



Liver Microabscesses in a Premature Neonate With Eosinophilic Colitis

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ABSTRACT

Hepatic abscesses in premature infants are rare with less than 100 case reports documented in literature. We report a case of a premature infant diagnosed with hepatic microabscesses secondary to eosinophilic colitis. A 33 4/7-week preterm female neonate presented with bilious emesis, abdominal distention, and severe hematochezia. Eosinophilic enterocolitis was suspected. Hypoechoic regions in the anterior liver identified on computed tomography were considered liver microabscesses. This unique case exemplifies how prematurity increases the risk of mucosal damage in the presence of eosinophilic colitis causing enteric bacteria to seed into the liver through the portal vein, resulting in hepatic microabscesses.

INTRODUCTION

Eosinophilic colitis is an uncommon condition originally identified by Kaijser in 1937.¹ Etiology is incompletely understood. Although hematochezia is a common complication of eosinophilic colitis, an uncommon complication of hepatic microabscesses arose in this case. To the best of our knowledge, this is the first case report that documents hepatic microabscesses in a patient diagnosed with eosinophilic colitis, underscoring a unique aspect of our patient's clinical presentation.

CASE REPORT

A 33 4/7-week, 2.22-kg female baby was born through spontaneous vaginal delivery after premature rupture of membranes to a 26-year-old G3P2 mother. Physical examination was unremarkable at delivery, and she stoolled spontaneously within the first 24 hours of life. Maternal breast milk (MBM) enteral feeds were started by gavage on day of life (DOL) 1. She was made nil per os on DOL 4 after episodes of bilious emesis.

Physical examination demonstrated distended abdomen tender to palpation with no rebound and normoactive bowel sounds. Neonate's physical examination was otherwise unremarkable; genitourinary examination did not reveal anal fissures. Laboratory evaluation on DOL 4 demonstrated leukocytosis ($21.1 \times 10^9/L$) and eosinophilia (14%). Frank bloody stools developed by DOL 5. Serial abdominal x-rays revealed nonspecific bowel gas fullness and no evidence of pneumatosis. Upper gastrointestinal series ruled out malrotation, and because bilious emesis resolved by DOL 5, our focus shifted to newly developed hematochezia. Abdominal distention resolved by DOL 6, but leukocytosis, eosinophilia, and hematochezia persisted. Family history was notable for sister with milk protein intolerance who developed hematochezia after introducing milk. This raised suspicion of milk protein-related allergic eosinophilic enterocolitis in our patient. Our differential included necrotizing enterocolitis (NEC), milk protein-induced allergic enterocolitis, transient eosinophilic colitis, intussusception, colonic ischemia, Meckel's diverticulum, and Hirschsprung's enterocolitis.



Figure 1. Sagittal grayscale ultrasound image demonstrates hypoechoic lesions (arrows) in anterior right hepatic lobe.

Broad-spectrum antibiotics ampicillin and cefepime were started on DOL 5. By DOL 7, vancomycin replaced ampicillin after abdominal ultrasound (AUS) to evaluate for ischemia, intussusception, and colitis, which showed several sub-centimeter hypoechoic, hypovascular liver lesions in the anterior liver characterized as microabscesses. Findings on AUS included scattered loops of thick-walled bowel with minimal ascites, sludge-filled gallbladder, and no intrahepatic or extrahepatic biliary ductal dilatation (Figure 1). No pneumatosis, pneumoperitoneum, portal venous gas, or complex ascites made NEC unlikely. Stool testing for infectious causes of enterocolitis was not performed because of no known nosocomial exposures. Sepsis was unlikely because neonate did not meet SIRS criteria, and she appeared well.

An abdominal computed tomography (CT) scan with contrast on DOL 8 elucidated findings on abdominal ultrasound. The CT revealed irregular hypodensities in the anterior liver, consistent with AUS findings of microabscesses, along with mild mucosal hyperenhancement and bowel wall thickening of the rectosigmoid (Figure 2). On DOL 8, Flagyl replaced ampicillin and vancomycin to treat the microabscesses. On DOL 10, Flagyl was stopped, and Zosyn started to broaden anaerobic coverage. Antibiotics were continued for 10 days total. She had persistent leukocytosis and eosinophilia with peak white blood cell count of $44.0 \times 10^9/L$ on DOL 10 and a striking eosinophil count of 20% or above from DOL 6 to 10. The infant exhibited hematochezia for 6 consecutive days (DOL 5–10) likely reflecting significant inflammation. This caused continual decline in hemoglobin (8.9 g/dL) and hematocrit (25.7%), requiring packed red blood cell transfusion



Figure 2. Contrast-enhanced computed tomography image in the sagittal plane demonstrates mildly hyperenhancing thick-walled descending colon (arrows).

by DOL 10. The hematochezia coupled with uptrending white blood cells and eosinophil count even after MBM was discontinued for more than a week reinforced a diagnosis of eosinophilic enterocolitis and not milk protein-induced allergic enterocolitis. Intestinal inflammation leading to enteric bacteria seeding into the liver likely caused hepatic microabscesses, which resolved on completion of broad-spectrum antibiotics. This made NEC and milk protein-induced allergic enterocolitis less likely. To minimize subjecting the infant to invasive tests, endoscopic evaluation was not performed because the patient improved after broad-spectrum antibiotics and transfusion. If there had been no improvement, endoscopy with biopsy would have been performed to assist in making a definitive diagnosis.

Repeat AUS on DOL 13 showed resolution of hypoechoic liver lesions. On DOL 24, unfortified MBM feeds were restarted after the mother eliminated milk-based products from diet. The

patient was discharged on DOL 43 at 39 3/7-week corrected gestational age on full oral feeds of unfortified MBM supplemented with 2 feeds of 22 kcal/oz elemental formula/day.

The infant's clinical presentation, laboratory test results, and diagnostic tests strongly supported a diagnosis of eosinophilic colitis and ruled out other possibilities (allergic enterocolitis and NEC).

DISCUSSION

Approximately 75% of patients with eosinophilic colitis reportedly have strong family history of cow's milk allergy,^{2,3} similar to our patient. Studies propose a non-Immunoglobulin (Ig) E-mediated hypersensitivity reaction to milk protein, causing a chronic immune reaction localized to colonic mucosa, leading to inflammation and eosinophilic infiltration.⁴ In utero transplacental sensitization of the colon by maternal bovine milk antigens contributes to early development of eosinophilic colitis.⁵ Colonic biopsy typically reveals eosinophils, lymphoid nodules, and infiltration into the intestinal tissue.⁴ One case report details a clinical presentation and laboratory values similar to our patient.⁶ The group did not perform a biopsy because the diagnosis of eosinophilic colitis was based on a diagnosis of exclusion. Treatment involves complete elimination of milk protein from diet of both infant and mother. Diet adjustments within first year of life can cause eosinophilic colitis to self-resolve in most cases.⁴

No literature to date has documented hepatic microabscesses in patients with eosinophilic colitis. Hepatic abscesses are rare in the preterm infant with <100 neonatal case reports in the literature. Prematurity is an important risk factor.⁷ Hepatic abscesses can form through ascending infection from the umbilical or portal veins, hematogenous spread from biliary tree, or direct spread from a neighboring site.⁷ Gram-negative cocci, Gram-positive bacilli, and *Candida* have all been identified as causative pathogens.⁷ This microbial diversity for hepatic abscesses supports bacteria seeding from a colonic infection through the portal vein into the liver. Hepatic abscesses are best visualized using ultrasound and CT with contrast.⁸ Therapy with broad-spectrum antibiotics is indicated for resolution.

This case exemplifies challenges in diagnosing eosinophilic colitis in premature infants and complications that may arise (hepatic microabscesses). A striking aspect was the development of hypochoic lesions in the liver consistent with microabscesses. Previous case reports^{5,6} detail similar

clinical presentation, but do not identify hepatic involvement, underscoring a unique aspect to this case.

DISCLOSURES

Author contributions: All authors meet ICMJE criteria. O. Parks and B. Mahmood conceptualized the manuscript. O. Parks drafted the initial manuscript and revised the manuscript in collaboration with the other authors. B. Mahmood, J. Squires, and M. Keith contributed to the manuscript design and interpretation of laboratory results in the case report. All authors critically revised the manuscript. All authors approved the final revised manuscript and agree to be accountable for all aspects of the work. B. Mahmood is the article guarantor.

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Informed consent was obtained for this case report.

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