Acinetobacterlwoffii - Emerging pathogen causing liver abscess: A Case Report

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Abstract
Acinetobacterlwoffii is a gram negative aerobic non-fermenter bacilli. It is now an important emerging pathogen responsible after *Acinetobacterbaumannii* for causing nosocomial infections. It also affects immunosuppressed individuals with co-morbidities. Here we report a rare case of community acquired pyogenic liver abscess caused by *Acinetobacterlwoffi*.

Keywords:*Acinetobacterlwoffii*, Carbapenems, Liver Abscess.

Introduction
Acinetobacter species are ubiquitous in environment. The role of A.baumannii in nosocomial infections have been well documented but the clinical effect of other *Acinetobacter* species has not been investigated.[1]*Acinetobacterlwoffii*, a non-fermentative aerobic gram-negative bacilluspresents in normal flora of the oropharynx, skin and perineum in approximately 25% of healthy individuals. It has been implicated as an opportunistic pathogen in immunosuppressed patients and nosocomial infections like septicemia, pneumonia, meningitis and wound infections.[2]Herein we present a case report of community acquired pyogenic liver abscess caused by *A.lwoffii* in an immunocompetent patient.

Case Report
A 30-year-old man, presented to the Emergency Department with chief complaints of high grade fever and weakness since last one week. The high grade fever was continuous and associated with chills and rigors. Patient gave history of pain in abdomen which was constant and usually located over right hypochondrium and got aggravated by cough. He was a non-smoker but used to drink about 40 grams of alcohol daily for last seven years. There was no history of vomiting, weight loss and yellowing of eyes or urine. .No history of tuberculosis, diabetes orextramarital sexual exposure were present. On examination he was conscious, febrile (temperature-39ºC), pulse rate 100/min and blood pressure 115/90 mm/Hg. He had mild pallor but no icterus. There were no signs of chronic liver disease. As per abdominal examination patient had distended abdomen and tenderness in right hypochondrium and epigastric region. There was tender hepatomegaly, mostly over left lobe, with a liver span of 16 cms. No hepatic rub or bruit were present. Rest of systemic examination was within normal limits. Laboratory hematological investigations revealed hemoglobin 11.24 gm/dL, total leukocyte count 23,100/cmm, (polymorphs 75%, lymphocytes 23%, eosinophil 2%), Platelet count of 90000/cumm, however liver enzymes (SGOT/SGPT-93/66) and alkaline phosphatase were elevated to 970 IU. Renal function and serum electrolytes were within references range. Serological markers for HIV, Hepatitis B and Hepatitis C were non-reactive.Widal test and IgMleptospira were also negative. Ultrasonography of the abdomen showed enlargement of the liver with features suggestive of abscess measuring 4.2cmx2.8cmx4.2cm and volume 680cc involving segment IV along with mild ascites. The liver measured 14.7 cm over mid clavicular line with normal intrahepatic and biliary radicals. The abscess being superficial over left lobe was drained under USG guidance with strict asepsis. The pus sample was received at our microbiology laboratory and was processed as per standard protocol. A wet mount of pus was negative for Trophozoites of *Entamoebahistolytica*. Direct Gram's stain showed few gram negative bacilli along with plenty of pus cells.Ziehl-Nelsen stain was negative for acid fast bacilli. Sample was inoculated on blood agar, MacConkey agar and chocolate agar plates. They were incubated at 35ºC and examined for growth at 24 hours. Non-lactose fermenting colonies (Fig 1) were processed and were identified as *A. lwoffii* on the basis of biochemical reactions. It was confirmed by Vitek-2 system and also by MALDI-TOF systems with a good confidence interval. Routine laboratory susceptibility testing procedures of *A.lwoffii* isolates for commonly used antibiotics were performed with the use of Kirby-Bauer disk diffusion method, as recommended by the Clinical and Laboratory Standards Institute (CLSI) guidelines. It was found to be susceptible only for carbapenems except for imipenem.He was treated with a two weeks courseof parenteral meropenem and recovered fully. He was reviewed after one month of discharge and was asymptomatic with normal liver size and complete resolution of the abscess on USG examination.

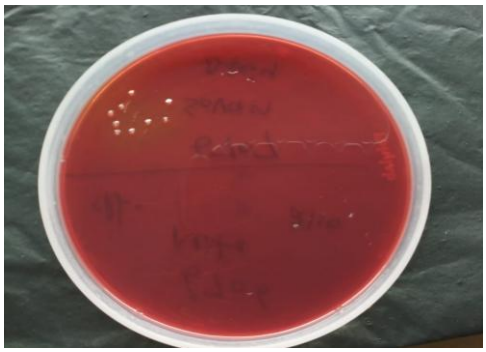

Fig. 1: MacConkey agar showing NLF colonies

Discussion

Liver abscesses are a burning problem in tropical countries and remain a formidable diagnostic and therapeutic problem. Pyogenic abscess if left untreated, invariably run a lethal course. They are prevalent all over the world and pyogenic abscesses are predominant over the amoebic ones. The incidence of pyogenic abscesses varies from 1.1 to 17.6/1, 00,000 individuals.[3]*E.coli* and *Klebsilella* are the commonest isolated organisms followed by *Staphylococcus aureus*, *Enterococcus*, *Streptococcus* and bacteroides.[4] Mycobacterium and fungal growth are extremely rare. A*cinetobacterspp* have received a great interest over the last two decades due to its ability to accumulate, with multiple mechanisms and multiple-drug resistant strains. These bacteria are responsible for majority of the intensive care (ICU) infections, ventilator associated pneumonia (VAP) and community–acquired infections.[5]Although ventilator dependent patients are at greater risk, use of powerful antibiotics like 3rd generation cephalosporins and carbapenems contribute as well.

Most of the affected patients have underlying comorbidities like renal disease, alcoholism, diabetes mellitus and heavy smoking.[5, 6]Among its species, *A.baumannii*is of greatest clinical importance and has been associated with several nosocomial outbreaks. But on the contrary the clinical effect of other *Acinetobacter* species, especially A.lwoffii (formerly known as Mimapolymorpha, A.calcoaceticus var. lwoffii), has rarely been reported.[7]Till date it has been associated with community acquired infections like pneumonia and bacteremia. In these cases the source of infection could not be established but it was hypothesized to have occurred due to poor sterilization of nebulizers.[8]It is slowly emerging as an important pathogen in hospital and communities. In this case it presented as a case of pyogenic liver abscess which as per our extensive research is unusual.Dietary history revealed that the patient was a regular consumer of sea-food as he was a resident of the coastal areas. It has been proven that variety of frozen food stuff and milk products harbor *Acinetobacter* species with *A.lwoffii* and *A johnsanii* being the predominant ones.[9]We thus presume that our patient became a colonizer of A.lwoffii which he acquired through food ingested. The patient was a chronic alcoholic and as a result had decreased immunity. Weak immune status and virulence potential could have led to the migration of the bacteria to the liver via portal system and eventually lead to liver abscess. Since our patient did not have any history of prior hospital admissions or prolonged course of antibiotics, we assumed it to be a community acquired infection.

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Conflict of interest: None
Funding: None
Cite this Article as: *Mohanty A, Kabi A, Mohanty A.* Acinetobacterlwoffii - *Emerging pathogen causing liver abscess: A Case Report. Natl J Integr Res Med 2018; 9(5):53-54*