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Case Report

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Paracetamol related autoimmune hemolytic anemia in a child with rotavirus infection

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ABSTRACT

The pathogenesis of antibody directed against erythrocytes induction is unknown, in autoimmune hemolytic anemia (AHA). Connective tissue disorders, lymphoproliferative disorders, neoplasms, chronic inflammatory diseases, drugs, and infections are considered as etiologic factors. We presented a child who developed AHA with concomitant presence of rotavirus infection and paracetamol use. She had no side effect of paracetamol in history. To our knowledge, this is the first reported case of AHA caused by paracetamol used in child who infected with rotavirus.

1. Introduction

Autoimmune hemolytic anemia (AHA) is a condition characterized by destruction of erythrocytes induced by immunoglobulin G and/or immunoglobulin M type of antibodies, which bind to red blood cell surface antigen intravascular or in the reticuloendothelial system (Gehrs and Friedberg, 2002; Chaudhary and Das, 2014). The hallmark of this group of diseases is the positive result of the direct antiglobulin (Coombs) test, which detects a coating of immunoglobulin or components of complement on the erythrocyte surface. It is described as idiopathic or due to connective tissue disorders, lymphoproliferative disorders, neoplasms, chronic inflammatory diseases, drugs, and infections (Chaudhary and Das, 2014).

Rotavirus infection is the most cause of acute gastroenteritis in all age groups. The most important life-threatening complications are dehydration and/or electrolyte imbalances (Aldemir-Kocabaş et al., 2013). Although it can lead to hemolytic anemia,

we could not find any report that AHA caused by rotavirus infection. Additionally, we could not find any report regarding AHA caused by paracetamol. In this paper, we present a patient developed autoimmune hemolytic anemia with use of paracetamol during the course of rotavirus gastroenteritis. However, we could not find any study reporting AHA caused by paracetamol in rotavirus infections. To the best of our knowledge, this is the first report that AHA caused by paracetamol ingestion during rotavirus infections.

2. Case report

An 11-year-old girl admitted to our clinic with weakness, fatigue, fever, vomiting and diarrhea of seven days duration. It was reported that she received supportive care at home with daily management of dietary and fluid intake. However, she developed dark urine, pallor, fatigue, and jaundice after paracetamol treatment for the last three days. Also, it was mentioned that her eight months-aged brother had vomiting and diarrhea at the same period. On physical examination; her general condition was moderate. She had pale skin, icteric sclera, 123/min heart, 2/6 systolic murmur, and palpable spleen with 2 cm below the costal margin. Laboratory: White blood cells (WBC): $3.2x10^9$/L, hemoglobin (Hb) 5.2 g/dL, platelet: $446x10^9$/L, mean corpuscular volume (MCV): 121.9 fl, red cell distrubition width (RDW): 29, reticulocytes: 2%, direct Coombs: (++), total bilirubin 4.7 mg/dL, indirect bilirubin 4.3 mg/dL, laktat dehidrogenaz (LDH): 1037 U/L (N=0-247), haptoglobulin: 7.6 mg/dL (N:22-164), and urine urobilinogen was (+++). Peripheral blood smear revealed that 51% neutrophils, 29% lymphocytes, 4% monocytes, and 16% normoblasts (Fig. 1a). Anisocytosis, polychromasia, poikilocytosis, macrocytosis, and spherocytes were shown. Atypical cells were not observed. Vitamin B12, folic acid and transferrin saturation were within normal limits. Bone marrow aspiration smear was found to be hyperactive in the erythroid series; abnormal blastic activity was not observed (Fig. 1b).

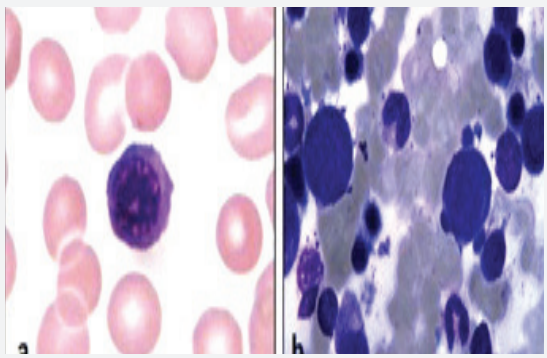

Fig. 1. Normoblast is seen in peripheral blood smear (a). Erythroid hyperactivity is seen in bone marrow smear (b).

Autoimmune screening kit and antinuclear antibody tests were negative. Our case and siblings diagnosed as gastroenteritis were positive for rotavirus antigen in the stool (with immunochromtographic method, Toyo Diagnostics test, the sensitivity is 99.9%). All cultures and viral serologic tests revealed no other infectious agents. Prednisolone (1 mg/kg/day, PO) was administered with a diagnosis of AHA and supported with folic acid, probiotics, and fluid and electrolyte therapy for rotavirus gastroenteritis. The clinical outcomes of the patient was began to progress. A week later, laboratory findings revealed that Hb 9 g/dL, MCV 112 fl, LDH 284 IU/L, indirect bilirubin: 0.48 mg/dL, urine urobilinogen negative, direct Coombs test (+), and haptoglobin: 27.4 mg/dL. On the tenth day of the treatment, hemoglobin level rose to 10 g/dL. The dose of steroid was begun to tapering and patient was discharged. At the control, after 20 days of the treatment, Hb level rose to 12 g/dL, and no evidence of hemolysis was found on peripheral blood smear.

3. Discussion

Mycoplasmas, parvovirus B19, EBV, CMV, Rubella, Hepatitis A, Hepatitis B, and Hepatitis C viruses were identified as aethiological injectious agents for AHA (Lambert and Nydegger, 2000; De Villartay et al., 2005). Our case represented AHA symptoms after a seven days of acute gastroenteritis. When the other secondary aethiological causes of AHA was excluded it is very likely to link the clinical symptoms of the patient with her Rotavirus gastroenteritis .

Rotavirus infection is the most source of gastroenteritis that affects all socioeconomic groups in all age groups in the world (Aldemir-Kocabaş et al., 2013). Although it's a common disease, the lack of a case that the cause of AHA in the literature, the explanation of the cause anemia with this infection alone is difficult in our case. Findings of jaundice were emerged in the last three days with the use of paracetamol during gastroenteritis, in our case. Thus, the drug is thought to be as a trigger agent. However, there is not a strong evidence of AHA caused only by paracetamol in the literature. There was the use of paracetamol during rotavirus infection, in our patient. Therefore, the association of rotavirus gastroenteritis with use of paracetamol was the overt cause of AHA. Our patient had used paracetamol previously. We considered that paracetamol use or rotavirus infection solely could not induce the AHA, however; it might be triggered by paracetamol taken in a case with rotavirus infection.

The auto antibody may be produced as an inappropriate immune response to an erythrocyte antigen or to another antigenic epitope similar to an

erythrocyte antigen known as “molecular mimicry”. Alternatively, this coincidence paracetamol with rotavirus infection may alter the erythrocyte membrane so that it becomes “foreign” or antigenic to the host.

Conclusion

According to the best of our knowledge, this is the first case associated with AHA due to coincidence with paracetamol used in a child infected with rotavirus, in the literature.

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