A case report: *Pseudomonas luteola* presenting with liver mass containing necrosis, sub-phrenic and pleural fluids in otherwise healthy young woman

**Abstract**

*Pseudomonas luteola* is agram-negative bacillus rarely causing human infections. However, Reports have been building up on different types of infections caused by this potentially emerging microorganism. Here we report a case of liver mass with necrosis, sub-phrenic and pleural effusions. *P. luteola* liver infection was treated successfully as inpatient for few days with imipenem followed by ciprofloxacin. The bacterial isolate was found susceptible to a wide range of antimicrobial agents but was resistant to colistin.

**Keywords:** *Pseudomonas luteola*, liver mass, liver abscess, colistin.

**Introduction**

*Pseudomonas luteola* is a motile, aerobic, lactose non-fermenter gram-negative bacillus and oxidase negative but catalase positive (formerly classified as CDC group Ve-1 and named *Chryseomonas luteola*) [1]. Also, *P. luteola* has been found frequently in natural water bodies, soil, and environments and is considered to be a saprophyte or commensal organism[1]. It has been reported to cause different infections; including indwelling vascular catheter causing repeated bacteremia, sever peritonitis in a patient requiring continuous peritoneal dialysis, bile infec-
tions in an obstructed jaundiced patient, meningitis in a newborn, prosthetic valve infective endocarditis implanted for a degenerative rheumatic valve disease in a 13-year-old boy, and aortic bioprosthesis infection in a 53 years old man 16 months following its implantation, neonatal sepsis, skin abscess with bacteremia in a healthy adult patient and non-traumatic septic arthritis in a nine years old boy[2-10]. Furthermore, in an unusual presentation, in 16 years old boy with autoimmune thrombocytopenia on steroids, *P. luteola* presented as a mediastinal mass mimicking a mediastinal malignant lymphoma and showing Splendore-Hoeppli phenomenon on cytological examination of the “mass”[11]. Here we present a unique case of *P. luteola* infection presenting as a liver mass with necrosis, sub-phrenic and pleural effusion (Figure 1).

The case

A 43 year old female patient, medically free, with history of surgeries for weight loss; gastric ring in 2007 which was removed in 2011 and sleeve gastrectomy was done in April, 2013 successfully without any post-operative complications, she lost 23 kg. On January, 2014 the patient was admitted to Medical Center of Jordan Hospital, complaining of right sided upper abdominal pain radiating to the back, continuous, stabbing in nature, increases with movement. Pain started mild and increased in severity in the 4 days prior to admission, and was alleviated with paracetamol. She denied changes in bowel habit or stool color, there was no fever, chills, night sweats, nausea or vomiting and jaundice and had good appetite. Liver function tests were within

![Figure 1. Liver involvement as seen; (A) shows the diaphragm (white arrow) with pleural and sub-phrenic fluid on both sides. Axial view (B) and coronal views (B and D) showing a big mass/abscess (yellow arrows).](http://www.iajaa.org)
normal limits except for albumin which was 3 gm/dl. White blood cells count was 20.7 X 10^3/mm^3 (86% neutrophils) on admission (16/1/2014) and 10.8 X 10^3/mm^3 on discharge (26/1/2014) hemoglobin was 10.2 gm/dl and MCV 86.0. A triple phase liver CT scan and ultrasound showed multiple ill-defined hypodense lesions mainly in segment 6 and 7 of liver with questionable abscess formation. A CT-guided liver biopsy was done for histopathological and microbiological studies. Liver histology revealed inflammatory lesion and tissue culture showed heavy growth of *P. luteola*.

On January 19th, 2014 the patient has developed spikes of fever, and her Chest X-Ray showed right sided pleural effusion and mild sub-phrenic abscess, which was drained under CT guidance. A tube left in place which was removed later, subphrenic and pleural fluids cultures were sterile. Antimicrobial susceptibility testing showed that *P. luteola* was susceptible to most antimicrobials tested, including carbapenems, β-lactams-β-lactamase inhibitors, aminoglycosides, quinolones, tetracycline, tigecyclin, ampicillin, aztreonam, cephalosporins and co-trimoxazole but Cefixime was intermediate susceptible and colistin was resistant as tested by Versa Trek automated system (1 Thermo Fisher Way Oakland Village, OH 44146. USA).

The patient was started on imipenem-cilastatin 500 mg every 6 hours for 6 days with effervescence of fever and abdominal symptoms, and then she was switched to oral ciprofloxacin 750 mg every 12 hours, stayed on it for few days as an inpatient with progressive improvement. Then, she was discharged on same oral regimen. Two weeks after discharge she was evaluated as an outpatient, she was well with no complaints; follow up abdominal CT scan showed near complete resolution of the liver mass/necrosis and residual minimal amounts of fluids in the pleural space and sub-hepatic above right kidney. She was kept on oral ciprofloxacin for ten more days. Around seven weeks later she was phoned at home and was back in good condition.

**Discussion**

Although this patient was a “healthy” woman, her albumin was low on admission and lost weight secondary to her two weight-reducing surgeries, during which stomach ring was placed in the first surgery for four years. Our patient presented with a liver mass/abscess and sub-phrenic as well as pleural effusion. Only the biopsied liver tissue grew *P. luteola*, while both the sub-phrenic and pleural fluids did not grow any microorganism including *P. luteola*. The patient responded swiftly to imipenem therapy and later improvement was sustained on oral ciprofloxacin, without the need for outpatient parenteral antimicrobial therapy (OPAT).

To our knowledge, we could not find in literature a similar presentation i.e. liver mass/abscess with intra-abdominal and intra thoracic fluid effusions. However, most of adult human infections occurred in patients with indwelling devices, except in pediatric age group where it has been reported to occur without indwelling devices in a neonate where it caused sepsis syndrome, without an indwelling device [12].

An interesting point to note is that the current *P. luteola* isolate was resistant to colistin, similar to *Burkholderia cepacia* complex [14]. *P. luteola* was reported from a clinical isolate to have a novel β-lactamase gene, bla_{LUT}-1, that is chromosomally-encoded [1]. It was observed that this R-gene develop resistance to cephalosporins with variable susceptibility to penicillins. This β-lactam resistance phenotype suggests that this microbe may produce a natural β-lactamase; it may be used as a marker to differentiate strains especially in infection control [1]. Nevertheless, this microorganism may require to
be kept under surveillance for potential inclusion as emergent infectious pathogen in a nosocomial setting.

References