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.

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.

Case Report
A previously healthy 8-month-old female infant was admitted to the emergency department 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/mm³, platelets: 596,000/mm³, 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/
bacterial invasion mechanism have not been well under -

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ary bacteremia after rotavirus gastroenteritis. Here we describe an infant with rotavirus gastroenteritis and 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 pneu-

moniae 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 bacte-

remia remain to be explained. Adler et al. (3) and Lowenthal et al. (4) described first cases of rotavirus-associated sec-

ondary 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 antibiot-

ics. 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 esti-

mate this vulnerability, the mechanism underlying second-

ary bacteremia after rotavirus gastroenteritis needs to be clarified with a large number of case studies (3-10).

**Discussion**

In children, rotavirus-related gastroenteritis burden is a common health issue, especially in developing coun-

tries. Despite the high frequency of rotavirus gastroenteri-

tis, 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. 

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**Conclusion**

In conclusion, K. oxytoca bacteremia should be kept in mind in infants during the course of rotavirus gastroen-

teritis 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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