

Rotavirus Rare Complications; Elevated Liver Enzymes and Hemorrhagic Cerebral Infarction: Case Report

Rotavirus'ün Nadir Komplikasyonları; Karaciğer Enzim Yüksekliği ve Serebral Hemorajik İnfarkt

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ÖZET Rotavirus gastroenteriti infant ve genç çocuklar arasında şiddetli diareye sebep olan etkenlerden biri olup tüm dünyadaki diareden ölüm nedenlerinin %25'inden sorumludur. Rotavirus bir Reoviridae sınıfından olup çift sarmal RNA cinsinden bir virus türüdür. Rotavirus gastroenteriti şiddetli dehidratasyon ve elektrolit dengesizliğine sebep olur. Bu enfeksiyon genellikle gastrointestinal sistemde lokalize olup, enfeksiyonun burada kendini sınırladığı bilinmektedir. Rotavirus gastroenteriti nadiren de olsa intestinal sistem dışı belirtilerle de ilişkili olabilmektedir. Bu olgu sunumunda rotavirus gastroenteritini takiben nadir, fakat potansiyel olarak ciddi karaciğer enzim yüksekliği, kanama bozukluğu, uzamış PT, aPTT ve dissemine intravasküler kuagülasyon (DIC) ile bağlantılı hemorajik serebral infarkt anlatılmıştır.

Anahtar Kelimeler: Rotavirus; komplikasyon; karaciğer fonksiyon testleri; serebral enfarkt

ABSTRACT Rotavirus is one of the leading cause of severe diarrhea among infants and young children and responsible for more than 25% of all deaths from diarrhea worldwide. Rotavirus is a genus of double-stranded RNA virus in the taxonomic family Reoviridae. Rotavirus gastroenteritis can cause severe dehydration and electrolyte imbalance, but it is usually known to be localized in the gastrointestinal system as a self-limited infection. Rotavirus gastroenteritis associated with unusual extraintestinal manifestations. We described in this case report, rare but potentially serious complication with hepatic transaminase elevation bleeding disorders, prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT) and cerebral hemorrhagic infarct associated with disseminated intravascular coagulation (DIC) following rotavirus gastroenteritis.

Key Words: Rotavirus; complications; liver function tests; cerebral infarction

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Case 1- A 12 month-old-boy with no previous medical history, approximately 9.5 kg admitted to the emergency department with a 3-day history of recurrent fever (axillary temperature 39°C) and intermittent episodes of agitation, confusion and convulsion. In the emergency department he had extensor (rigid) response, decerebrate posture, sunken eyes, dry mouth and tongue, dry skin and fever was 38.5°C. Initial investigation revealed the following laboratory values: hemoglobin level, 11.4 g/dL; leukocyte count, 8.5×10^9 cells/L (69% neutrophils, 25.2% lymphocytes, 5% monocytes, 0.4% eosinophils, and 0.4% basophils); and platelet count, 110×10^9 cells/L, C-reactive protein 19 mg/L, ESR 17 mm/h, serum

glucose 81 mg/dl, liver function tests revealed abnormal values of transaminases AST 1150 U/L, ALT 870 U/L. Vital signs included heart rate of 135 beats·min⁻¹, blood pressure ^{70/45} mmHg, respiratory rate of 32 breaths·min⁻¹, and hemoglobin oxygen saturation (SpO₂) of 91% in room air. Auscultation of lungs revealed bilateral equal air entry. When the abdominal wall examined 1.5-2 cm below the costal margin liver palpated. Abdominal USG showed minimal hepatomegaly. The child was transferred to the intensive care unit with mouth-to-mask oxygen, decision to intubate was taken on the basis of decreased GCS (E₂M₂V₃). The child was intubated with size 3.5 cuffed orotracheal tube and put on mechanical ventilation. Cardiovascular and respiratory monitoring was carried out and further management was carried. Central venous catheter was inserted and he was treated with empiric antibiotic therapy and IV acyclovir therapy for possible herpes simplex virus (HSV) encephalitis, and with anticonvulsants.

Specific diagnosis of infection with rotavirus A was made by identification of the virus in the patient's stool by enzyme immunoassay. When examined cerebrospinal fluid normal findings detected. Initial CT of the brain yielded normal results. Bacterial cultures of blood, urine, CSF samples showed no growth. Acyclovir therapy was continued for 2 weeks, despite negative viral culture.

6 days after the onset of illness showed the following values: leukocyte count, 21 × 10⁶ cells/L (63% lymphocytes, 18% neutrophils, and 19% monocytes); Blood coagulation studies showed Abnormal prolong prothrombin time (PT) and activated partial thromboplastin time (aPTT) (at 2.0-2.5 times the normal PTT/APTT values), decreased blood platelet count 85 × 10⁹ cells/L and prolong bleeding time associated with DIC was found. Immediately Prolong PT and aPPT normalized within three days after the administration of vitamin K and fresh frozen plasma 10 mL/kg/day.

We detected that our case unaware of what's happening intensive care and child's field of vision affected was found. On 7th day second CT of the brain yielded hemorrhagic cerebral infarct. He re-

quired mechanical ventilation for 18 days. After 24 days of intensive care, the patient maintained a normal vital and mental status and was discharged to the pediatric service and 30th day he discharged at home.

Case 2- A 15 month-old girl was admitted to the emergency department with a 3-day history of fever, abdominal discomfort, and watery diarrhea, nausea and vomiting. The child was dehydrated and had diffuse abdominal tenderness. Physical examination she had incomprehensible speech, withdraws from pain. Initial laboratory findings only abnormal AST levels 850 IU/L and ALT levels 550 IU/L were found. Cranial tomography and abdominal ultrasonography not to determined pathologic findings. The child was transferred to the intensive care unit and followed with mask oxygen, and not required entubation. Specific diagnosis of infection with rotavirus A was made by identification of the virus in the patient's stool by enzyme immunoassay. Standart with empiric antibiotic therapy and IV acyclovir therapy were given via intravenous line. The child experienced normal development with neither physical nor neurologic sequelae. After 13 days of hospitalization the child was discharged, with decreased liver enzymes and no residual deficit.

DISCUSSION

Human Rotavirus infection is an important cause of gastroenteritis in children. The incidence of rotaviral disease is similar in developed and developing countries but the number of deaths is higher in developing countries. Each year rotaviruses cause approximately 111 million episodes of gastroenteritis in children, which result in 25 million visits to clinics, 2 million hospitalizations, and 352.000 to 592.000 deaths. On a worldwide basis, nearly every child experiences rotavirus gastroenteritis by age 5, 1 in 5 visits a clinic, 1 in 65 is hospitalized, and 1 in 293 dies. Children in the poorest countries account for 82% of rotavirus deaths¹ The disease has a seasonal occurrence with the majority of the cases being diagnosed between the months of December and June. This disease burden underscores a need for interventions such as vaccines. A

vaccine was developed and approved, but recommendation for its use was withdrawn because of vaccination-associated adverse events² Some clinical case reports suggested that rotaviruses could be found at extraintestinal sites following infection . Some studies reported that rotavirus has been associated rarely with other syndromes, include the finding of virus in the liver following fatal disease,³ the finding of elevated liver enzymes associated with virus infection^{4,5} some studies showed that rarely rotavirus including respiratory infection, necrotizing enterocolitis (in infants), pneumatosis intestinalis, hepatic abscess, and pancreatitis. However, a rotavirus etiology has not been proven.⁶ Recent studies confirm sporadic case reports that rotavirus infection is not confined to the intestine as was generally assumed. Rotavirus spread to extraintestinal tissues has been renewed.⁷ Rotavirus antigens (antigenemia), RNA, or infectious virus (viremia) has been demonstrated in the serum and many extraintestinal tissues in all experimental animal models.⁸ It appears that some of this spread depends on viral factors, but other components of the spread may be related to viremic hosts. It remains unclear if viremia is, in any way, related to diarrheal disease. It is important to determine if systemic infection with rotavirus is responsible for,

or plays a role in, clinical syndromes not currently associated with rotavirus.⁹

Recently, there have been reports with various neurological presentations or central nervous system complications, including convulsions and encephalopathy, in association with rotavirus infection.¹⁰⁻¹² The frequency of central nervous system involvement in cases of rotavirus gastroenteritis remains unknown, but may be as high as 2% for children under 2 years of age.¹³

CONCLUSION

Rotavirus infections are an important cause of child morbidity and mortality in all of the world. A large number of clinical case reports suggested that rotaviruses could be found at extraintestinal sites following infection. In our cases we thought that rotavirus responsible for extraintestinal sides affects. Because after rotavirus gastroenteritis, we detected that affected liver and elevated liver enzymes. In one case bleeding disorders, prolonged PT, aPTT and cerebral hemoragic infarct was seen associated with DIC following rotavirus gastroenteritis. Physician should be alert to rare but potentially serious complication of rotavirus gastroenteritis.

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