Pyogenic liver abscess caused by community-acquired multidrug resistance *Pseudomonas aeruginosa*

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A previously healthy 28-year-old man was admitted to our clinic with a two weeks history of pain in the right upper abdomen, followed by fever, chills, and vomiting. On admission day, physical examination revealed that he was in pain and feverish (38.6° C), and had hepatomegaly. Laboratory findings included total white blood cell (WBC) count of 14,800/mm³, erythrocyte sedimentation rate was 87 mm/h with a C-reactive protein (CRP) measuring 63 mg/dL (range, 0-8 mg/dL), and total bilirubin, alanine aminotransferase, aspartate aminotransferase, gamma glutamyl transpeptidase, alkaline phosphatase were within normal limits. Abdominal ultrasonography (US) showed solid mass at right hepatic lobe, which suggested a liver abscess, according to clinical findings. It was also confirmed by abdominal computed tomography (CT). The abscess was localized with transverse CT images and percutaneous drainage with 20 Fr gauge was performed. Fluid samples were sent for microbiological cultures. He was initially treated with ceftriaxone 2 g/day plus metronidazole 1 g/day with no improvement. After three days, the aspiration culture grew MDR *P. aeruginosa*. The bacteriological resistance results led us to change the initial treatment to imipenem 2 g/day plus amikacin 1.5 g/day for four weeks. The patient was discharged 21 days after admission without evidence of any complication.

Despite continuous improvement in image modalities, availability of potent antibiotics and advancement in the knowledge and treatment of PLA, morbidity and mortality remains high. Its incidence ranges from 0.008 to 0.0022% in hospitalized patients.^{2,3} The diagnosis of PLA was based on clinical findings and evidence from imaging studies, either abdominal US or CT. Treatment with intravenous antibiotics and application of catheter drainage or aspiration are the primary therapeutic strategies.

The most common pathogenic agents, which enter the liver by vascular routes, are *Escherichia coli, Klebsiella pneumoniae, Salmonella typhi*, Proteus vulgaris, streptococci, and staphylococci, but anaerobes may also be present.² Nevertheless, *P. aeruginosa* has long been regarded as a relatively rare pathogen of PLA, especially in patients without underlying conditions, such as lung and renal disease, malignancies, organ transplantation, immunosuppression.⁴ *P. aeruginosa* was defined as being MDR when the organism was resistant to all agents studied (ampicillin, amoxicillin-clavulanate, ceftriaxone, ceftazidime, cefepime, aztreonam, chloramphenicol, ciprofloxacin, gentamicin, piperacillin, and trimetoprim-sulfamethoxazole), except imipenem, amikacin, and colistina.⁵ When searching Pub Med, we find only one case report of PLA caused by *P. aeruginosa*, but it was not MDR.⁴ On the other hand, PLA caused by *P. aeruginosa* was seen 2-6% in literature,³ and this is the first case of PLA caused by community-acquired MDR *P. aeruginosa* in literature. The incidence of MDR *P. aeruginosa* increased in recent years. For this reason, we attach great importance to the rational use of antibiotics in inpatients and outpatients.

[Braz J Infect Dis 2010;14(3):218]©Elsevier Editora Ltda.

Submitted on: 03/04/2010 Approved on: 03/22/2010

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