Fortified milk: a rare cause of intestinal obstruction in pre-term patients

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ABSTRACT

Introduction. Intestinal obstruction secondary to the use of fortified milk is a rare cause in pre-term patients.

Case report. We present the case of a female pre-term newborn admitted as a result of abdominal distension and rectal bleeding, which mimicked necrotizing enterocolitis. On abdominal X-ray, she had an obstruction pattern, and on ultrasonography, echogenic masses at the distal ileum were observed. Given the lack of improvement with conservative management, urgent exploratory laparotomy was decided upon. At surgery, compact milk masses at the level of the distal ileum were identified as the cause of intestinal obstruction. Appendicostomy and lavage with saline solution through the ileocecal valve were performed. This allowed milk masses to come out towards the colon, and a great amount of acholic stools to be expelled.

Conclusion. The increase in "milk curd syndrome" cases should lead us to consider this cause in the differential diagnosis of intestinal obstruction in pre-term newborns fed with fortified milk.

KEY WORDS: Milk curd syndrome; Infant, pre-term; Intestinal obstruction; Lactobezoar.

LECHE FORTIFICADA: UNA CAUSA INFRECUENTE DE OBSTRUCCIÓN INTESTINAL EN PACIENTES PREMATUROS

RESUMEN

Introducción. La obstrucción intestinal secundaria al uso de leche fortificada es una causa infrecuente descrita en pacientes prematuros.

Caso clínico. Presentamos el caso de una recién nacida prematura que ingresa por distensión abdominal y rectorragia, simulando una enterocolitis necrotizante. En la radiografía abdominal presenta patrón obstructivo y en ecografía se identifican masas ecogénicas en íleon distal. Dada la no mejoría con manejo conservador, se decide laparotomía exploradora urgente. En la intervención se detectan masas compactas de leche a nivel de íleon distal como causa de la obstrucción intestinal. Se realiza apendicostomía y lavado con suero fisiológico a través de la válvula ileocecal, permitiendo salida de moldes hacia colon y expulsión de gran cantidad de heces acólicas.

Conclusión. El repunte de casos de "milk curd syndrome" nos obliga a considerar esta causa en el diagnóstico diferencial de obstrucción intestinal en prematuros alimentados con leche fortificada.

PALABRAS CLAVE: Síndrome de la leche espesa; Prematuros; Obstrucción intestinal; Lactobezoar.

INTRODUCTION

Intestinal obstruction caused by compact masses of non-digested milk (or lactobezoar) was first described in 1959[1], whereas the term "milk curd syndrome" was coined in 1969[2]. Most cases described were full-term newborns, fed with cow milk rich in casein and fatty acids. Once fat and casein contents in the milk were reduced, the prevalence of intestinal obstruction secondary to this syndrome decreased to near zero. In the last decades, however, intestinal obstruction cases as a result of the use of fortified milk in pre-term patients have emerged[3].

The increase in pre-term newborn viability in the last decades has caused fortified milk preparations to be used at an ever younger age. This, along with the intestinal immaturity of pre-term neonates, can explain the rise in milk curd syndrome cases. These patients are characterized by delayed gastric voiding and slow bowel transit, as well as decreased digestive secretions for adequate milk digestion. Therefore, the fortified preparations used to replace baseline requirements can precipitate within the gastrointestinal tract and form compact masses, thus causing obstruction[4].

CASE REPORT

We present the case of a 3-month-old female patient referred to our institution from her reference hospital with blood remnants in the stools and abdominal distension.

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Regarding obstetric and perinatal history, extreme prematurity was notable, since the patient was born by Cesarean section at gestational week 26+3. She was the second twin of a dichorionic diamniotic gestation that resulted from an embryo transfer in-vitro fertilization (IVF).

Pregnancy was under control, with prenatal normal ultrasonographies up to gestational week 24, when she required inhibition and intrauterine diversion as a result of sac prolapse, with subsequent rupture and initiation of uterine dynamics at week 26.

The APGAR score was 4 and 7 at minutes 1 and 5 of age, respectively. She required ventilation with continuous positive pressure, which was gradually reduced until full removal on day 28 of life.

Weight at birth was 945 g, with an adequate weight gain following enteral nutrition initiation in the first hours of life, which allowed for progressive fortification up to 4%.

The patient was discharged at 3 months of age, with a chronologic age of 39+3 gestational weeks and a weight of 2,550 g.

She presented at the emergency department of her reference hospital 24 hours following discharge with blood remnants in the diaper. Her family was not able to tell whether they were of urinary or fecal origin. She also had moderate abdominal distension, with no further symptoms.

In the reference hospital, an urgent blood test was performed, with decreased hemoglobin levels, mild neutropenia, and mild monocytosis standing out. No acute-phase reactant increase was noted. In addition, X-rays revealed a distension of small bowel loops and a "breadcrumb" pattern in the right hemiabdomen (Fig. 1).

In light of the clinical worsening and complementary test results, the Pediatrics and Pediatric Surgery Department of our institution was contacted by the other hospital, and the patient was referred for assessment purposes.

At admission, a nasogastric probe was placed, and a nothing-by-mouth diet with intravenous fluid therapy was decided upon. Empiric antibiotic therapy with amikacin, metronidazole, and teicoplanin was initiated and maintained for 7 days.

A control blood test was conducted. It revealed a decrease in hemoglobin levels vs. the previous one, leukopenia, and mild neutropenia. A small increase in C-reactive protein (CRP) levels, which were previously normal, was also detected.

To complete the study, an abdominal ultrasonography was carried out. It revealed a segmentary dilatation of the distal ileum, with echogenic content within it and a caliber change proximal to the ileocecal valve (Fig. 2).

In light of the clinical signs and complementary tests, suspected diagnosis was lower intestinal obstruction, proximal to the ileocecal valve, with the cause of it being unknown.

Clinical progression was unfavorable. She had progressive clinical worsening, with an increase in abdominal distension, little reactivity, and an earthy skin color. As a result of this, along with blood parameter deterioration and imaging test findings, urgent exploratory laparotomy was decided upon.

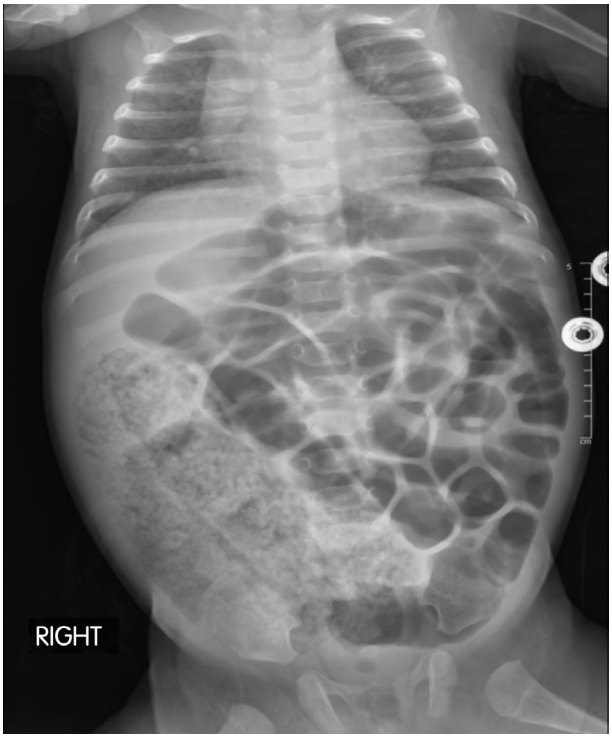

Figure 1. Abdominal X-ray: "breadcrumb" pattern and proximal loop dilatation.

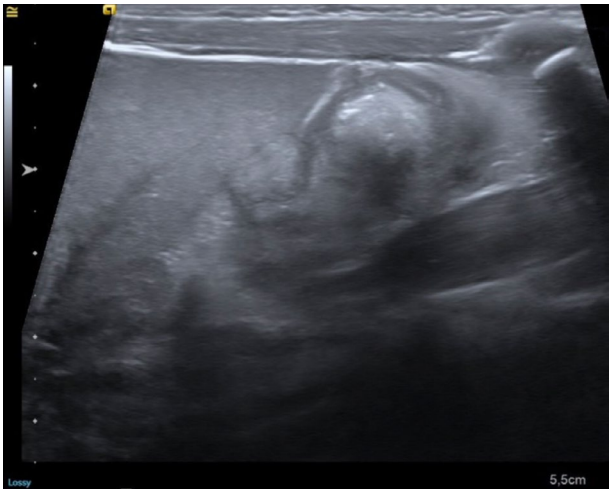

Figure 2. Abdominal ultrasonography: hyperechogenic masses within the small bowel.

At surgery, moderately dilated small bowel loops were noted, especially at the level of the distal ileum, where non-digested milk masses were identified (Fig. 3) as the cause of the obstruction. Appendicostomy and lavage with

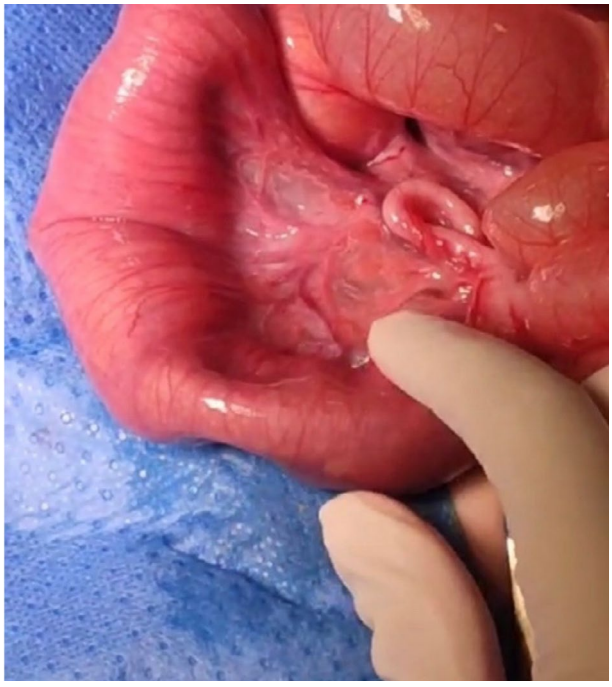

Figure 3. Surgical image. Dilated small bowel loops. Non-digested milk masses identified within the intestinal lumen as the cause of intestinal obstruction.

saline solution through the ileocecal valve were carried out until the masses disintegrated and came out towards the colon. Subsequently, a Nursing rectal lavage was conducted, which allowed a large amount of acholic stools to be expelled.

Following surgery, the patient had a favorable progression. She required red blood cell concentrate transfusion and oral iron administration, with blood parameters subsequently improving. Parenteral nutrition alone was administered until postoperative day 3, when enteral nutrition –with hydrolyzed milk– was initiated, which allowed for a progressive increase in oral feeding and weaning of parenteral nutrition until discontinuation, with a good tolerance and an adequate weight gain.

DISCUSSION

Maternal breastfeeding alone does not provide pre-term patients with sufficient proteins, electrolytes, and micronutrients. Therefore, they require fortified preparations to satisfy metabolic needs[5]. The increase in pre-term newborn viability, along with the intestinal immaturity of these patients, has led to a rise in intestinal obstruction cases secondary to the formation of compact non-digested milk masses[6], also known as the "milk curd syndrome."

Even though most cases described are pre-term newborns and neonates with a low weight at birth –typically under 1,000 g–, cases of full-term patients, even with small amounts of enteral nutrition, have also been published. Therefore, this cause should be considered in the differential diagnosis of food intolerances.

Abdominal distension is the most frequent clinical presentation. Sometimes, it can be associated with vomit and abdominal wall erythema, which mimics necrotizing enterocolitis[7], with a simultaneous alteration of blood parameters. It should be highlighted that, in certain cases, intestinal perforation can lead to severe complications such as peritonitis or septic shock.

Radiologically speaking, the "breadcrumb" pattern as a result of non-digested milk mass occupation, with proximal loop dilatation, is characteristic, as in our case. If diagnosis is unclear, abdominal ultrasonography can be performed, which will allow the intestinal lumen occupied by compact hyperechogenic masses to be visualized.

Conservative management consists of intravenous fluid therapy and parenteral nutrition until resolution, as well as fortified milk removal.

In some of the cases published, water-soluble contrast enemas have allowed for obstruction resolution, but this can increase the risk of intestinal perforation. Consequently, routine use is not recommended[8-10]. Surgery should only be conducted in patients who do not improve with conservative management, or in those who develop complications such as intestinal perforation.

Considering the increase in cases noted over the last years, and even though this is a rare cause, it should be included in the differential diagnosis of intestinal obstruction in pre-term patients fed with fortified milk, since early diagnosis can prevent highly severe and even fatal complications.

Further studies are required to determine which amount of calcium, proteins, and fatty acids is tolerable in the gastrointestinal tract of pre-term patients so that precipitation of these substances, and therefore secondary intestinal obstruction and its consequences, are avoided[11].

REFERENCES

1. Wolf RS, Bruce J. Gastrotomy for lactobezoar in a newborn infant. J Pediatr. 1959; 54(6): 811-2.
2. Cook RC, Rickham PP. Neonatal intestinal obstruction due to milk curds. J Pediatr Surg. 1969; 4(6): 599-605.
3. Longardt AC, Loui A, Bührer C, Berns M. Milk curd obstruction in human milk-fed preterm infants. Neonatology. 2019; 115(3): 211-6.
4. Elkhouli M, Aleali F, Alzamrooni A, Chiu P, Gauda E. Ileal lactobezoar in extreme premature infant complicated by intestinal perforation: A case report. Int J Surg Case Rep. 2022; 96: 107303.
5. Richard J. Schanler. The use of human milk for premature infants. Pediatrn Clin North Am. 2001; 48(1):207-19.

6. Pavan M, Pillon R, Bua J, Codrich D, Murru FM, Travan L, et al. Milk curd problems in preterm infants are not just about obstructive symptoms. Acta Paediatr. 2021; 110(2): 487-8.
7. Berkowitz GP, Buntain WL, Cassady G. Milk curd obstruction mimicking necrotizing enterocolitis. Am J Dis. Child. 1980; 134: 989-90.
8. Watanabe T, Takahashi M, Amari S, Ohno M, Sato K, Tanaka H, et al. Olive oil enema in a pre-term infant with milk curd syndrome. Pediatr Int. 2013; 55(4): e93-5.
9. Flikweert ER, La Hei ER, De Rijke YB, Van de Ven K. Return of the milk curd syndrome. Pediatr Surg Int. 2003; 19(9-10): 628-31.
10. Konvolika CW, Frederick J. Milk curd syndrome in neonates. J Ped Surg. 1989; 24: 497-8.
11. Stanger J, Zwicker K, Albersheim S, Murphy JJ 3rd. Human milk fortifier: an occult cause of bowel obstruction in extremely premature neonates. J Pediatr Surg. 2014; 49(5): 724-6.