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A case of neonatal amoebiasis with after-birth vomiting and bloody stool

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E. histolytica has a simple life cycle with two stages: an infective cyst and an invasive trophozoite. It lives on humans as its host. Its infection occurs through the ingestion of the cyst form, and the disease begins when the trophozoite, converted at the small intestine, adheres to colonic epithelial cells with a latent period of two days to four months. In some instances, amoebic abscess formations can occur at the liver, lung, brain, or spleen via the lymphoid system. Rare cases of amoebiasis in neonates have been reported, much less any intrauterine infections in the world that may have occurred during the gestation period. We've recently experienced a case of neonatal amoebiasis that entailed after-birth vomiting and bloody stool. The infant seemed pre-infected with *E. histolytica* before birth. **(Korean J Pediatr 2007;50:1257-1260)**

Key Words : *Entamoeba histolytica*, Infection, Infant, Newborn

Introduction

Amoebiasis is a parasitic disease infected by oral ingestion of *Entamoeba histolytica*. It is associated with over 50 million cases of symptomatic diseases, and is the third leading parasitic cause of death, second only to malaria and schistosomiasis. Amoebic colitis is the most common form of symptomatic infection with a notably poor prognosis in young children and an even worse one for infants[1-4]. Only rare cases of amoebiasis in infants have yet been reported in Korea, nor have any intrauterine infections that may have occurred during the gestation period.

We ve recently experienced a case of neonatal amoebiasis that entailed after-birth vomiting and bloody stool. Our case, in which the infant seemed to have been infected with *E. histolytica* before birth, has been examined with three entry scenarios 1) Transplacental passage during gestation: unlikely due to theoretical infeasibility; 2) Infection through maternal genitalia right before birth: though feasible due to the premature rupture of amniotic membrane, considering the minimum latency period of two days, the reason that the infant, born by Cesarean, vomited blood at birth is not clear; 3) After inoculation and colonization of *E. histolytica* in the uterus through the maternal lymphoid system, the parasite invaded directly into the amniotic fluid and infected it, and the infant ingested the infected fluid (the most feasible scenario). This paper reports the case with relevant references.

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Case Report

Patient : One-day-old female

Chief complaint : After-birth currant jelly-like vomiting and bloody stool

Present illness : The patient was at birth 2.9 kg, female, born by Cesarean at 38 weeks of gestation because of a premature rupture of amniotic membrane.

Family history : The second baby in the family. Seven days before delivery, the mother suffered abdominal pain and diarrhea after eating swine intestines stuffed with rice and vegetables (a dish commonly known as Soon-dae in Korea). The symptoms lasted for two days. The mother had not received any medical intervention for her symptoms before she was hospitalized for delivery.

Physical examination : The patient's vital signs were all normal. Her abdomen was soft and flat with decreased bowel sound, and with no evidence of organomegaly. Other examinations of chest, central nervous system and skin were

all shown to be normal.

Initial laboratory data : Her CBC at admission was normal (Hb 18.7 g/dL, Hct. 56.4%, platelet 167,000/mm^3, WBC 8900/mm^3 with segmented neutrophils 47%, band forms 10%, lymphocytes 28% and monocytes 10%). Biochemistry showed blood sugar (208 mg/dL), BUN (10.4 mg/dL), serum total protein (5.1 g/dL), Na (135 mEq/L), K (5.1 mEq/L), Ca (9.2 mg/dL), P (6.7 mg/dL), AST/ALT (37/8 U/L) and LDH/CK (832/196 U/L). PT/PTT were prolonged (15.6/46.7 sec) and CRP was 11.1 mg/L (Ref :0-5). A urine test was normal, and a TORCH test was negative. Chest radiographs showed no particular abnormality. Abdominal radiographs showed no specific findings except gas in the colon and stomach.

A stool exam revealed positive occult blood with many leukocytes. Stool rotavirus was negative. A culture of gastric juice and blood was negative. Meckel s scan and abdominal CT were normal.

Progress & Laboratory Data : The patient was initially administered a fluid infusion with antibiotics and Vit K, and was maintained without enteral feeding. Six hours after birth, her bloody mucoid stool and vomiting were still continuing, and decreased bowel sound and abdominal distension were revealed. Her bloody stool persisted until 10 days after birth (Fig. 1).

A stool exam that was done three days after birth showed trophozoites of E.histolytica (Fig. 2, 3). Stool CK19 (cytokeratin 19) was positive. Gastric juice aspirated six days after birth also showed trophozoites of E.histolytica (Fig. 4). The patient was treated with metronidazole (oral dose, 30-50 mg/kg/day, for 10 days) from the fifth day after birth.

Fig. 1. She showed bloody mucoid stool one hour after birth.

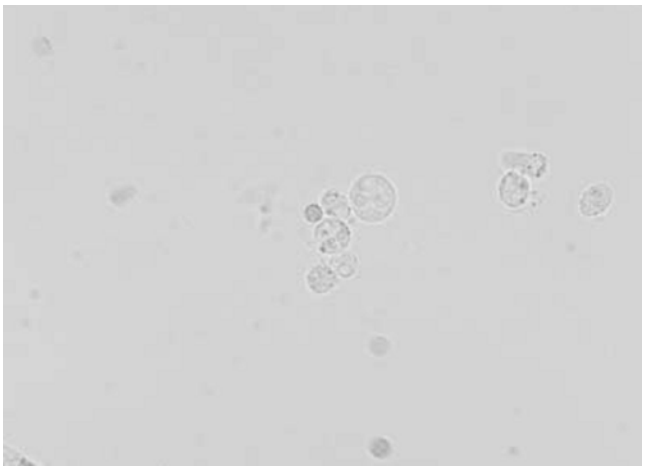

Fig. 2. The trophozoite of *E. histolytica* found in the neonate's stool (Direct wet mount ×400).

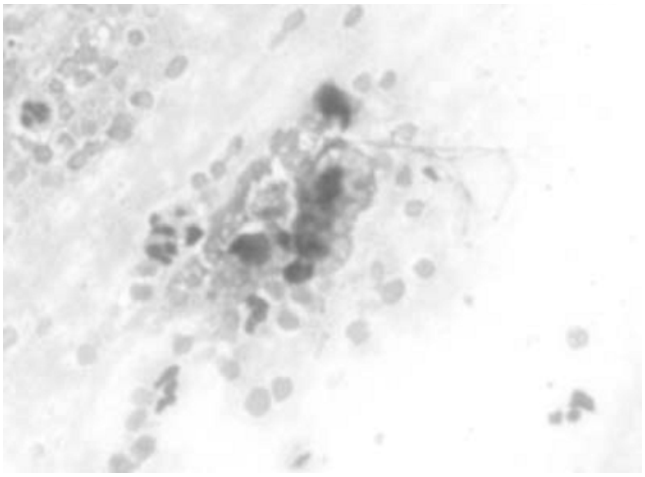

Fig. 3. Gram stain shows the trophozoite of *E. histolytica* with many leukocytes (Gram stain ×400).

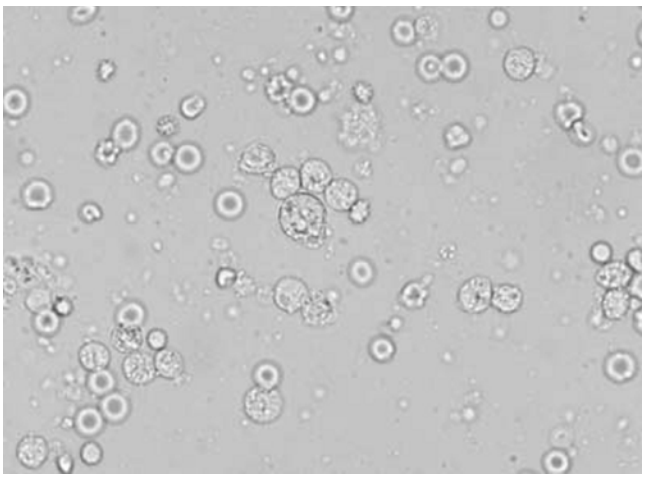

Fig. 4. The trophozoite of E. histolytica found in the neonate's gastric juice (Direct wet mount ×400).

Her stool was normalized in contour on the ninth day after birth and after fourteenth day, stool exams were negative for occult blood, leukocytes, and amoeba. The results of the laboratory exams - such as stool exams, an indirect hemagglutination test, CBC, chemistry, and abdominal CT - on the patient's mother all were negative. The patient was discharged in 24 days without complications and has been followed up until recently.

Discussion

Amoebiasis was first reported in 1875, by the St. Petersburg physician Fedor Aleksandrovich Lösch, who described amoebic trophozoites in the stool and colonic ulcerations of a farmer with a fatal case of dysentery[4]. In the following years, much knowledge has been gained about the clinical manifestations of *E. histolytica* and effective treatments for the disease have been developed[5]. The parasite, however, still infects approximately 10% of the world population annually about 50 million in total, from which approximately 100,000 people die of the disease. It s a prevalent disease in developing countries on the Indian subcontinent, South Africa, the Far East, and South and Central America. In endemic areas, about 25% of the population has antibodies to *E. histolytica* from previous asymptomatic infections, and fecal-oral spread of infection is quite frequent[1-3]. In our case, the disease is not endemic to the city of Bucheon, and the city s water supply facilities are known to be safe. And the mother of the patient had never drunk contaminated water. The Soon-dae that the patient s mother had eaten 7 days before delivery was suspected to be the source of the infection, but this couldn t be proven. The mother showed no symptoms before or after delivery, and there were no fecal amoebae found in her stool.

The infection of *E. histolytica* usually occurs in the colon, and *E. histolytica* cysts change into trophozoites which attach to the colonic mucous glands, epithelial cells, and to leukocytes by galactose-inhibitable adherence lectin. Once attached to the colonic epithelia, the trophozoites release a proteinase that causes ulcers[6, 7]. The organisms penetrate beneath the submucosal tissue, and spread laterally to produce flask-shaped ulcers and amoebic colitis. In some cases, the intestine-resident amoebae reach the liver through the lymphatic vessels and portal vein to cause amoebic liver-abscess and produce abscesses in the lungs, pleura, brain, and spleen[2-3, 5, 8]. This may imply that amoebae move in lymphatics and can create colonies in an environment that offers proper fuel.

Hosts, however, do not passively provide places for amoebic proliferation. They activate defence mechanisms such as interleukin, neutrophil, macrophase, and complement system[9, 10]. But, in acute invasive amoebiasis, the responses of T lymphocytes are limited by various causes created by the parasite.

Amoebic death rates vary: they are less than 1% in the case of uncomplicated liver-abscess, greater than 50% in fulminant amoebic colitis, greater than 15% but less than 20% in pleurisy, near 40% in pericarditis, and greater than 90% when the brain is infected. Higher death rates are common among infected infants and babies. Those patients with pregnancy, malignant tumors, or malnutrition, and who take adrenocortical hormones show bad prognoses[5, 11, 12]. Our case, too, shows that the rapidly multiplied amoebae in the pregnant woman have created colonies in the uterus through the lymphatics.

Though no cases of amoebic colonization within the uterus have been reported, we saw a strong possibility that an amoebic colony, after having ruptured, had flowed directly into the amniotic fluid.

It s generally not easy to examine trophozoites in feces for diagnosis, especially in the case of diarrheal stool that usually starts autolyzing approximately 30 minutes after defecation, as this further hinders analysis. And even when fecal analysis is not obstructed, methods such as EIA (enzyme immunoassay), IHA (indirect hemagglutination antibody), and ID (immunodiffusion)[8, 13, 14] may not be of great help to neonates. We detected amoebic trophozoites in our patient s stool three days after birth-amoebiasis was the last diagnosis for a neonate who was born in a non-endemic area and had been kept NPO right after birth.

Amoebiasis has a bad prognosis for infants, and we presume that, if our case could be proven to be an intrauterine infection, it would have been highly fatal. The patient was lucky in that metronidazole worked well for her, and she has shown no relapse since being discharged.

한 글 요 약

출생 직후 혈변과 구토 증상이 시작된 Amoebiasis 신생아 1례

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저자들은 출생 직후부터 심한 혈변과 구토 증상을 보인 신생아

의 위액과 변에서 아메바를 확인하여 자궁 내에서 감염된 것으로 판단되는 신생아 아메바증을 경험하고 치료하였기에 문헌 고찰과 함께 보고하는 바이다.

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