

Portal vein thrombosis secondary to *Klebsiella oxytoca* bacteriemia

Klebsiella oxytoca bakteriyemisine sekonder portal ven trombozu

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To the Editor,

Pylephlebitis, also called septic thrombophlebitis of the portal vein, is a life-threatening complication of intra-abdominal infection. It usually develops secondary to infection in the drainage area of the portal venous system [1,2].

Klebsiella spp are opportunistic pathogens that cause a wide spectrum of severe diseases such as septicemia, pneumonia, urinary tract infection, and soft tissue infection. *Klebsiella oxytoca* (*K. oxytoca*) has been isolated from human clinical specimens to a much lesser degree when compared to the other members of the genus *Klebsiella* [3]. Herein, we report a rare case of pylephlebitis secondary to *K. oxytoca* bacteriemia related with urinary infection, without any other pathology.

A 54-year-old male patient was hospitalized with hyperpyrexia, chills, general weakness, and dysuria for one week. He had been taking ampicillin-sulbactam orally for five days for a urinary system infection. The other medical history included essen-

tial hypertension and coronary artery disease. Family history was unremarkable.

On examination, he appeared unwell, body temperature was 39.5°C, blood pressure 120/70 mmHg, and pulse regular at 100/min. His liver was palpable 3 cm below the costal margin without tenderness. There was no jaundice, ascites or stigmata of chronic liver disease. No other abnormalities could be found.

Urea nitrogen and creatinine, partial thromboplastin time and prothrombin time were all within normal limits. Chest radiograph showed no abnormalities. There were 10-12/hpf (high power field) white blood cells, 3/hpf red blood cells and no bacteria in the urinalysis. Other laboratory test results are shown in Table 1.

After blood and urine cultures were performed, intravenous (i.v.) ampicillin-sulbactam 4 g/d was started. On day 3, *K. oxytoca* was isolated in the blood culture of the sample drawn at admission. In the antibiotic sensitivity testing, the microorganism was sensitive to ampicillin-sulbactam, so his treatment was continued. Urine culture was negative.

Table 1. Laboratory findings

	Lab. Findings	Normal Range
WBC (x10 ⁹ /L)	10.9	(4.4-11.3)
Neutrophil (%)	75	(37-73)
Platelet (x10 ⁹ /L)	363	(150-450)
Hb (g/dl)	12.9	(14-17.5)
CRP (mg/L)	314	(0-5)
AST (U/L)	59	(10-34)
ALT (U/L)	21	(7-33)
ALP (U/L)	405	(15-250)
GGT (U/L)	244	(8-61)
T. bilirubin (mg/dl)	3.03	0.2-1.3
HBsAg, Anti-HIV, Anti-HCV	(-)	
Anti CMV Ig M (IU/ml)	(-)	
Anti-EBV (VCA, EBNA) Ig M (IU/ml)	(-)	
Anti-CMV Ig M (AU/ml)	(-)	

ALP: Alkaline phosphatase; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CMV: Cytomegalovirus; CRP: C-reactive protein; EBV: Epstein-Barr virus; GGT: Gamma-glutamyl transpeptidase; HCV: Hepatitis C virus; HIV: Human immunodeficiency virus

On day 10, the patient had persistent fever, and echocardiography was performed, but no vegetation was shown. Abdominal ultrasound scan showed hepatomegaly, but a normal biliary tree with no focal liver abnormality and no evidence of intra-abdominal abscess. Due to worsening of his symptoms, gentamicin was added. Five days later, the patient deteriorated; ampicillin-sulbactam and gentamicin were stopped and meropenem (1 g i.v. every 8 hours) was started. Computed tomography (CT) scan of the abdomen, pelvis and thorax was performed. Although thorax CT appeared normal, abdominal CT revealed complete thrombosis of the main stem of the portal vein with a total occlusion, which was confirmed by color Doppler ultrasound. Small bowel and colon were normal in appearance, and there was no evidence of appendicitis or diverticulitis in the pelvis. Endoscopic study of the gastrointestinal tract showed no tumor, varices or diverticulum. Coagulation tests including for lupus anticoagulant, dysfibrinogenemia, resistance to activated protein C, deficiencies of protein S, C or antithrombin, and mutations of factor V were all detected as normal. Antiphospholipid and anticardiolipin antibodies were negative and the plasma homocysteine level was normal (normal value: 5-14 μ mol/L).

The patient was treated with meropenem for two weeks and heparin for 10 days (continuous

infusion of 25,000 IU/24 h after a bolus of 5000 IU), followed by oral anticoagulation. Within 10 days, duplex ultrasound showed partial recanalization of the thrombus. The fever did not recur. The patient made a complete recovery and was discharged home on day 26, at which time liver enzymes and C-reactive protein had normalized; he had been afebrile for three days. Two months later, follow-up Doppler ultrasound showed complete recanalization of the thrombus. He remained asymptomatic after discharge, with no abnormal findings on clinical examination, and warfarin was discontinued six months after diagnosis.

Portal vein thrombosis (PVT) is an infrequent but serious complication occurring in several diseases such as abdominal malignancy, infections or after surgical intervention. It is a complex situation that is associated with several risk factors including mostly acquired and inherited local precipitating factors. Approximately 8%-15% of cases are reported as idiopathic [4-6]. Intra-abdominal inflammatory conditions include bowel inflammatory disease, cholecystitis, pancreatitis, or intra-abdominal sepsis [7]. Our patient did not have an intra-abdominal infection but he had urinary infection complicated with bacteriemia. To the best of our knowledge, this is only the second case of pylephlebitis secondary to urinary infection in the literature; however, this is the first report in association with *K. oxytoca* bacteriemia.

Clinical manifestations of PVT have a wide spectrum ranging from no symptoms to acute massive hematemesis [8]. Our patient had a symptomless PVT, most probably of recent origin.

As there are no specific symptoms of PVT, imaging studies should be performed to confirm the presence of thrombus if PVT is suspected. Imaging studies include color Doppler-ultrasonography (CDU), CT, magnetic resonance imaging (MRI), and angiography. Fresh thrombus can go undetected by sonography because of the low echogenicity but can be recognized by (CDU) [9]. However, there were no pathologic findings in our patient's abdominal ultrasound, while his abdominal CT revealed complete thrombosis of the main stem of the portal vein, which was confirmed by (CDU).

Spontaneous repermeation of PVT is possible but uncommon. Anticoagulation is recommended in acute PVT as complete or partial repermeation can be achieved in up to 80% of patients. The reported recurrence rate of PVT ranges from 6% to 40%, so some researchers recommend anticoagu-

lant therapy be continued for at least six months [10]. No widely-accepted guidelines for anticoagulant therapy are available. To prevent portal hypertension with long-term complications and to accelerate resolution of the thrombus, our patient was treated with anticoagulants.

In summary, although rare, pylephlebitis remains a less-recognized entity with a high rate of mortality. We report a patient with urinary infection of *K. oxytoca* complicated by PVT. We emphasize the importance of clinical suspicion and early radiology for an early diagnosis of this entity, which makes it possible to establish an efficient treatment. Eradication of infectious foci and judicious administration of antimicrobials are essential to reduce the catastrophic morbidity and mortality of pylephlebitis.

Conflict of Interest

No author of this paper has a conflict of interest, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included in this manuscript.

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