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CASE REPORT

Mucosal pre-pyloric diaphragm producing repeated vomiting in a newborn - Case report and Literature review

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Abstract

Objective: To report the case of a newborn with a pre-pyloric mucosal diaphragm with repercussions on weight gain and food acceptance. This is a rare congenital gastrointestinal malformation and corresponds to approximately 1% of all etiologies of gastrointestinal obstruction in newborns. **Description:** Female NB, started post-feeding vomiting at 4 hours of life. Multiple attempts of oral feeding reintroduction were performed, episodes of post-feeding vomiting and inadequate weight gain were maintained. Imaging exams did not show intestinal obstruction, an upper digestive endoscopy (UDE) was performed on the 19th day of life, which identified and corrected a pre-pyloric mucous diaphragm with transposition of the membrane by the device. Formula was reintroduced, with a gradual increase in supply and satisfactory weight gain. He was discharged at 24 days of life, with total acceptance of the diet and complete remission of the condition.

Comments: This study shows that it is important to consider the performance of UDE in cases of prolonged neonatal vomiting, after excluding more prevalent causes, such as APLV and GERD, as it is capable of diagnosing and treating some causes of obstruction of the gastrointestinal tract, such as the diaphragm pre-pyloric mucosa.

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INTRODUCTION

Congenital gastric obstructions are rare conditions that represent approximately 1% of congenital gastrointestinal obstructions and are limited to the antropyloric region. Among its anatomical variations, pyloric atresia is the most prevalent, with an incidence of 1/100,000 live births, classified into 3 types. Type I, pre-pyloric membrane or diaphragm, accounts for 57% of cases, characterized by a thin mucous septum at the location of the pylorus, with similar epidemiological distribution between the sexes. Types II and III, respectively, pyloric tissue replaced by solid cord and pyloric atresia with a gap between stomach and duodenum are less common and have a worse prognosis^{1,2,3}.

Congenital pyloric atresia can be clinically classified as isolated, which are most cases, associated with other genetic conditions or associated with other intestinal atresias. When associated with genetic conditions, the most common are epidermolysis bullosa and/or aplasia cutis congenita, which has a worse prognosis^{1,3}.

The clinical picture of partial obstruction by the pre-pyloric membrane is sometimes more subtle and insidious than complete obstructions and can present with polyhydramnios on prenatal ultrasound, abdominal distension and bilious vomiting after birth, in addition to important water and electrolyte changes.^{1,4} The newborn's food acceptance may vary according to the degree of obstruction caused by the membrane and, therefore, the clinical picture may mimic food intolerance, gastroesophageal reflux disease, hiatal hernia, intestinal motility disorders and other congenital intestinal obstructive pathologies^{4,5,6}.

The objective of this paper is to report the case of a newborn with congenital gastric obstruction of the pre-pyloric mucous diaphragm type, with repercussions on weight gain and food acceptance, highlighting the importance of considering this disorder within the differential diagnosis of repetitive gastrointestinal obstruction and vomiting in neonates. The information was obtained through a review of medical records and photographic records of diagnostic imaging methods, with a review of the literature regarding the subject of congenital gastric atresia.

CASE REPORT

Female newborn, second twin, born by cesarean section due to cormic presentation with a gestational age of 37 weeks and 2 days as of the first trimester ultrasound. The mother was followed up in high-risk prenatal care for chronic arterial hypertension using Methyldopa, without complications. She denied smoking, drinking, drug addiction or consanguinity with the father.

At birth, she weighed 2,360 g (p3-p10), was 44 cm long (<p3) and had a head circumference of 33 cm (p50-p90). She was born hypotonic and in apnea, with immediate clamping

of the umbilical cord and the need for two cycles of positive pressure ventilation (PPV), with an Apgar score of 4/7. She maintained early respiratory distress (ERD), being attached to CPAP, had an orogastric tube (OGT) inserted and was transferred to the neonatal ICU.

At 4 hours of age, using maintenance serotherapy, she showed partial improvement in ERD, still requiring CPAP, and we decided to start nutrition with a formula through the OGT. She had an episode of vomiting, in large quantities, before the first feeding. Gastric lavage was performed and an enteral diet was maintained.

Upon completing 24 hours of life, the newborn's breathing pattern improved. She developed two episodes of non-bilious vomiting after the diet, which was subsequently suspended. On examination, she presented a globular, painless abdomen, with decreased bowel sounds. A simple abdominal x-ray demonstrated only apparent dilation of some intestinal loops in the upper abdomen and absence of air in the rectal ampulla. Rectal examination was performed with a large amount of meconium coming out.

At 3 days of age, the newborn had lost 140 g (5.9%) in relation to her birth weight. She remained on enteral fasting with open OGT until she was 6 days old, when a new attempt was made to reintroduce the diet via OGT, due to the decrease in collected output; however, again, there was an episode of vomiting after breastfeeding. An intestinal transit test was ordered with a preparation that did not reveal obstructive factors or stenosing lesions (Figure 1).



Figure 1. Intestinal transit.

Given the result, we decided to start her on peripheral parenteral nutrition, maintaining her in oral fasting with an open OGT and observe the occurrence of new episodes of vomiting. At 9 days of age, Erythromycin was prescribed to stimulate intestinal transit and an attempt was made to reintroduce the fractionated oral diet with smaller intervals and volumes, maintaining daily vomiting, partial acceptance of the diets and weight between 2,400 and 2,500g.

At 18 days of life, already in the Intermediate Care Unit (ImCU), the newborn developed significant abdominal distension. We ordered a new simple abdominal x-ray which was similar to the one at 24 hours of life, showing gastric distension, edema of intestinal loops and absence of air in the rectal ampulla. The milk formula was changed to a hydrolyzed form, due to the hypothesis of allergy to cow's milk protein (CMPA), together with the introduction of oral domperidone as a measure for probable gastroesophageal reflux (GER).

Due to the persistence of post-feeding vomiting, impacting weight gain, despite previous measures, the Pediatric Surgery team discussed the performance of an upper digestive endoscopy (EDA) to better understand a possible obstructive factor that had not yet been diagnosed. At 19 days of age, the newborn was taken to the surgical center and underwent the procedure. During endoscopy, no changes were noted in the esophageal region or gastric body. However, when viewing the antral region, there was an incomplete pre-pyloric mucous diaphragm, easily transposed by the device (Figure 2), releasing the pre-pyloric region without complications.

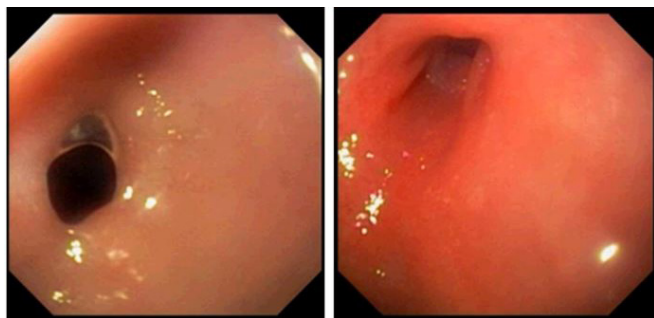


Figure 2. On the left, presence of a mucous diaphragm, in the pre-pyloric gastric region. On the right, view of the pre-pyloric region after transection of the membrane with an endoscopic device.

The newborn had a satisfactory response to endoscopic intervention, with good acceptance of the standard milk formula and increased oral diet supply and, at 24 days of life, she had shown complete improvement in vomiting episodes, maintaining weight gain within normal limits, with total acceptance of diets. She was discharged with a weight of 2,595 g for outpatient follow-up.

COMMENTS

The case presented corresponds to the endoscopic diagnosis and treatment of an incomplete congenital pre-pyloric diaphragm in a newborn with feeding difficulties, recurrent episodes of vomiting and unsatisfactory weight gain. Its etiology is not yet completely understood, but the main theory is the persistence of the intestinal solid center during embryology with incomplete canalization a posteriori^{7,8}. Accordingly, congenital hypertrophy of the pylorus presents a similar pathophysiology and origin, resulting in a condition that may be difficult to distinguish from the one presented and, therefore, it is essential to consider the hypothesis of a pre-pyloric diaphragm in neonates with a condition that differs from the classic presentation of congenital pyloric hypertrophy^{9,10,11}.

Based on initial imaging studies with air in the four abdominal quadrants and unremarkable intestinal transit, the hypothesis of complete obstructions of the gastrointestinal tract, such as duodenal atresia, was ruled out. Despite the introduction of antireflux measures such as elevating her head, diet fractioning and introduction of prokinetics, the NB did not show an improvement in symptoms or weight gain¹². With the persistence of the clinical picture, the hypothesis of CMPA was raised, a prevalent disease that can lead to vomiting after feedings, although the newborn did not present the classic clinical picture with diarrhea and traces of blood in the feces. Even with the use of hydrolyzed formula, the NB maintained her symptoms, which ruled out the hypothesis of CMPA and corroborated the need to seek less prevalent differential diagnoses^{12,13}.

During EDA, the pre-pyloric diaphragm was seen as a probable cause of the partial obstruction of food transit and episodes of vomiting after the diet. As supported by the literature, the therapeutic approach was transfixation of the membrane using the endoscopic device and, upon resuming the oral diet, the patient did not present new episodes of vomiting and had an acceptable weight gain for her age^{4,14,15}. Other treatment options depend on the type of atresia. There is the possibility of excision and Heineke-Mikulicz or Finney pyloroplasty. In type III, the treatment of choice is gastroduodenostomy, with a worse prognosis and greater chance of complications³. In this case, EDA was less invasive and more suitable for immediate diagnosis and treatment, without the need for other more morbid surgical procedures.

The prognosis of this case is favorable, given the impossibility of membrane recurrence, as well as no association with other malformations^{3,12}. If there is diagnostic and treatment delays, or an association with structural or genetic malformations, the prognosis is unfavorable, with inadequate

weight gain and delayed neuropsychomotor development^{3,4,14}. It is important to highlight that the determining factor of morbidity and mortality in cases of pyloric atresia is the association with genetic abnormalities, with sepsis related to epidermolysis bullosa being the main cause of death³.

Most incomplete gastric or pre-pyloric obstructions of the mucous diaphragm type are diagnosed in adulthood^{8,9}. In the long term, if the diagnosis is not made, the patient may experience deficits in weight gain, malnutrition and difficulty in neuropsychomotor development due to inadequate nutrition, in addition to cyclical vomiting and progressive abdominal discomfort, if the degree of obstruction worsens^{2, 6,9}.

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