INTRODUCTION:

Unlike recent times, SBP remained an obscure entity earlier. Although Laënnec's name had been connected with cirrhosis since the early 1800s, it was only much later that SBP was diagnosed as a separate entity. The papers of Kerr et al2 and Conn3, which were published within a short duration of each year described SBP. Conn2 was the one who eventually coined the term (SBP) in his 1964 paper.

Though SBP can occur in nephrotic syndrome and other causes of ascites, most cases of SBP occur in cirrhosis with severe hepatic derangement.

SBP is one of the best characterized infectious complications in patients with cirrhosis and ascites. Though it has been approximately six decades since SBP has been known, the prevalence rate almost remains the same despite all the developments. This makes a study essential to know the various determinants of SBP in a cirrhotic patient and various methods available to prevent and treat the same.

AIMS AND OBJECTIVES:

To determine the prevalence of Spontaneous Bacterial Peritonitis in Decompensated liver disease.

PLACE OF STUDY :- Department of General Medicine and Department of Pathology, Govt. Stanley Medical College

STUDY DESIGN :- Observational Study

STUDY PERIOD :- May 2015 to January 2016

INCLUSION CRITERIA

All patients with clinical features of Decompensated liver disease.
EXCLUSION CRITERIA
Patients on antibiotic therapy
Patients with ascites due to a nonhepatic cause

SAMPLE SIZE :- 100

MATERIALS AND METHODS:
Ethical committee approval was obtained and with due informed consent from the study population, the study was undertaken during the time period of May 2015 to January 2016. This is an observational study wherein 100 patients admitted in medicine ward with features of Decompensated Liver Disease were subjected to a diagnostic paracentesis. About 10 ml of ascitic fluid was tapped and sent for analysis to determine the cell count and cytology. Ascitic fluid cell count was determined by manual method. Patients with ascitic fluid PMN >250 cells/cumm were considered positive for SBP. Other relevant investigations done and results analysed.

OPERATIONAL DEFINITION:
SBP is defined as ascitic fluid infection characterized by PMN cells ≥ 250/mm3 in the absence of any identifiable surgically treatable intra-abdominal source of infection.

Descriptive statistics was done for all data. The data is analyzed using EpiInfo software (7.1.0.6 version: Center for disease control, USA) and Microsoft Excel 2010.
RESULTS:

SBP POSITIVE

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>10</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>8</td>
</tr>
<tr>
<td>Abdominal tenderness</td>
<td>7</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>5</td>
</tr>
</tbody>
</table>

DISCUSSION:

The study concluded that the prevalence of SBP in cirrhotic patients was 15%. 13 out of 15 patients with SBP were symptomatic. This is in accordance to most of the studies conducted worldwide.

Of the 13 patients who were symptomatic, 10 patients presented with fever, 8 with abdominal pain, 7 patients had abdominal tenderness and 5 patients presented with altered mental status.

It is also interesting to note that 7 patients presented with symptoms suggestive of SBP but the laboratory analysis was against the diagnosis.

About 60% of SBP patients were in the age group of 41-50 years.

Among the SBP positive patients bilirubin was >6mg in about 40% of patients and the rest had equal distribution between 2.1-6 mg/dl.

The evidence for transaminitis as seen in levels of AST and ALT proved that more than 50% of patients with SBP had their enzyme levels around 80-100. Only 20% of patients had their enzyme levels more than 200 U.

Studies have indicated that the prevalence rate of SBP in Decompensated liver disease related ascites is around 20-25%. The value of an early diagnostic paracentesis (<72 hours) has been proven by a reduction in the in-hospital mortality rate as compared to those patients who are subjected to a late paracentesis (after 72 hours).

Patients who suffer from DCLD related ascites may not present with the classical clinical features of SBP and it is imperative to determine whether the patient has SBP in order to institute appropriate antibiotic therapy to which the most prevalent organisms are susceptible.

This study aims to bring about a reduction in the mortality rate associated with SBP by diagnosing and instituting specific therapy at the earliest.

Similar study was conducted on 122 cases admitted in Department of Medicine, through emergency, in Guru Gobind Singh Medical College and Hospital, Faridkot, Punjab, India. Cases of cirrhosis with ascites between the ages of 18-75 years were included in this study. Of the 122, 27 (20.4%) patients were diagnosed as having SBP and its variants. Monomicrobial Bacterascites (BA) was present in 5 patients and Culture Negative Neutrocytic Ascites (CNNA) was present in 22 patients.

LIMITATIONS OF THE STUDY:

- The correlation with the duration of cirrhosis and the prevalence of SBP has not been made since most of the patients present late.
- The culture of ascitic fluid was not done.

REVIEW OF LITERATURE:

SBP is the first and most common Ascitic Fluid infection described.

SBP is always monobacterial and the presence of more than one bacterial species in ascitic fluid should raise the suspicion of diagnosing secondary peritonitis.

EPIDEMIOLOGY:

The frequency of SBP in cirrhotic patients may be as high as 18%. This number has grown from 8% over the
past 20 years, most likely due to an increased awareness of spontaneous bacterial peritonitis and a lowered threshold to perform diagnostic paracentesis

In patients with ascites, both sexes are affected equally.

PATHOGENESIS:

Bacterial translocation (BT), is the key mechanism in the pathogenesis of SBP. 10-14

An alternative proposed mechanism for bacterial inoculation of ascites is haematogenous transmission in combination with an impaired immune system. Nonetheless, the exact mechanism of bacterial displacement from the GI tract into ascitic fluid remains controversial.

A variety of factors contributes to peritoneal inflammation and bacterial growth in ascitic fluid. Intestinal bacterial overgrowth, along with impaired phagocytic function, low serum and ascites complement levels, and decreased activity of the reticuloendothelial system, contributes to an increased number of microorganisms and decreased capacity to clear them from the bloodstream, resulting in their migration into and eventual proliferation within ascitic fluid.

OTHER VARIANTS OF ASCITIC FLUID INFECTION

- **CULTURE NEGATIVE NEUTROCYTIC ASCITES**

  Culture-negative neutrocytic ascites (CNNA) is diagnosed when (1) the ascitic fluid culture grows no bacteria, (2) ascitic fluid PMN count is 250 cells/mm3 (0.25 × 10^9/L) or greater

- **MONOBACTERIAL NONNEUTROCYTIC BACTERASCITES**

  Monomicrobial non neutrocytic bacterascites is a form in which the cultures from the ascitic fluid are positive but the number of PMN is <250/cumm. mm3.

- **SECONDARY BACTERIAL PERITONITIS**

  Secondary bacterial peritonitis is diagnosed when the ascitic fluid culture is positive (usually for multiple organisms), the PMN count is 250 cells/mm3 or greater, and an intra-abdominal surgically treatable primary source of infection has been identified.

- **POLYMICROBIAL BACTERASCITES**

ETIOLOGY:

Traditionally, three fourths of spontaneous bacterial peritonitis infections have been caused by aerobic gram-negative organisms (50% of these being Escherichia coli).

The remainder has been due to aerobic gram-positive organisms (19% streptococcal species).

However, some data suggest that the percentage of gram-positive infections may be increasing. One study cites a 34.2% incidence of streptococci, ranking in second position after Enterobacteriaceae. Viridans group streptococci (VBS) accounted for 73.8% of these streptococcal isolates. Anaerobic organisms are rare because of the high oxygen tension of ascitic fluid. A single organism is noted in 92% of cases, and 8% of cases are polymicrobial.15

CLINICAL MANIFESTATIONS OF SBP:

The clinical manifestations of SBP are subtle and require a high index of suspicion.

SBP mostly occurs in large volume ascites in patients with cirrhosis. Patients with cirrhosis usually have hypothermia; Therefore, any temperature > 37.8deg should be investigated. Fever is the most common symptom. Other features include abdominal pain and tenderness, nausea, vomiting and gastrointestinal [GI] bleeding15. In patients with variceal haemorrhage, the frequency of SBP is significantly increased, and prophylaxis against SBP is recommended when patients present with UGI bleeding. There may be alterations in mental status. Thirteen percent of patients have no signs or symptoms. Paralytic ileus and hypotension are seen in advanced illness.

Only few patients with SBP present with typical symptoms of peritoneal infection as fever, abdominal pain and peripheral leukocytosis, SBP is more frequently suspected when the patient develops signs of hepatic encephalopathy, increase of the abdominal volume or renal dysfunction without any apparent precipitating factor. In addition to this, in a significant part of the cases, SBP can be completely asymptomatic and the diagnosis can be done only by analyzing the paracentesis results. If ascitic fluid infection is suspected, ascitic fluid total and differential count and ascitic fluid culture should be done, with inoculation of the material in blood culture bottles at the bedside.

TECHNIQUES AND LABORATORY:

The diagnostic algorithm proposed by Runyon remains the method of choice for diagnosing SBP. Diagnostic paracentesis is now regarded as a safe procedure. The accepted area of preference is away from the midline, at the point of maximal dullness, and ideally in the left iliac fossa, two fingerbreadths medial and two ventral to the anterior superior iliac spine (“Runyon's spot”).
**DIAGNOSIS:**
Diagnosis is made by looking for signs and symptoms of SBP and confirmation with ascitic fluid examination. Physical examination findings include hypotension, tachycardia, altered mental status and abdominal tenderness. Ascitic fluid neutrophil count >250 cells is the accepted criterion. Ascitic fluid should be cultured to identify bacteria. Granulocyte elastase and lactoferrin released by activated PMNs are elevated in patients with SBP. Lactoferrin shows notable sensitivity (95.5%) and specificity (97%) for diagnosing SBP.

Bacterial cultures require several days to obtain results. Hence, bacterial DNA detection and sequencing is increasingly being used to diagnose various infectious diseases. The reagents used for DNA extraction procedures carry a risk of exposing the clinical samples to exogenous bacterial DNA. Although PCR is a very sensitive method for detecting DNA, PCR-based methods display discrepant and controversial findings with respect to diagnostic performance in detecting the causative pathogens in SBP.

**TREATMENT:**
Appropriate antibiotic therapy should achieve resolution of infection in most cases of SBP.

**INTRA VENOUS ANTIBIOTICS**
Third generation cephalosporins are the antibiotics of choice with many studies confirming high levels of SBP resolution. Cefotaxime 2 g every eight hours, has been shown to result in excellent ascitic fluid levels (20-fold killing power after one dose). Cefotaxime, has been shown in a controlled trial to be superior to ampicillin plus tobramycin for the treatment of spontaneous bacterial peritonitis. 98% of causative organisms were susceptible to cefotaxime, which did not result in superinfection or nephrotoxicity. In patients with a serum creatinine level greater than 3 mg/dL, the dosing interval may be extended to 12 hours.

**ORAL ANTIBIOTICS**
Oral ofloxacin has been reported in a controlled trial to be as effective as parenteral cefotaxime in the treatment of spontaneous bacterial peritonitis in patients who do not have vomiting, shock, bleeding, or renal failure. Because of the possibility of fluoroquinolone resistance in patients receiving fluoroquinolones to prevent spontaneous bacterial peritonitis, the empirical use of this class of drug to treat suspected spontaneous bacterial peritonitis should be avoided(1).

**INRA VENOUS ALBUMIN**
Renal impairment occurs in 33% of episodes of spontaneous bacterial peritonitis. Spontaneous bacterial peritonitis leads to increased intraperitoneal nitric oxide production, which in turn further increases systemic vasodilatation and promotes renal failure. Intravenous albumin (1.5 g/kg of body weight at the time the infection is detected and 1.0 g/kg on day three) can increase intravascular volume and, in combination with cefotaxime, has been shown in a large randomized trial to reduce the risk of renal failure(1).

**PROPHYLAXIS:**
The recurrence rate of SBP following a first episode is up to 70% at 1 year. Therefore, it is necessary for prophylaxis to this group of patients and referral for transplant assessment. This therapy is backed up by evidence showing a reduction in recurrence of SBP from 68% to 20% in one study. Norfloxacin 400 mg/d or ciprofloxacin 500 mg/d orally is the most studied and commonly recommended regimes.

**CONCLUSION:**
SBP is one of the serious complications of cirrhosis with the prevalence rate of 10-30%. It is imperative to diagnose SBP early and treat them with appropriate antibiotics. In view of the possibility of asymptomatic infection and high mortality of SBP, it is advisable to carry out routine diagnostic tap in all cirrhotics with ascites at hospital admission. Further, the success rate of treatment with early institution of antibiotics in SBP is high thus emphasizing the value of early diagnostic tap.

**REFERENCE:**
1. Sleisenger and Fordtran’s, Gastrointestinal and liver disease:9th edition, Vol 2;Pg 1528-34.
5. Angeloni S, Leboffe C, Parente A, Venditti M,


15. Koulaouzidis A et al. Spontaneous bacterial peritonitis