;50(7):875-83.

55. Sargenti K, Prytz H, Strand A, Nilsson E, Kalaitzakis E. Healthcare-associated and nosocomial bacterial infections in cirrhosis: predictors and impact on outcome. Liver Int. 2015;35(2):391-400.
56. Klimova K, Padilla C, Avila JC, Clemente G, Ochoa A. Epidemiology of bacterial infections in patients with liver cirrhosis. Experience in a Spanish tertiary health center. Biomedica. 2016;36(1):121-32.
57. Salerno F, Borzio M, Pedicino C, Simonetti R, Rossini A, Boccia S, et al. The impact of infection by multidrug-resistant agents in patients with cirrhosis. A multicenter prospective study. Liver Int. 2017;37(1):71-9.
58. Bajaj JS, O'Leary JG, Reddy KR, Wong F, Olson JC, Subramanian RM, et al. Second infections independently increase mortality in hospitalized patients with cirrhosis: the North American consortium for the study of end-stage liver disease (NACSELD) experience. Hepatology. 2012;56(6):2328-35.
59. Baijal R, Amarapurkar D, Praveen Kumar HR, Kulkarni S, Shah N, Doshi S, et al. A multicenter prospective study of infections related morbidity and mortality in cirrhosis of liver. Indian J Gastroenterol. 2014;33(4):336-42.
60. Machines IB. IBM SPSS Statistics for Windows, Version 22.0. IBM Corp Armonk, NY; 2013.
61. Bleichner G, Boulanger R, Squara P, Sollet JP, Parent A. Frequency of infections in cirrhotic patients presenting with acute gastrointestinal haemorrhage. Br J Surg. 1986;73(9):724-6.
62. Bruns T, Zimmermann HW, Stallmach A. Risk factors and outcome of bacterial infections in cirrhosis. World J Gastroenterol. 2014;20(10):2542-54.

63. Strauss E. The impact of bacterial infections on survival of patients with decompensated cirrhosis. Ann Hepatol. 2013;13(1):7-19.
64. Suthar HN SK, Mewada, BN. Clinical profile of cases of alcoholic liver disease. Int J Med Sci Public Health. 2013;2:394-8.
65. Kamani L, Mumtaz K, Ahmed US, Ali AW, Jafri W. Outcomes in culture positive and culture negative ascitic fluid infection in patients with viral cirrhosis: cohort study. BMC Gastroenterol. 2008;8:59.
66. Cruz Rde C, Tanajura D, Almeida D, Cruz M, Parana R. Urinary tract infection in non-hospitalized patients with cirrhosis and no symptoms of urinary tract infection: a case series study. Braz J Infect Dis. 2006;10(6):380-3.
67. Evans LT, Kim WR, Poterucha JJ, Kamath PS. Spontaneous bacterial peritonitis in asymptomatic outpatients with cirrhotic ascites. Hepatology. 2003;37(4):897-901.

ANNEXURE I

STUDY PROFORMA

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

OCCUPATION

COMPLAINTS AT PRESENTATION:

Past history:

Family history

Personal history

Treatment history

PHYSICAL EXAMINATION:

GENERAL CONDITION:

PALLOR- YES/NO

ICTERUS-YES/NO

LYMPHADENOPATHY-YES/NO

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

TEMPERATURE

PULSE

RESPIRATORY RATE

BLOOD PRESSURE

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

INVESTIGATIONS

ANNEXURE II

PATIENT INFORMATION SHEET AND

INFORMED CONSENT FORM

Title Of Research Study: CLINICAL PROFILE OF INFECTIONS IN CIRRHOTIC PATIENTS- ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY.

Principal Investigator:-

Dr. _____

Post Graduate Student,

Department Of General Medicine,

JNMC, Belgaum.

Guide:-

Dr. _____

Professor

Department of General Medicine,

JNMC, Belgaum.

Introduction and Purpose:-

He is a well recognized clinical complication of cirrhosis of liver and the presence and prompt identification of well defined precipitating factors is extremely important in diagnosis and treatment of this fatal condition. About 30-40% of patients with cirrhosis of liver are prone to infections

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood and urine samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study. If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1. Dr. J.N.M.C Ethical Committee for Human Research	2. Dr._____ Professor Dept of General Medicine, JNMC, Belgaum.

3. Dr. _____
Investigator,
PG in General Medicine,
JNMC, Belgaum.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name:……...

Signature / Left thumb impression:……...

of the participant

Name of the legally authorized:……...

representative / guardian

Signature / Left thumb impression:……...

Witness' name :……...

Signature / Left thumb impression:……...

Investigator's name and signature :……...

Date:

Place:

S.NO	IPNO	NAME	AGE	SEX	HB	TOTALCOUNT	TOTAL BILIRUBIN	DIRECT BILIRUBIN	SGOT	SGPT	ALK PHOSPHATE	HCV	HBsAG	ASCITIC FLUID ANALYSIS CELL COUNT(CELLS)	ASCITIC CULTURE(negative)	ASCTIC CULTURE(positive)	URINE MICROSCOPY RBC/WBC HPF	URINE CULTURE	URINE CULTURE(POSITIVE)	BLOOD CULTURE	BLOOD CULTURE(POSITIVE)	ALCOHOL HISTORY	DIAGNOSIS 1	2	3	4	5	6	7
1	684898	DEEPAK R SAWANT	37	M	10.3	10300	5.71	4.33	130	80	140	NR	R	350	NO ORGANISM		NORMAL			NO ORGANISM		>15 YRS	CIRRHOSIS OF LIVER	SBP				HBSAG	
2	699888	ISWAR A KUMBAR	53	M	8.2	16000	4.77	2.66	63	30	60	NR	NR	-	-		12-16/10-12			NO ORGANISM		>15 YRS	CIRRHOSIS OF LIVER		UTI				
3	806446	BHARMAPPA D RAKKASAGI	60	M	10.5	13800	3.45	2.33	52	11	168	NR	NR	225	NO ORGANISM		NORMAL			-		>15 YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
4	585935	VENKATESH K KULAKARNI	52	M	11.8	8100	2.21	0.74	67	48	115	NR	NR	480	NO ORGANISM		NORMAL	-		-		>15 YRS	CIRRHOSIS OF LIVER	SBP					
5	804059	SUBADHRA TUKARAM	70	F	5.7	9500	4.19	0.68	30	16	67	NR	NR	-	-		NORMAL	-		-		NO	CIRRHOSIS OF LIVER			PNEUMONIA			
6	683783	KASHAVVA AKHOT	50	F	13	7000	6.3	2.83	98	40	178	R	NR	380		E.COLI	NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
7	684892	ANNAPURNA	68	F	14.4	17800	6.53	3.76	626	343	151	R	NR	12	NO ORGANISM		NORMAL	-		-	KLEBSIELLA PNEUMONIAE	NO	CIRRHOSIS OF LIVER			PNUMONIA			HCV
8	731768	SHAHID HAMID	37	M	6.2	4500	1.3	0.53	51	28	126	NR	R	300	NO ORGANISM		NORMAL	-		NO ORGANISM		<15 YRS	CIRRHOSIS OF LIVER	SBP				HBSAG	
9	732258	PADMA RBALAKKAR	55	F	9.5	5700	8.35	6.8	88	55	156	NR	R	300		E.COLI	NORMAL			NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
10	649444	PRAKASH M UJJAKAR	59	M	10	4000	20.38	15.17	51	49	232	R	NR	400	NO ORGANISM		12-14/18			-		NO	CIRRHOSIS OF LIVER	SBP	UTI				HCV
11	762110	MALLIKARJUN B DODDABIR	58	M	9.2	9000	2.76	1.95	40	30	141	R	NR	2200		E.COLI	NORMAL	-		-	E.COLI	NO	CIRRHOSIS OF LIVER	SBP					
12	690949	SADASHIV MALLAPPA	76	M	11.8	8500	2.04	0.64	40	31	82	NR	NR	490	NO ORGANISM		NORMAL	-		-		NO	CIRRHOSIS OF LIVER	SBP					
13	693142	MARUTHI F MISHRIKOTI	62	M	11.1	7300	3.14	1.28	113	66	141	NR	NR	380	NO ORGANISM		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
14	742367	BHARMAPPA D RAKKASAGI	42	M	8.6	2200	7.1	5.31	81	47	78	NR	NR	400	NO ORGANISM		NORMAL	-		-	STAPHYLOCOCCUS AUREUS	>15YRS	CIRRHOSIS OF LIVER	SBP			CELLULITIS		
15	695804	MANGAL MOHAN KHABADE	58	M	11	13100	2.78	1.57	144	138	119	NR	NR	-	-		NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER			PNEUMONIA			
16	764600	SUVARNA	53	F	15.1	5400	1.08	0.42	162	146	105	R	NR	-	-		NORMAL	-		-	KLEBSIELLA PNEUMONIAE	NO	CIRRHOSIS OF LIVER			PNEUMONIA			
17	814487	SATISH.P.SALONKE	48	M	12.4	6200	1.55	0.83	51	47	105	NR	NR	-	-		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSISNOF LIVER,						
18	814499	ASWINI S VAJJARAMATHI	58	F	10.6	20000	3.14	1.61	44	41	97	NR	NR	600	NO ORGANISM		15/18		E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP	UTI				
19	690677	BASALINGAPPA BAB NAIK	56	M	7.4	14500	0.88	0.3	34	28	98	NR	NR	-	-		10-12/14-16		KLEBSIELLA PNEUMONIAE	NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER		UTI				
20	692143	PANDURANG B KORE	35	M	10.3	4800	3.48	1.74	63	50	145	NR	R	90	NO ORGANISM		FEW/12-14		E.COLI	-	E.COLI	NO	CIRRHOSIS OF LIVER						
21	742367	BHARMAPPA D RAKKASAGI	45	M	10.3	8600	18.64	14.24	43	32	128	NR	NR	400	NO ORGANISM		NORMAL	NO ORGANISM		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
22	982274	SHRISHAIL S KHAVATOKOP	54	M	9.8	4700	1.78	0.78	35	18	160	NR	NR	100	NO ORGANISM		NORMAL	-		-	STAPHYLOCOCCUS AUREUS	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
23	693142	MARUTHI FAKIRAPPA	62	M	12.3	8900	0.9	0.3	113	66	141	NR	R	300	NO ORGANISM		NORMAL	-		-		<15YRS	CIRRHOSIS OF LIVER	SBP				HBSAG	
24	739154	RACCHANA S LINGADAL	46	M	7.3	5500	0.46	0.19	18	17	155	NR	R	500		E.COLI	NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
25	799265	MADDAPPA B BIRADAR	74	M	6.5	1500	1.07	0.36	23	12	53	NR	R	-	-		NORMAL	-		-		NO	CIRRHOSIS OF LIVER					HBSAG	
26	787268	LAKSHMIKANTH G SAWAT	58	M	12	6700	6.23	3.71	80	54	88	NR	NR	-	-		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER						
27	965749	RAMESH S MADGUM	36	M	9.1	2600	2.08	0.9	60	48	149	NR	R	-	-		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER						
28	761804	GANGAWWA V BASAMATH	50	F	10.6	6300	4.64	4.43	76	43	291	NR	R	-	-		NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER					HBSAG	
29	721497	SUNITHA APATIL	50	F	8.9	4900	2.91	1.39	65	31	131	R	NR	-	-		15/14	-	E.COLI	NO ORGAMISM		NO	CIRRHOSIS OF LIVER		UTI				HCV
30	787045	KANTHU B HONGAL	32	M	10.1	7600	30.63	21.12	123	58	224	NR	NR	6300	ENTEROCOCCUS GALLIN		NORMAL	-		-	ENTEROCOCCUS GALLINARUM	<10YRS	CIRRHOSIS OF LIVER	SBP					
31	738216	VENKATESH R PATIL	43	M	9.1	6600	2.87	1.58	85	32	111	NR	NR	300	NO ORGANISM		NORMAL	NO ORGANISM		NO ORGANISM		>15 YRS	CIRRHOSIS OFLIVER	SBP					
32	766154	JYOTHIBA M MORE	44	F	7.9	7400	2.97	2.12	46	24	147	NR	R	300	NO ORGANISM		24-26/42	NO ORGANISM		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
33	770928	MANGAL M KHABADE	58	F	11	23100	6.6	2.4	136	116	176	R	NR	-	-		NORMAL	-		-	STAPHYLOCOCCUS AUREUS(pus)	NO	CIRRHOSIS OF LIVER				CELLULITIS		HCV
34	770958	NINGAVVA V NARLI	66	F	7.2	23200	6.65	2.2	31	20	67	NR	R	-	-		NORMAL	-		-	COAGULASE NEGATIVE STAPHYLO COCUC SPECIES	NO	CIRRHOSIS OF LIVER			PNEUMONIA		HBSAG	
35	725362	PAMPANGOUDA K SANNA	54	M	8	49900	1.76	1.2	68	40	128	NR	NR	-	-		NORMAL	-		-	COAGULASE NEGATIVE STAPHYLO COCUC SPECIES	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
36	786806	GANGABAI S WALI	65	F	14.5	5300	1.08	0.53	57	47	89	R	NR	300	-		NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER						HCV
37	799723	SURAJ R HUBLIKAR	42	M	9.1	7900	11.92	6.83	131	58	147	NR	NR	-	-		NORMAL	-			KLEBSIELLA PNEUMONIAE(pus)	>15YRS	CIRRHOSIS OF LIVER				CELLULITIS		
38	683867	PAYAPPA N SANGODI	46	M	11.6	2700	5.48	2.61	117	158	111	NR	NR	10000	ACINETOBACTER IWOF		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
39	701837	MARSHAL S DINDAR	54	M	8.8	8200	5.67	2.62	59	36	40	NR	NR	600	NO ORGANISM		NORMAL	NO ORGANISM		NO ORGANISM		<15YRS	CIRRHOSIS OF LIVER	SBP					
40	728001	MURTWAJASAB R KERUR	40	M	8.3	4300	8.93	6.92	41	31	141	NR	NR	600	NO ORGANISM		NORMAL	NO ORGANISM		NO ORGANISM		>15 YRS	CIRRHOSIS OF LIVER	SBP					
41	661348	GANGABAI K PAVADDI	62	F	11.6	14700	2.59	1.35	30	30	138	NR	R	600		E.COLI	NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
42	766973	ASWINI S VAJJARAMATHI	55	F	10.6	7300	3.14	1.61	44	41	94	NR	NR	-	-		18/22-24	-	E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER		UTI				
43	650264	SADASHIVF KAMAT	76	M	8.5	7400	1.75	0.85	27	17	100	NR	R	120	-		NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER					HBSAG	
44	755948	RISHIKESH RAJU MANE	63	M	12.3	41300	3.96	2.99	74	42	258	NR	NR	-	-		NORMAL	-		-	COAGULASE NEGATIVE STAPHYLO COCUC SPECIES	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
45	721992	ALTAF ABDUL MANIYAR	50	M	6	3300	2.33	1.4	58	41	139	NR	R	300	NO ORGANISM		NORMAL	-		NOORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP				HBSAG	
46	686102	MARALSHIDAPPA G DINDUR	54	M	10.3	6700	4.93	1.73	50	42	46	NR	R	470	-	BSIELLA PNEUMON	NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
47	699888	ISWAR A KUMBAR	53	M	8.2	6000	4.77	2.66	63	30	50	NR	NR	-	-		NORMAL	-			KLEBSIELLA PNEUMONIAE	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
48	768811	BASAVARAJ	40	M	8.4	10400	9.35	7.67	22	34	119	NR	NR	1000	-	E.COLI	NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
49	808369	RUKMAVVA D KAMBAR	56	M	9.1	3300	1.89	0.99	41	19	65	R	NR	-	-		NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER						HCV
50	722965	SURESH M BUVA	38	M	8.6	4500	2.71	0.61	54	38	19	NR	NR	-	-		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER						
51	728124	MARUTHI S TADAWALE	53	M	4.8	5600	1.16	0.57	40	32	110	NR	NR	-	-		40/100		E.COLI	NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER		UTI				
52	817127	BIPIN D SAHANI	35	M	11.8	27500	1.52	1.`13	144	141	258	NR	R	-	-		NORMAL	-		-	ENTEROBACTER SPECIES(PUS)	NO	CIRRHOSIS OF LIVER				CELLULITIS	HBSAG	
53	816612	GOPAL S AKAKDE	53	M	8.6	21500	9.6	7.67	189	110	222	R	NR	-	-		NORMAL	-		-	KLEBSIELLA PNEUMONIAE	NO	CIRRHOSIS OF LIVER			PNEUMONIA			HCV
54	817143	VINAYAK L BABALI	32	M	6.9	2800	5.51	4.53	60	24	76	NR	NR	-	-		NORMAL	-		-	KLEBSIELLA PNEUMONIAE	>15TRS	CIRRHOSIS OF LIVER			PNEUMONIA			
55	819170	PRAVIN R BARIGAB	55	M	5.5	15200	18.71	13.29	73	33	66	NR	NR	290	NO ORGANISM		NORMAL	-		-	E.COLI	>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			
56	825683	ANIL.S.BORAGAVE	53	M	13.4	17000	1.79	1.28	104	73	90	R	NR	-	-		NORMAL	-		-	STAPHYLOCOCCUS HEMOLYTICUS	NO	CIRRHOSIS OF LIVER			PNEUMONIA			HCV
57	825683	PRAVEEN SHANKAR	55	M	12.6	14600	2	1.78	95	93	66	R	NR	900	-	E.COLI	NORMAL	-		NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP					HCV
58	76859	AHMED HUSSAIN SHAIK	62	M	11.7	5400	4.12	1.77	72	39	129	NR	NR	-	-		20/60	-	E.COLI	NOORGANISM		>15YRS	CIRRHOSIS OF LIVER		UTI				
59	820310	CHANNAPPA B ANGADI	60	M	6.9	15000	4.99	2.3	68	23	81	NR	NR	11000	-	BSIELLA PNEUMON	NORMAL	-		-	KLEBSIELLA PNEUMONIAE	>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			
60	819862	HANUMANTH G CHOVAN	55	M	11	5500	7.88	5.4	222	180	212	NR	NR	700	-	E.COLI	NORMAL	-		-		>15YRS	CIRRHOSIS OF LIVER	SBP					
61	712362	BASAVARAJ H HONNAVAR	56	M	12.6	9000	4.99	2.3	198	111	82	R	NR	2000	-	BSIELLA PNEUMON	NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					HCV
62	768555	HONNAMA	62	F	11	22000	2.88	1.92	88	98	188	R	NR	-	-		NORMAL	-		-	STAPHYLOCOCCUS AUREUS	NO	CIRRHOSIS OF LIVER			PNEUMONIA			HCV
63	788546	BASAVARAJ	48	M	8.8	2200	16.88	14.22	298	111	190	NR	NR	170	NO ORGANISM		NORMAL	-		NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER						
64	654987	BAB RAO	55	M	8.8	14000	15.22	14.22	590	222	222	NR	NR	800	-	E.COLI	NORMAL			-		>15YRS	CIRRHOSIS OF LIVER	SBP					
65	666555	UMESH C CHAVAN	46	M	11.3	12000	4.66	2.22	163	120	150	NR	NR	770		KLEBSIELLA PNEUMON	NORMAL	-		-		>15YRS	CIRRHOSIS OF LIVER	SBP					
66	659714	RAJAMAA HONNAVAR	62	F	12.8	4400	5.23	1.22	123	126	222	R	NR				15/20		E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER		UTI				HCV
67	654321	GAYATHRT DEVI	55	F	8.8	4100	14.22	13	222	250	222	R	NR	550		KLEBSIELLA PNEUMON	NORMAL				KLEBSIELLA PNEUMONIAE	NO	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			HCV
68	654896	FAKIRAWWA	55	F	8.2	3900	5.22	2.22	156	120	106	R	NR	-			20/20		E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER		UTI				HCV
69	654000	HANUMANTH H HAMPI	62	M	10.2	15000	4.88	3.66	145	111	112	NR	R	550		E.COLI	NORMAL			-		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
70	654987	GOVIND N PATIL	52	M	14	11000	4.52	2.22	222	150	108	NR	R	-			18/20		KLEBSIELLA PNEUMONIAE	-		NO	CIRRHOSIS OF LIVER		UTI			HBSAG	
71	621356	HIREMATH	48	M	12.5	15000	12.22	11.22	566	256	222	NR	NR	600	ENTEROCOCCUS GALLIN		NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
72	651023	KATTAMANI	55	M	14.2	3800	4.22	2.22	160	152	100	NR	R	120			NORMAL				STAPHYLO COCCUS AUREUS	NO	CIRRHOSIS OF LIVER			PNEUMONIA		HBSAG	
73	689745	RAJUH TALWAR	48	M	15.6	18000	4.88	2.22	156	148	98	NR	NR				18/18		E.COLI	-		>15YRS	CIRRHOSIS OF LIVER		UTI				
74	666541	MISHRIKOTI	54	M	8.2	3300	9.66	7.23	258	147	98	NR	R	550		E.COLI	NORMAL			-		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
75	784258	HANUMANTH	45	M	10	21900	8.52	5.28	148	50	131	NR	NR	550		E.COLI	NORMAL			-		>15YRS	CIRRHOSIS OF LIVER	SBP					
76	818532	SHIVAPPA	32	M	11.3	17300	9.43	7.33	74	38	115	NR	R	800	NO ORGANISM		NORMAL			-		>15YRS	CIRRHOSIS OF LIVER	SBP				HBSAG	
77	693142	MARUTHI F MISHRIKOTI	62	M	11.1	7300	1.14	0.3	63	32	101	NR	R	300	NO ORGANISM		NORMAL			-			CIRRHOSIS OF LIVER					HBSAG	
78	764698	SHRISHAIL S KHAVATOKOP	55	M	8.8	2300	2.01	0.6	24	10	142	NR	NR				NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
79	784556	TULSHIDAS	55	M	8.2	3300	6.6	2.84	40	32	109	NR	R	300	NO ORGANISM		NORMAL			-		>15YRS	CIRRHOSIS OF LIVER,	SBP				HBSAG	
80	779194	LAXMAN K KALKHABKAR	61	M	12.2	4000	5.04	4.4	87	36	111	NR	NR				NORMAL			-		>15YRS	CIRRHOSIS OF LIVER						

81	733348	ANNAPURNA B GATADAKI	71	F	10.1	5800	4.12	1.77	49	31	107	R	NR	800	KLEBSIELLA PNEUMON		NORMAL				KLEBSIELLA PNEUMONIAE	NO	CIRRHOSIS OF LIVER,	SBP					HCV
82	745852	GAJANAND	55	M	8.2	18000	4.55	4.12	123	156	107	NR	R	-	-		NORMAL				STAPHYLOCOCUS AUREUS	NO	CIRRHOSIS OF LIVER			PNEUMONIA		HBSAG	
83	789654	RAJA REDDY	33	M	8.8	5400	9.23	5.45	350	12	106	NR	R	-			NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OFLIVER					HBSAG	
84	735563	PAMPANGOUDA	56	M	5.3	17300	0.93	0.49	65	40	178	NR	NR				NORMAL				COAGULASE NEGATIVE STAPHYLO COCUC SPECIES	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			
85	789526	MUTTHURAJ	42	M	8.8	12000	2.6	1.07	36	40	205	NR	R	-			NORMAL			NO ORGANISM		NO	CIRRHOSIS OF LIVER					HBSAG	
86	813813	RENEVVA	45	F	8.2	4500	3.54	1.35	40	30	81	R	NR	450		E.COLI	18/18		E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP					HCV
87	879655	MUTTHAPPA	48	M	10.8	14000	9.66	7.23	258	146	106	NR	NR	550	KLEBSIELLA PNEUMON		NORMAL				KLEBSIELLA PNEUMONIAE	>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			
88	784456	RAVALIJAIN	58	F	8.2	4500	1.8	1	36	32	107	R	NR				67/100		ENTEROBACTER SPECIES	NOORGANISM		NO	CIRRHOSIS OF LIVER		UTI				HCV
89	789412	KANTHAMMA	54	F	6.2	12000	1.14	0.3	63	32	101	NR	R	-			18/22		ENTEROBACTER SPECIES	NOORGANISM		NO	CIRRHOSIS OF LIVER		UTI			HBSAG	
90	784123	SUVARNA K KATTAMANI	55	F	8.8	21000	5.04	4.4	81	36	98	NR	R	900		E.COLI	NORMAL				STAPHYLOCOCUS AUREUS	NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
91	745126	BABU RAO	48	M	8.2	5400	4.22	2.99	126	88	107	NR	NR	1100	KLEBSIELLA PNEUMON		NORMAL			NO ORGANISM		>15YRS	CIRRHOISIS OF LIVER	SBP					
92	745698	MAHADEV M KANT	62	M	10.2	4400	6.23	4.22	98	106	111	NR	R	400	NO ORGANISM		NORMAL			NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP				HBSAG	
93	785644	BASAVARAJ	51	M	11.2	15000	9.22	4.66	256	146	108	NR	NR	800	NO ORGANISM		NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP					
94	758965	KANTHA R MUDHOL	55	M	8.8	3300	14.22	11.22	285	264	98	NR	R	800		E.COLI	NORMAL				E.COLI	>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA		HBSAG	
95	745698	LILA	56	F	7.2	2200	5.06	2.22	80	98	128	R	NR	-			18/22		E.COLI			NO	CIRRHOSIS OF LIVER		UTI				HCV
96	785614	RAVI S PARDHI	45	M	8.8	21000	9.22	7.22	222	198	104	NR	NR	550		E.COLI	NORMAL				KLEBSIELLA PNEUMONIAE	>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			
97	785674	RAJU	62	M	10.2	5000	2.22	1.02	98	45	107	NR	R								KLEBSIELLA PNEUMONIAE	>15YRS	CIRRHOSIS OF LIVER			PNEUMONIA			HCV
98	785623	LAKSHMMA	55	F	12.2	2200	8.25	4.22	125	98	106	R	NR	440		E.COLI	18/28		E.COLI	NO ORGANISM		NO	CIRRHOSIS OF LIVER	SBP	UTI				HCV
99	783265	GANGIRAJU	48	M	8.8	45000	12.22	10.08	564	222	200	NR	NR	>10000	ACINETOBACTER IWOF		NORMAL			NO ORGANISM		>15YRS	CIRRHOSIS OF LIVER	SBP		PNEUMONIA			
100	753692	JAMPA KEDUP TSERING	52	M	10.2	5600	4.22	2.22	98	56	108	NR	R	400		E.COLI	NORMAL				STAPHYLOCOCCUS AUREUS(pus)	NO	CIRRHOSIS OF LIVER	SBP			CELLULITIS	HBSAG	