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Case Report

10-Week-Old Infant with Lactobezoar

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Abstract

There are different causes for reflux, recurrent vomiting, constipation, poor weight gain, and other GI related issues in infants. Lactobezoar, a rare GI condition, can present with these nonspecific findings. Early diagnosis should be made, however, to prevent further complications and to ensure adequate growth and development in children.

ABBREVIATIONS

GI: Gastrointestinal; **NAC:** N-acetylcysteine

INTRODUCTION

Lactobezoar is a mass of undigested milk located within the gastrointestinal tract [1]. It is a rarely reported disorder (96 cases since its first description in 1959) [2]. Several risk factors can contribute to the formation of lactobezoar, including prematurity, low-birth weight and the use of high-caloric density formulas [1-3]. However, interestingly, this condition has also been reported in term, breast-fed patients (4). High casein concentration was thought to contribute to the formation of lactobezoar, and since formulas have changed to whey dominance, the reported incidence of this condition has decreased [1]. Medications used to reduce vomiting and diarrhea or to decrease gastric secretion and motility have also been reported to cause lactobezoar formation [2].

CASE PRESENTATION

A 10-week-old boy was admitted to the hospital from his pediatrician's office for a history of prolonged vomiting, constipation, and poor weight gain. The vomiting was described as non-bloody, non-bilious and started about 4 weeks after birth. Vomiting occurred about 6 times per week, usually post-prandial with associated discomfort. An ultrasound done when he was about 2 months of age showed borderline thickening and elongation of the pylorus muscle (between 15 and 17mm in length, and just under 3mm for muscle thickness). An upper GI series performed at that time showed possible reflux but no evidence of pyloric stenosis. He was started on ranitidine without relief. He had his first bowel movement within 48 hours of birth, and maintained regular bowel movements until 4 weeks of age. However, between 4 weeks of age until hospital admission, he had only 3 bowel movements. He still maintained good appetite and adequate urine output but did have trouble gaining weight possibly due to recurrent vomiting. He had gained only 5 ounces since birth. The patient was initially on Similac Advance but was switched to different formulas around one month of age due to frequent spitting up and finally stayed on Similac Sensitive, which appeared to help somewhat. Parents stated they have been mixing his formula as directed on the label.

On review of systems, parents denied him having fever, cough, runny nose, and congestion. He did not attend daycare. The patient was born preterm at 36-3/7 weeks via vaginal delivery without complications except for being hospitalized for 2 days for phototherapy due to neonatal jaundice. Family history was significant for pyloric stenosis in his older brother and history of reflux in his father. Physical exam was grossly normal except he appeared cachectic and malnourished.

Patient was initially started on pantoprazole for gastroesophageal reflux and metoclopramide for vomiting while pending further workup. Repeat abdominal ultrasound on admission showed single muscle wall thickness of 1.2mm and pyloric channel length of 4.7mm, which ruled out pyloric stenosis. Upper GI was performed as a different modality to assess for pyloric stenosis given the inconsistent results of the ultrasounds. The result showed no evidence of pyloric stenosis, but did show several episodes of reflux and "a large, nonobstructing, mobile filling defect compatible with residual food from recent feeding versus lactobezoar".

In our case, due to the prolonged history of vomiting, fussiness, constipation, and poor weight gain, we decided to perform an endoscopy to confirm the presence of the lactobezoar and look for eosinophilic disorders. Endoscopy was done the day after, which showed a large whitish non-obstructing particle occluding most of the pylorus (Figure 1) and the mucosa was normal otherwise. The non-obstructing particle seen during endoscopy was presumed to be a lactobezoar. We injected 20ml of 10% N-acetylcysteine mixed in 100ml fluid via endoscopy to flush the lactobezoar, then kept the solution running for another 2 minutes to completely dissolve it. The lactobezoar was successfully

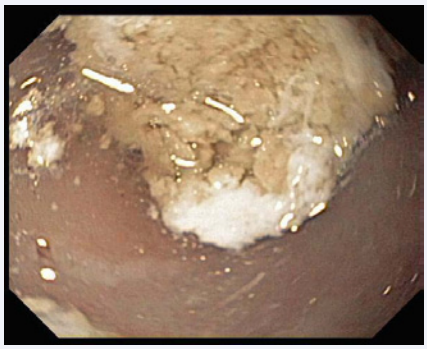

Figure 1 The lactobezoar occluded most of the pylorus.

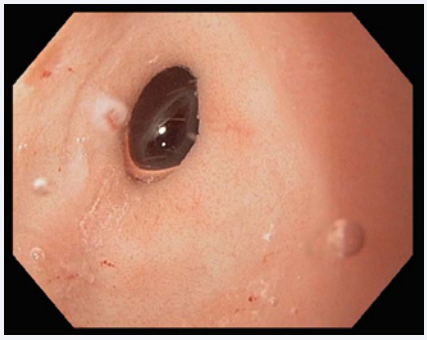

Figure 2 Normal appearance of antrum and pylorus after the lactobezoar was dissolved with high velocity fluid and 10% N-acetylcysteine.

dissolved with high velocity fluid and 10% N-acetylcysteine, with resultant clearance of the antrum and pylorus (Figure 2).

After endoscopy, he was restarted on his regular feeds and medications. Overnight, parents reported him eating well, having significantly less vomiting, and 3 normal bowel movements. He was discharged home with the same feeding regimen and medication the next day.

DISCUSSION

Presenting symptoms of this condition can include abdominal distention, vomiting, diarrhea, gastric residuals, palpable mass, and dehydration (1). Due to these nonspecific findings, diagnosis might not be clear in a newly presenting patient. In our patient, his vomiting could have been attributed to normal reflux seen in infants that usually resolves around 6 months of age. His constipation and poor weight gain could have been explained by the fact that he had excessive vomiting contributing to insufficient caloric intake. However, his poor weight gain (only 5 ounces since birth) and cachectic appearance did warrant further workup.

Diagnosis of lactobezoar can be achieved most commonly by the use of upper GI series [1]. There are also studies that report the successful use of other modalities to diagnose this condition, such as ultrasound and roentgenography [3,5].

Management of lactobezoar centers around conservative therapy and expectant management. The mainstay therapy is to withhold oral feedings and administer parenteral fluids for several days, which usually leads to resolution [1]. In severe cases with perforation or obstruction, surgery might be indicated. Several previous studies also proposed the use of 10% N-acetylcysteine (NAC), a mucolytic agent, to help break down the lactobezoar [6,7]. NAC works by breaking the disulfide bonds within the mucus, thereby decreasing its viscosity [6]. 10% N-acetylcysteine, as proposed by several previous studies, was successfully used in our case to break up the lactobezoar [6,7].

Patients with lactobezoar can present with nonspecific findings, such as abdominal distention, vomiting, diarrhea, and palpable abdominal mass. Early detection and treatment of this condition is critical as delays in treatment can cause the patient to deteriorate quickly into a condition mimicking necrotizing enterocolitis [8]. Another study also suggested that gastric lactobezoar should be considered as a cause for anemia in children, due to induced bleeding from the gastric mucosa from mechanical irritation and also due to decreased duodenal iron absorption secondary to increased gastric pH caused by emesis [9]. In conclusion, although a rare condition, one should keep it in mind during a workup in a patient presenting with these nonspecific gastrointestinal symptoms.

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