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Case Report

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Blood in Stool in The Immediate Neonatal Period

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Abstract

The presence of blood in stool in the immediate neonatal period is of serious concern to the parents and the care providers. Hematochezia describes the appearance of bright red blood coming from the anus. In neonates, there is a diverse array of etiologies that can cause this disease presentation. Etiology could be as simple as swallowed maternal blood, or it could be a serious surgical or potentially life-threatening situation. Our goal is to discuss the differential diagnosis for hematochezia in neonates along with the clinical presentation, management, and outcomes for each. We are describing two infants that presented with blood in stool in the immediate neonatal period.

Introduction

Hematochezia is a clinical diagnosis based on the observation of bright red blood presumed to be coming from the rectum. The blood can be gross or streaked. In contrast to melena, hematochezia usually indicates blood coming from a source involving the distal gastrointestinal tract and is typically bright red because the blood has not yet oxidized [1]. In neonates, the differential diagnosis can include anal fissure, milk protein allergy, swallowed maternal blood, vitamin K deficiency, gut malrotation with volvulus, and necrotizing enterocolitis (NEC) [2, 3, 4].

Case presentations

Case 1

Baby A was a 2.34 Kg, male infant who was born prematurely by a spontaneous vaginal delivery at 34 weeks of gestation. Mom is a 32-year-old female, gravida one. The pregnancy was conceived by in vitro fertilization and was complicated by pre-eclampsia. She had a history of hypothyroidism and infertility. The medications taken during pregnancy were levothyroxine, magnesium sulfate, and prenatal vitamins. Maternal urine drug screen was negative. The infant was vigorous at birth and was exhibiting mild expiratory grunting with subcostal and intercostal retractions. APGAR scores were eight and eight at one and five minutes respectively. The infant was initially on bubble CPAP of 6 cm on admission to the neonatal intensive care unit. He was weaned off CPAP within 24 hours of admission. A nasogastric tube was placed and feeding protocol was initiated. The infant was initially on intravenous fluids with dextrose and amino acids. The complete blood counts, serum electrolytes, and arterial blood gas were unremarkable. Chest X-ray on the day of birth showed mild RDS with mild haziness and increased interstitial markings, normal lung volumes and air-filled loops in the abdomen. Feedings were introduced with Enfacare 15 ml every three hours. On the 3rd day of life, the patient appeared jaundiced with the total bilirubin 13.8; phototherapy was initiated. On the evening of the 6th day of life, the patient had frank bloody stools during diaper change. There was no abdominal distension or tenderness of the abdomen. Vital signs were stable. Plain anteroposterior view abdominal X-ray showed small rounded lucencies of the ascending and descending colon suspicious for stool or early pneumatosis. The complete white count was in normal range, the differential count showed 57% lymphocytes, C-reactive proteins were less than 5 mg per liter, and stool APT was negative for fetal hemoglobin. The patient was started on vancomycin and gentamycin, feedings were withheld, and the infant was on started on hyperalimentation. Antibiotics were discontinued when follow up abdominal X-rays were reported negative for any pneumatosis, blood culture and urine culture were negative. The infant had normal examination and the feedings were restarted and advanced as per protocol.

Case 2

Baby B was 2280 grams, male infant, born by a repeat C-section, delivered at 36 weeks and 3days of gestation. Mom is 26-year-old mother, gravida three, para 2002 with one prior C-section delivery. The pregnancy was complicated by monochorionic diamniotic twin gestation, fetal growth retardation, and maternal depression. She was taking sertraline during pregnancy. Her urine drug screen was negative.

The infant was vigorous at birth with APGARs of 7 and 9 at 1 and 5 minutes, Initially, he was showing signs of labored breathing. The patient was placed on bubble CPAP 6cm with 21% FiO2 and admitted to the NICU for respiratory distress and intrauter-

ine growth retardation. The patient did well on bubble CPAP and maintained oxygen saturation above 95%. The infant was started on intravenous maintenance fluids with Dextrose 10% and calcium gluconate was started. Orogastric tube was placed, and he was started on feeding protocol with Enfacare 22 calories and breast milk. CBC, electrolytes, and arterial blood gases were unremarkable. Chest Xray showed minimal interstitial streaking and perihilar opacities and air-filled loops of bowel throughout the abdomen. The patient was weaned off CPAP at approximately 24 hours after birth. Respiratory distress was attributed to transient tachypnea of the newborn. On the evening of the 6th day of life, the patient developed blood-streaked stool. He was getting feeds of Enfacare at 3 hours intervals. Vital signs were stable, infant was in no distress, the abdomen was soft, non-tender and the bowel sounds were present. There was skin breakdown and irritation with blood localized to the left side of the anus. Complete blood counts were normal, C-reactive proteins were in normal range, serum electrolytes were in normal range. The abdominal Xray was showing a normal bowel gas pattern. Stool lactoferrin was positive. Patient was switched from Enfacare feeds to Nutramigen 20 calories. No subsequent hematochezia was reported.

Discussion

Presence of blood in stool requires careful assessment of maternal history and detailed assessment of the newborn infant. The first step in assessment is getting a detailed maternal history, details of the conception or pre-pregnancy surgical history, maternal use of prescribed or non-prescribed medications, any associated or pre-existing medical conditions, any history of previous pregnancies with eventful outcomes. It is important to get the delivery room record, any instrumentation during delivery, maternal sedation required, or any medications administered during labor.

The next step is a detailed physical examination for any respiratory distress, vital signs to include blood pressure, any skin rashes, bruises or erythema, assessment of skin perfusion, assessment of muscle tone and activity and complete neurological examination. It is imperative to examine abdomen for distention, tenderness, bowel sounds, or any mass. Anal opening should be carefully examined for any fissures or abnormality [19]. Begin by observing how much blood is in the stool and its color.

Detailed physical examination should focus on evaluating for acute changes in skin perfusion status that could indicate cardiovascular instability [20]. Laboratory assessment includes basic chemistry, complete blood counts, blood and urine culture, and blood type in case a transfusion is needed.

Once the patient is stable, rule out inflammatory causes by obtaining C-reactive protein, albumin, and fecal lactoferrin. Other laboratory works up includes APT test for presence of maternal hemoglobin. Abdominal X-ray is the first imaging option to be pursued and can also be followed up with an ultrasound or contrast enema if indicated.

if symptoms appear hours to days later [6, 7], skin-prick testing, and Ig E serum testing are available but usually not needed. If milk protein allergy is suspected, first try swapping feeds to hypoallergic formula [7, 8]. Nutramigen, a strictly hydrolyzed formula consisting of Casein protein instead of milk protein, is an option. Prognosis for milk protein allergy is favorable. Most infants are successfully treated with avoidance of milk protein for the first 2-3 years of life [7, 8].

Anal fissures are tears in the rectal mucosa that can cause pain and bleeding. The cause is usually trauma from passing stools or during delivery [5]. Clinical presentation in neonates usually involves mild bleeding with blood-streaked stool. Look for localized lesions, ulcerations, or blood around the anus [4, 5]. Blood coming from lesion only is a diagnostic sign. Anal fissures usually heal on their own and usually do not require any operative intervention [2, 5]. If the fissure is secondary to constipation, re-evaluate the infant's volume status and ensure that the infant is getting the appropriate volume of fluids from feeds.

Milk protein allergy describes an immune response to proteins found in milk. Incidence is reported to be in 2% -7.5% of all infants and 0.4% -2% in exclusively breastfed infants [6]. It can be further differentiated into IgE and Non IgE reactions.

In IgE reactions, basophils and mast cells crosslink pre-sensitized IgE bound to the protein allergen [6]. This triggers an inflammatory cascade mediated by inflammatory cytokines that can cause the immature intestinal mucosa in neonates to bleed [6, 7]. Symptoms appear rapidly after feeding, usually within an hour [7]. Common findings include urticaria, vomiting, diarrhea, and wheezing. Anaphylaxis is rare but possible.

Non IgE reactions resemble a type IV hypersensitivity reaction because they are typically delayed for 2 hours to days and involve allergen specific T cells [6, 7]. It is thought that the accumulation and expansion of CD4/8 T cells and eosinophils in the colonic mucosa causes stretching, thinning, and subsequent bleeding. This type has also been reported to account for up to 46% of cow milk allergy [7]. Clinical presentation includes transient enteropathy, hematochezia, and failure to thrive secondary to nutritional deficiency.

Diagnosis of milk protein allergy is usually contextual. Consider IgE reaction if symptoms occur within minutes to hours of feedings and consider non IgE mediated reaction if symptoms appear hours to days later [6, 7]. Skin-prick testing and Ig serum testing are available but usually not needed. If milk protein allergy is suspected, first try swapping feeds to hypoallergic formula [7, 8]. Nutramigen, a strictly hydrolyzed formula consisting of Casein protein instead of milk protein, is an option. Prognosis for milk protein allergy is favorable. Most infants are successfully treated with avoidance of milk protein for the first 2-3 years of life [7, 8].

Swallowed maternal blood occurs when the infant swallows maternal blood during delivery. The infant will then pass the blood into the stool, usually several hours later. All other parameters will be normal including volume status, complete blood count, and hemoglobin. The definitive test is called the APT test or alkaline denaturation test [9]. This test is based on the different rates of denaturation of fetal versus adult protein when exposed to sodium hydroxide. A positive value indicates that the blood originated from the neonate. Generally, swallowed maternal blood is a benign condition. In rare cases, large amount of swallowed blood could

be a strong irritant to the bowel and could potentially predispose to colitis.

Vitamin K Deficiency should also be considered when encountering bloody stool in a neonate. The corresponding disease is called vitamin K deficiency with bleeding (formerly called hemorrhagic disease of the newborn). This disease is rare and can present anytime from the time of birth up to 2 weeks of life [11]. Early signs will usually include deep bleeding into the abdomen or cranium. Classical signs include bleeding and bruising around the umbilicus, gastro-intestinal bleeding, bruising around venipuncture sites, and bleeding associated with circumcision [10, 11]. Advanced disease presentation includes retinal hemorrhages and intracranial hemorrhages which has an associated 20% mortality rate [11]. Even though it is routine for newborns to be supplemented with vitamin K before birth, many neonates are admitted to the hospital with limited history. Vitamin K may be missed in home delivered infants. Thus, vitamin K deficiency should still be considered. Diagnosis is made by analysis of prothrombin time (PT) greater than 4 times the normal limit [10]. Treatment consists of the administration of vitamin K IM or IV [12].

Midgut volvulus arises from congenital malrotation that can cause pinching and ischemia of the gut wall which can lead to hematochezia. The most common presentation includes bilious vomiting, abdominal pain and distension, and changes in bowel movements. The incidence of malrotation is approximately 1 in 1000 births and up to half will involve volvulus of the midgut [13]. It is also associated with other conditions such as congenital diaphragmatic hernia, gastroschisis, intestinal atresia, Hirschsprung's disease, and situs inversus. Diagnosis is usually made by an abdominal Xray to rule out a distal obstruction [14, 15]. Upper GI series is the preferred method of confirmation. CT scan is usually not needed in neonates. Ultrasound can be performed but has low sensitivity comparatively. The mainstay of treatment is the Ladd procedure [16]. It involves detorsion of the affected segment, lysis of any restricting bands of tissue, and expansion of the mesentery to maximize the distance between the duodenum and cecum [15, 16]. The primary complication of malrotation is volvulus and the primary complication of volvulus is ischemia and necrosis. If untreated, the bowel necrosis that results from volvulus can be fatal.

Necrotizing enterocolitis describes intestinal inflammation that often leads to necrosis [17]. A single unifying cause has not been identified but it is thought that imbalances in the intestinal microbiome, immaturity of the gut wall, and inexperienced immune responses of the neonate contribute to a vigorous inflammatory response that can ultimately result in necrosis of the affected bowel segment [18, 19]. Premature infants are the most affected; especially those with low birth weight (<1,500 g). Only 10% of cases are attributed to term neonates [17]. There has been a reported prevalence of 5-10% in very low birth weight infants [17, 18]. Clinical presentation often includes feeding intolerance, abdominal distension and tenderness, hematochezia, visible bowel loops, bradycardia, lethargy, hypotension, temperature changes, cyanosis, and mottling of the skin [19]. When found in term neonates, they are usually already admitted to the intensive care unit for another reason [17]. Diagnosis is confirmed with an abdominal x ray that shows air in the wall of the bowel (pneumatosis intestinalis) [19]. Other signs include gas in the portal venous system and pneumoperitoneum. Evaluation should also include CBC with differential, CMP, CRP, lactate, coagulation studies, and an arterial blood gas [19]. The treatment for NEC is largely supportive. Making the patient NPO, giving total parental nutrition (TPN), and performing gastric decompression via NG tube will decrease the workload on the gut and promote healing. Fluids and broad-spectrum antibiotics are also standard of care. Surgery is indicated in 30-50% of cases. Frank bowel perforation is an absolute indication for surgery. Other concerning signs that could likely need surgery include the presence of gas in the portal system, discoloration of the bowel wall, persistent bowel looping on Xray, worsening thrombocytopenia, and hemodynamic instability.

Sridhar et al, have reported infantile hepatic hemangioendothelioma and associated necrotizing entero-colitis presenting in a preterm newborn with abdominal distention, altered nasogastric aspirates, and hematochezia [21]. The typical triad of presentation is multiple enlarging cutaneous hemangiomas, hepatomegaly, and congestive cardiac failure. Anemia and thrombocytopenia may be seen, caused by trapping of thrombocytes within the hemangioendothelioma with consumptive coagulopathy. Infantile hemangioendothelioma appears as a complex, mostly solid hepatic lesion. The condition has an excellent prognosis, especially with spontaneous regression after the first year of life. Medical therapy includes steroids, interferon, vincristine, and propranolol to accelerate the natural involution of the mass and radiation therapy or chemotherapy, as well as supportive care for congestive heart failure and coagulopathy.

Neonatal salmonella infection and hematochezia has been reported in two infants on day one of life [22]. These infants had relatively asymptomatic presentation with blood in stool. Salmonella was resistant to ampicillin, and they were treated with ten days course of cefotaxime. Heterotopic gastric mucosa can be present outside of the stomach and has been reported with very early–onset inflammatory bowel disease with recurrent gastrointestinal bleeding, chronic inflammation, and stricture in a newborn patient. It commonly occurs with Meckel diverticulum and gastrointestinal tract duplication [23]. Technetium-99m pertechnetate scan is helpful to make the diagnosis. Hirschsprung disease, Henoch-Schonlein purpura and intussusception are unlikely etiologies for hematochezia in the immediate neonatal period. Stress gastritis occurs in up to 20% of patients cared for in neonatal intensive care units. Prematurity, respiratory distress, and mechanical ventilation are all associated with stress gastritis. Indomethacin used for patent ductus arteriosus in neonates, may cause GI bleeding through intestinal vasoconstriction and platelet dysfunction. Maternal drugs easily cross the placenta and can influence growing infant. Aspirin, cephalothin, and phenobarbital are well-known causes of coagulation abnormalities in neonates. Stress ulcers in preterm infants can be associated with dexamethasone therapy. Stress ulcers may require treatment with omeprazole, ranitidine, sucralfate, or famotidine [24]. Viral infections such as salmonella, shigella, yersina, herpes or cytomegalovirus can lead to infectious colitis. In a rare case of congenital adenovirus infection, a three-day infant has been reported with hematochezia and thrombocytopenia [25].

Baby A's presentation is consistent with swallowed maternal blood.

He was stable and physical exam was normal at the time that the hematochezia was noted, sepsis was a less likely diagnosis. Xray showing nonspecific inflammatory changes that could have represented early pneumatosis warranted the initiation of antibiotics until a follow up Xray showed an improvement without evidence for systemic inflammation. There was no abdominal distension, normal bowel sounds were present, CRP was in normal range, and complete blood counts were normal. APT confirmed the diagnosis. A negative APT means that the observed blood had no fetal hemoglobin and therefore came from the mother. The maternal blood could also have corrosive effects on the colonic mucosa and may have caused the inflammatory changes noted on Xray.

Baby B's presentation is consistent with perianal fissure. According to our diagnostic process we have discussed, this specified diagnosis could be confirmed very early on. After the initial hematochezia was recognized, a physical exam was performed which revealed irritation and skin breakdown with bleeding. The additional workup this patient received was purely precautionary and further reinforced the diagnosis. There was no evidence of systemic infection and no acute findings on abdominal Xray.

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