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Case Report

Campylobacter bacteremia: A case report

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Abstract

Campylobacter bacteremia is rarely encountered, probably due to the fastidious nature of the organism. Moreover, it could be transient in nature, especially in immunocompetent individuals, but life-threatening in immunocompromised patients. We report a case of probable transient bacteremia in a recently diagnosed case Cirrhosis of Liver due to Autoimmune hepatitis with Primary Sclerosing Cholangitis.

Keywords: Campylobacter, Bacteremia, Immunocompromised.

Introduction

Campylobacter, a fastidious bacterium, remains an uncommon cause of bloodstream infection.[1] The incidence of campylobacteraemia varies between 0.1% and 0.4% of the total incidence of Campylobacter infections.[2] At least 10 different *Campylobacter* species have been documented in bacteremia cases; but predominantly it is associated with *C. jejuni*, *C. coli*, and *C. fetus* infections.[3] *Campylobacter* bacteremia occurs mainly in immunocompromised patients.[1] Presenting one such case of C jejuni bactermia.

Case Report

A 52 year old woman presented with high grade fever with chills, nausea and decreased appetite. She was recently diagnosed as a case of Cirrhosis of Liver due to autoimmune hepatitis (Antinuclear antibodies positive; Anti-smooth muscle antibody negative) with Primary Sclerosing Cholangitis. She received immunosuppressant therapy with azathioprine for about a couple of months and later switched to steroids, causing steroid-induced diabetes mellitus. She was admitted with similar complaints to another hospital a month back where her blood culture grew Klebsiella pneumoniae. On examination the patient had bilateral oedema, icterus, temperature was 37^0C, heart rate was 86 beats/minute, blood pressure was 100/56 mm of Hg. Complete blood count revealed haemoglobin of 10.3 g/dl, total leucocyte count of cells/mm^3 and platelet count of 73,000/mm.[3] Biochemistry work-up showed AST of 22 U/L, ALT of 72 U/L, total bilirubin of 10.9 mg/dl (direct 7.7 mg/dl), CA-125 of 321 U/ml. Urine and two sets (comprising an aerobic and anaerobic bottle) of blood cultures were drawn and patient was put on artesunate, piperacillin-tazobactam, teicoplanin and fluconazole. Malaria antigen was not detected. Her CMV IgG was reactive. Urine culture had no growth. Ultrasound of the abdomen revealed minimal ascitis.

Blood culture was performed using an automated system (BacT/Alert; bioMérieux, France;) using BacT/Alert plus culture media bottles. Aerobic bottles turned positive after 72 hours which revealed Gram negative spiral bacilli of various size (2 - > 20 spirals).

Fig. 1: Campylobacter agar plate

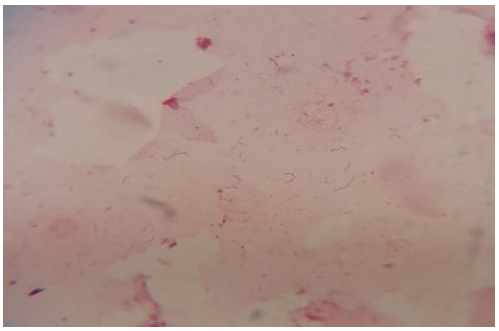

Fig. 2: Gram Stain 100X

Subcultures were performed on common bacteriological culture media (5% sheep blood agar, chocolate agar and MacConkey agar but no microorganism growth on the plate culturewas seen after 48 hours of incubation at 35°C. The clinician was alerted about the possibility of microaerophilic organism (Helicobacter,

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Campylobacter or Arcobacter species). Since the patient had no further fever spikes and the bacterial identification was awaited she was continued on piperacillin-tazobacam and rest of the antibiotics were stopped. Patient was discharged after 7 days. One of the bottle was sent to our reference laboratory. The organism was kept viable by subculturing into another bottle. Simultaneously, it was subcultured on Campylobacter agar plate (HiMedia) and incubated at 42^0C for 72 hours under anaerobic and microaerophilic conditions. The colonies were tiny, translucent, convex, entire and glistening, which were oxidase and catalase positive.

Meanwhile the reference laboratory identified the isolate as Campylobacter jejuni by MALDI-TOF (Metropolis Mumbai) only after 7 days. However, antimicrobial susceptibility testing could not be done due to the fastidious nature of the organism.

Discussion

One of the most common extraintestinal manifestations of *Campylobacter* species is bacteremia.[3] *Campylobacter jejuni* is one of the main bacterial cause of enteroinvasive diarrhea. As compared with *Salmonella* infections, infections caused by C. jejuni are only rarely complicated by extra intestinal localization orbacteremia.[1] A pubmed search revealed most of these bacteremias are reported from developed countries,[1-3] while there are a few reports from the developing countries.[4] Similarly, in India bacteremia due to Campylobacter was reported by Waghmare A. et al following scaling and root planing.[5] However, this is the first report from India with an underlying liver disease. Since blood cultures are not routinely performed for patients with acute gastrointestinal illnesses[4] and the absence of specific *Campylobacter* isolation protocols, and the unknown sensitivity of the automated systems for thedetection of *Campylobacter* spp., may be resulting in an underestimation of *Campylobacter*-related bacteremia.[4,6] In a large percentage of cases the clinical presentation of Campylobacter bacteraemia may show a febrile illness without gastrointestinal symptoms,[7] which was also the case in our patient. C. jejuni bacteremia can be transient in immunocompetent patients[1] and this can resolve without antimicrobial therapy. Conversely, individuals with immune deficiency or another serious underlying condition (cardiovascular disorders, hematological malignancies, liver disease, human immunodeficiency virus infection and hypogammaglobulinemia) are exposed to an increased risk of bacteremia due to C. jejuni. In these individuals, an effective antimicrobial treatment has been significantly associated with an improved outcome.[7] A lack of specific clinical findings of Campylobacter bacteremia[1] and absence of consensus on optimal antibiotic regimen makes it difficult for the clinician to select an appropriate antimicrobial therapy.[7] Our patient was treated with piperacillin-tazobactam for 14 days with no untoward consequences. However, Campylobacter isolates are not regularly susceptible to penicillins [22–24] and the β-lactamase enzyme found in C. jejuni is preferentially inhibited by clavulanic acid, but not by tazobactam or sulbactam.[7] Due to the fastidious nature of the organism antimicrobial susceptibility could not be performed. Since the patient did not relapse, it could have been a transient bacteremia which got resolved on its own.

Thus to conclude Campylobacter bacteremia should be considered in immunocompromised patients especially when the organism doesn't grow under routine aerobic conditions and appropriate antimicrobial therapy should be started.

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Conflict of Interest

None.

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