



## Case Study

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## Spontaneous Pneumococcal Ascites Fluid Infection about a Case

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## Summary

**Introduction:** Spontaneous infection of ascites fluid is a serious and progressive complication of decompensated cirrhosis. Ascites contamination is usually caused by enteric germs, mainly *Escherichia coli*.

**Observation:** A 74-year-old patient, with a history of post *Hepatitis C cirrhosis*. Presented to the emergency department for a sudden onset abdominal pain with fever. After exploration, the diagnosis of ascites fluid infection was suspected. Thus, a fluid puncture was realized evacuating purulent fluid. Pneumococcus was identified in the ascites fluid. Despite antibiotic therapy the evolution of the patient was unfavorable, and he died due to hepatic encephalopathy.

**Conclusion:** Cirrhotic and ethyl patients are prone to *Streptococcal pneumonia* infections. Mortality is high in this immunocompromised population, but rapid diagnostic and therapeutic management improve the prognosis.

**Keywords:** Infection; Ascites fluid; Cirrhosis; *Streptococcus pneumonia*

## Introduction

Spontaneous infection of ascites fluid, also called spontaneous bacterial peritonitis is a common complication in cirrhotic patients which bears severe consequences (hospital mortality rates: 30-50%) [1]. Gram negative rods are the most isolated germs (70%) [2]. However, cirrhotic and alcoholic patients are particularly prone to infection with *Streptococcus pneumoniae*. We report a case of an ascites fluid infection with pneumococcus in a post-hepatitis C cirrhotic patient.

## Case

Mr X. is a 74 years old patient, hospitalized at the emergency department of Hassan II's teaching hospital of Fez. He was admitted for an ascitic decompensation of his cirrhosis caused by hepatitis C genotype 1 subtype 1a. His history is significant for diabetes mellitus type II treated with oral hypoglycemics, essential hypertension treated with calcium channel blockers and diabetic nephropathy under treatment. His cirrhosis was first diagnosed in 2009 revealed by an episode of variceal gastro-intestinal bleeding that manifested with hematemesis and melena.

The patient currently presents with severe generalized abdominal pain, postprandial vomiting but no GI bleeding. He did not present any upper or lower respiratory tract symptoms

prior to his admission. The evolution was marked by fever and a deterioration of his general status. Physical examination found a conscious patient with temperature of 39° C, tachycardic at 100 bpm, he also presented with jaundice, pallor and peripheral edema.

The abdominal examination found an important ascites (significant abdominal volume, generalized tenderness and dullness to percussion). Rectal examination found faeces of normal color.

Complete blood count showed a normal leucocyte count of 9000 per mm<sup>3</sup>, a normochromic normocytic anaemia with haemoglobin of 10 g/dl and a mild thrombocytopenia of 135 000/mm<sup>3</sup>. The electrolyte panel revealed a mild hyponatremia of 133 mmol/ml and normal potassium levels. Liver function tests showed normal total and conjugated (5/3 µmol/l) bilirubin levels, as well as GOT and GPT levels. Alpha Fetoprotein (AFP) was elevated 2000 ng/ml and albumin levels were low (5.5 g/l). Renal function tests showed chronic kidney failure (creatinine of 75 mg/l and urea of 2,36 g/l). CRP was elevated (255 mg/l). Urine and blood cultures were sterile.

Ascites fluid analysis on the day of admission showed a cloudy fluid with 3948 leucocytes/mm<sup>3</sup> with a predominance of neutrophils 96%. Culture demonstrated the presence of *Streptococcus pneumoniae* susceptible, to penicillin, amoxicillin, amoxicillin clavulanic acid, cefotaxime, imipenem, tetracycline, erythromycin, lincomycin, pristinamycin, rifampicin, vancomycin and teicoplanin to ofloxacin, to the combination of trimethoprim

Sulfamethoxazole and to gentamicin. Hence the diagnosis of pneumococcal bacterial peritonitis was confirmed.

Management was based on administration of antibiotics (ceftriaxone 2 g/day) and ascites fluid control analysis that showed a decrease in white blood cells to 200/mm<sup>3</sup> still predominantly made of neutrophils (80%) and a sterile culture. CRP levels also decreased to 150 mg/l.

On day five of his stay the patient presented a GI bleeding of low abundance due to oesophageal varices rupture. The next day the patient presented a hepatic encephalopathy which led to his death.

## Discussion

*S. Pneumoniae* has a facultative anaerobic metabolism, homofermentative, catalase and oxidase negative. It is a Gram-positive coccus of less than 2 µm in diameter, non-motile and non-sporulating. It forms diplococcus or short chains. The virulence of the pneumococcus varies depending on the age, sex, and geographic area of the patient. It is classically susceptible to penicillin, but the emergence of pneumococcal strains resistant or of reduced susceptibility to penicillin, even to cephalosporins, sometimes causes difficult therapeutic problems [3,4].

Spontaneous ascites fluid infection is defined as the association of a positive ascites fluid culture, with a predominantly neutrophilic white blood cell count greater than 250 / mm<sup>3</sup>, in the absence of an obvious source of an intra-abdominal infection [5].

The main pathogenic mechanisms leading to the invasion of the peritoneum in spontaneous bacterial peritonitis are:

The contamination of the peritoneal cavity resulting from the spread of a pleural infection through the lymphatic vessels that cross the diaphragm creating a communication between the two cavities. Hence, in the presence of a pneumococcal pulmonary infection, the germ can gain the peritoneum via this route [6]. In our case the patient did not present any pulmonary symptoms, nor did he have abnormal findings on chest X ray.

Peritoneal contamination can also be caused by bacteraemia. Thus, peritonitis could be explained by this bacterial metastasis from an unknown primary location or during a transient asymptomatic bacteraemia [7]. However, blood cultures that were realized turned out negative ruling out this theory.

The last mechanism would be a contamination by oral ingestion of the micro-organism. Nevertheless, pneumococcus have only rarely been isolated in intestinal flora [8]. It's a hypothesis that can explain the ascites fluid infection. Cases of spontaneous bacterial peritonitis caused by pneumococcus are rare, they represent 2,6 to 8% of cases in literature review [7,9]. Before the

era of antibiotics, this complication had high fatality rate [10,11].

Whatever the origin of the peritoneal contamination, the persistence of the infection is due to the ubiquitous immunosuppression in patients suffering from decompensated chronic hepatopathy [1] Indeed, this population suffers from a decreased serum bactericidal activity due to hypocomplementaemia especially in the C3 portion. And due to low concentration, in both blood and ascites fluid, of fibronectin, an essential opsonin to the process of phagocytosis of bacteria, but also due to the decrease in chemotaxis of neutrophils and a disturbance of phagocytic function of neutrophils and monocytes [5].

The clinical presentation is usually typical, it involves the occurrence or aggravation of encephalopathy, the most constant warning sign, fever, spontaneous abdominal pain or tenderness to palpation, diarrhea, a decrease or disappearance of digestive noises. In extreme cases the infection may remain asymptomatic or manifest as septic shock [2]. Our patient had presented a characteristic clinical presentation of peritonitis with fever and abdominal pain. The physical examination showed significant distension, diffuse dullness and tenderness to palpation.

The study of ascitic fluid remains the key examination that allows the discovery of an early infection or clinically asymptomatic. Bacteriological diagnosis is based on direct microscopic examination, culture on blood agar showing alpha-hemolytic colonies, smooth, bulging, shiny and optochin sensitive. Identification can, if necessary, be supplemented by lysis by bile or bile salts of a broth culture. As for the search for soluble antigen by latex agglutination, it has a sensitivity of almost 80% but does not detect non-capsulated strains. In order to improve the sensitivity of the bacteriological diagnosis, it is recommended to inoculate the ascites fluid in a blood culture bottle at patient's bed side.

In a cohort study, done between 2000-2007 in Korea including 853 samples amongst these patients 292 had cirrhosis. *S.pneumoniae* was identified in 10 patients. None of these patients had pneumonia or any upper respiratory tract infection. However, two patients presented pneumococcal bacteraemia. Our patient did not present any respiratory symptoms and chest X ray was normal and blood culture was sterile.

The best choice of antibiotics is third generation cephalosporins while awaiting for the antibiogram susceptibility result.

## Conclusion

*Streptococcus pneumoniae* is a rare cause of ascites fluid infection. Gram negative rods are more frequently observed. A combined early diagnosis and treatment improve prognosis. The risk of recurrence in these patients is considerable thus the possible indication for liver transplant.

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