

# Klebsiella oxytoca Bacteremia after Rotavirus Gastroenteritis in An Infant

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## Abstract

Rotavirus is an important health issue as a cause of diarrhea worldwide in children, especially under 5 years old. Despite the high frequency of rotavirus gastroenteritis, till date, rotavirus-associated secondary bacterial complications have rarely been reported in children. Herein, we share our experience of a case of acute rotavirus gastroenteritis in an infant complicated by *Klebsiella oxytoca* bacteremia.

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**Keywords:** *Klebsiella oxytoca*, rotavirus, gastroenteritis, infant

## Introduction

Rotavirus is the leading cause of severe acute gastroenteritis among infants and young children, being responsible for about 20% of diarrhea-related deaths in children under 5 years of age in developing countries (1, 2). It has been well documented in human and animal studies that rotavirus replicates in the gut and infect the enterocytes of the villi of the small intestine, leading to structural and functional changes in the epithelium. These changes facilitate bacterial translocation, leading to secondary bacteremia by microorganisms of the normal intestinal flora. Despite these changes and the vast number of endogenous bacterial flora, till date, only limited number of cases of secondary bacteremia caused by enteric organisms after rotavirus gastroenteritis in children has been reported in the literature (3-9).

Herein, to the best of our knowledge, we describe for the first time a case of *Klebsiella oxytoca* bacteremia after rotavirus gastroenteritis in an infant from Turkey.

ment with a 2-day history of fever, vomiting (seven episodes in 24 h), and diarrhea (10 loose stools in a day). Physical examination revealed a weight of 6.9 kg (25-50 p.) and a body temperature of 38.7°C. She appeared moderately dehydrated and the remaining examinations were unremarkable. Laboratory data were as follows: white blood cells: 18,400/mm<sup>3</sup>, platelets: 596,000/mm<sup>3</sup>, urea: 75 mg/dL, creatinine: 0.7 mg/dL, and C-reactive protein (CRP): 0.2 mg/L (Normal: 0-5 mg/L); serum electrolyte levels were in normal limits and venous blood gas analysis showed mild metabolic acidosis. Urine analysis was normal. The stool was completely fluid and watery with no solid particles. There was no blood or mucus in the stool examination. Stool rotavirus antigen (Genx®, Diamed-Lab, İstanbul, Turkey) was positive. Normal hydration status was achieved by initial: 0.9% NaCl (20 mL/kg) within 30 min and maintenance: 0.45% NaCl daily requirement plus deficit within 24 h. The patient also received supportive therapy, which were probiotics and zinc. On the following day, fever and the hydration status were in normal ranges. Stool frequency dropped down to 4 times a day. On the third day of admission, fever rose to 39.3°C, the patient became lethargic with no focus of fever. Laboratory tests were repeated and revealed that leukocytosis (16,400/

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## Case Report

A previously healthy 8-month-old female infant was admitted to the emergency depart-



mm<sup>3</sup>), thrombocytopenia (103,000/mm<sup>3</sup>), and a high level of CRP (109 mg/L). To exclude central nervous system infection, cerebrospinal fluid (CSF) was obtained. The microscopic examination revealed no cells and the protein, glucose, and chlorine levels were 25 mg/dL, 60 mg/dL, and 130 mEq/L, respectively. Blood, urine, and CSF cultures were taken before initiating cefotaxime (200 mg/kg/day). Blood and urine cultures showed growth of *K. oxytoca*, which was susceptible to the antibiotic regimen prescribed. The CSF culture showed no growth. Control blood and urine cultures obtained 72 h after the initial treatment and showed no growth. Fever was normal after 72 h of treatment. The patient received a total 14-day course of antibiotics. The patient became clinically stable without fever and diarrhea as well as with a good appetite. One month after her discharge, she was completely asymptomatic and has also continued to show normal growth. Parental approval was obtained.

## Discussion

In children, rotavirus-related gastroenteritis burden is a common health issue, especially in developing countries. Despite the high frequency of rotavirus gastroenteritis, till date, rotavirus-associated secondary bacterial complications have rarely been reported. The paucity of the reports describing rotavirus-associated secondary bacteremia is probably related to the lack of awareness of this complication and apparently its rarity as well as failure to obtain blood cultures later in the course of rotavirus gastroenteritis. Here we describe an infant with rotavirus gastroenteritis who developed a recrudescence of fever with no obvious focus several days after admission. This clinical course forced us to obtain blood culture and thus, we detected *K. oxytoca* bacteremia.

*K. oxytoca* is a part of the normal intestinal flora. These facultative anaerobes, gram negative rods are located all over the intestine, especially in the small intestine (10). The local intestinal response to rotavirus and the secondary bacterial invasion mechanism have not been well understood yet. Mucosal damage and blood flow redistribution due to vasoactive agents are likely parts of rotavirus gastroenteritis that render the small intestine vulnerable to bacterial invasion. The secondary infection has been tried to be explained by the same mechanism as that seen in bacterial lung infection caused by *Streptococcus pneumoniae* secondary to lower respiratory tract infection (1-3). Despite these explanations, a definitive interaction between rotavirus and intestinal mucosa or enteric bacterial flora as well as the mechanism(s) responsible for secondary bacteremia remain to be explained. Adler et al. (3) and Lowenthal

et al. (4) described first cases of rotavirus-associated secondary bacteremia caused by *Escherichia coli* and *K. pneumoniae*. After this, several case reports have been published with various enteric gram-negative bacteria (5-9). In these reports, patients typically showed a recurrence of high body temperature on day 3 or 4 of admission without an obvious focus of infection, and they were initiated on early broad-spectrum antibiotics and treated uneventfully. In our patient, we encountered the same pattern of fever and obtained a fast response to broad-spectrum antibiotics. The majority of cases reported till date consisted of infants. One can conclude that infants seem to be prone to secondary bacteremia after rotavirus gastroenteritis. Although several mechanisms have been proposed to estimate this vulnerability, the mechanism underlying secondary bacteremia after rotavirus gastroenteritis needs to be clarified with a large number of case studies (3-10).

## Conclusion

In conclusion, *K. oxytoca* bacteremia should be kept in mind in infants during the course of rotavirus gastroenteritis when an increase in body temperature with no apparent source of fever is detected. In these patients, blood cultures should be obtained and empiric antibiotic treatment should be initiated until the culture results.

**Informed Consent:** Written informed consent was obtained from the parents of the patient who participated in this case.

**Peer-review:** Externally peer-reviewed.

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