## Case Report

# A rare association of liver abscess and rhabdomyolysis induced by *Klebsiella* oxytoca

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#### Abstract

Introduction: We report the case of a 76-year-old male who was hospitalized with severe dehydration, pain in the hepatic region, and weakness in the limbs.

Methodology: A contrast-enhanced abdomen CT and a contrast-enhanced ultrasound identified a large liver abscess. The patient underwent percutaneous drainage of the abscess.

Results: The culture examination, analyzed by multiplex polymerase chain reaction test, showed the presence of *Klebsiella oxytoca*. The laboratory report identified a resistance mechanism involving a plasmid-mediated SHV-1 extended-spectrum-beta-lactamase (ESBL).

Conclusions: *K. oxytoca* is a Gram-negative bacterium and is potentially associated with a large variety of infections. The association between the liver abscess by *K. oxytoca* and rhabdomyolysis had not yet been described in the literature.

Key words: Liver abscess; K. oxytoca; rhabdomyolysis; CEUS.

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### Introduction

*Klebsiella oxytoca* is a Gram-negative bacterium of the genus *Klebsiella* belonging to the family of *Enterobacteriaceae*, widely distributed in nature and present in the normal gut microflora [1] and has been detected in the stool of 8% to 10% of healthy adults by culture-based methods. *K. oxytoca* is potentially associated with a large variety of infections ranging from mild gastroenteritis to life-threatening bacteremia and meningitis [2]. Notwithstanding its relevance, the role of *K. oxytoca* is largely masked by its notorious relative, *Klebsiella pneumoniae* [3]. Here we report a case of liver abscess by *K. oxytoca*, an occurrence not frequently described in the literature.

### **Case report**

A 76-year-old man was admitted to our observation for falling to the ground due to weakness of the lower limbs. The patient presented with sepsis, severe dehydration, pain in the hepatic region, and weakness of the limbs while fever was not present. A head computed tomography (CT) scan and the neurological examination performed in the emergency room ruled out a stroke event. Medical history reported fever in the last month, but not on admission, hypertension, hyperlipidemia, diabetes mellitus, and chronic coronary artery disease previously treated with angioplasty. He assumed medical therapy consisting of bisoprolol, cardioaspirin, valsartan/hydrochlorothiazide, metformin, omeprazole.

Table 1 reported laboratory parameters on admission. The laboratory data suggested a picture of rhabdomyolysis and, according to Sepsis-3 criteria [4], a diagnosis of sepsis was made (3 points from a likely baseline of 0, due to a marked increase of serum creatinine). An intravenous 0.9% NaCl solution and an empirical antimicrobial therapy with piperacillin/tazobactam (4.5 g every 6 hours) were started. On the second day, an abdominal ultrasound exam was performed, and the presence of a complex lesion on the right liver lobe, with irregular coarse septa surrounding a large anechoic-necrotic area, was identified. A contrast-enhanced abdomen CT performed two days later, when the patient had normal serum

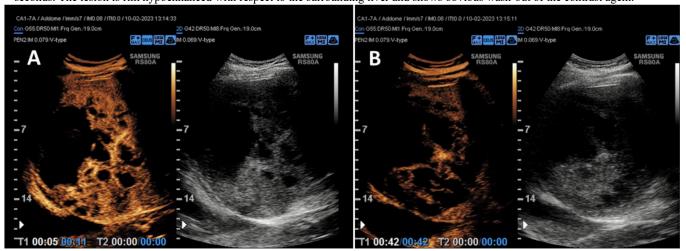
LAB TEST normal range (n.r.)	Three months earlier	AT admission	After 10 days	After 20 days
Procalcitonin (ng/mL) (n.r. $< 0.10$ )	-	74.10	2.04	0.34
CRP (mg/dL) (n.r. $< 0.50$ )	1.02	40.07	15.43	3.10
WBC (mm3) (n.r. 4000-10000)	8700	17700	10300	6700
Creatinine (mg/dL) (n.r. 0.7-1.2)	1.2	3.9	2.2	1.3
Azotemia (mg/dL) (n.r. 10-50)	65	178	116	68
Creatine kinase (UI/L) (n.r. 20-200)	150	19508	101	70
AST (UI/L) (n.r. < 40)	30	964	74	35
ALT (UI/L) (n.r. $< 41$ )	21	511	49	15

Table 1. Time course of biochemical data.

creatinine levels, confirmed the presence of a  $17 \times 13.5$ cm multiloculated formation with postcontrastographic peripheral enhancement and a perihepatic fluid layer. A contrast-enhanced ultrasound (CEUS) [5-8] showed enhancement in the arterial phase of the solid areas of the lesion while absent wash-in of the anechoic areas was compatible with large abscess formation of the right hepatic lobe (Figure 1). Meanwhile, antibiotic therapy was modified by replacing piperacillin-tazobactam with intravenous meropenem (2 g every 8 hours), considering the high prevalence of extended-spectrum beta-lactamases (ESBLs) in our region plus metronidazole (750 mg every 8 hours) to counter a potential amebic abscess, although risk factors were absent. A few days later the patient underwent percutaneous drainage [9,10] of the liver abscess after obtaining informed consent. Threehundred and fifty mL of cafe-au-lait-colored purulent liquid were aspirated for culture tests (three samples). Meanwhile, a percutaneous drainage was placed but was poorly tolerated by the patient. After the displacement of the drainage tube, he refused to undergo another interventional radiology procedure. The culture examination analyzed by multiplex polymerase chain reaction test (BioFire® FilmArray®

gastrointestinal panel, bioMerieux, USA) showed the presence of K. oxytoca and, in accord to the antibiogram generated by standard culture of material from drainage tube, meropenem was confirmed while metronidazole was discontinued. The laboratory report identified a resistance mechanism involving a plasmid-mediated SHV-1 extended-spectrum-beta-lactamase (ESBL). The antimicrobial susceptibility testing report showed resistance to third-generation cephalosporins and to piperacillin-tazobactam. Instead, blood cultures have always been negative, probably in consideration of the ongoing antimicrobial therapy. After 10 days of antimicrobial therapy, the serum procalcitonin values were rapidly decreasing, as well as the values of serum creatinine, creatine kinase, CRP, and WBC (Table 1). On the thirtieth day, he underwent ultrasound control which showed a reduction in the size of the liver abscess (about  $10 \times 8$  cm) with complete disappearance of the fluid portion. The creatinine values had returned to normal with a further reduction of CRP, procalcitonin, and WBC (Table 1). He denied permission for a surgical approach aiming at definitive source control, preferring conservative management. Unfortunately, despite the demonstrated efficacy of the therapy, the patient died about a month after hospitalization.

**Figure 1.** CEUS examination: A. Arterial-phase image obtained 11 seconds after contrast agent administration shows a heterogeneous rim hyperenhanced of the septa and peripheral of the lesion, with an irregular nonenhanced areas. B. Portal-phase image obtained at 42 seconds. The lesion is rim hypoenhanced with respect to the surrounding liver and shows obvious wash-out of the contrast agent.



### Discussion

Liver abscess is the most common type of visceral abscess presenting an incidence of only 2.3 cases per 100,000 population per year. Most liver abscesses are classified as pyogenic by bacteria or amoebic by Entamoeba histolytica while only a minority depend on parasites or fungus. Several bacteria such as Escherichia coli, Klebsiella spp., Streptococcus spp., Staphylococcus spp., and anaerobic pathogens are pyogenic responsible for abscesses. Instead, rhabdomyolysis occurs when muscle fibers, injured by diseases, trauma, or toxic substances, break down releasing their contents into the bloodstream. Common symptoms include muscle pain, weakness, and reddishbrown (tea-colored) urine.

In the literature, most cases of liver abscesses are due to other *Enterobacteriaceae spp.* [2,11,12]. The forms ascribed to *K. oxytoca* are usually secondary to abdominal surgeries, malignancies, end-stage liver or renal diseases, or immunocompromised conditions, such as renal transplantation and diabetes [2]. Our patient did not present any of these conditions.

At present, the association of liver abscess and rhabdomyolysis is described in only three previous reports where the investigated patients came from Eastern countries and the most frequent bacterium isolated was *Klebsiella pneumoniae* [13,14]. Thus, the identification of *K. oxytoca* infection in our case report is the first described case involved in this rare association.

Moreover, in our case *K. oxytoca* infection was complicated by a mechanism of resistance linked to the plasmid-mediated overproduction of SHV-1 ESBL linked with resistance to third-generation cephalosporins and to piperacillin-tazobactam.

In conclusion, we describe an aggressive infection by an ESBL-producing strain of *K. oxytoca*, an oftenneglected pathogen compared with the better-known *K. pneumoniae*. Despite appropriate antimicrobial treatment, the patient died, probably because of the lack of source control procedure.

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Conflict of interests: No conflict of interests is declared.