

Unexpected finding of a gastric lactobezoar in a 71-year-old

Lukas Birkner, Tim Dudziak

ABSTRACT

Gastric lactobezoar is a rare disorder clinically presenting as indigestible conglomerations of milk and mucus components located in the gastrointestinal tract. Non-specific symptoms pose a challenge in diagnosing the gastric lactobezoar correctly. The majority of gastric lactobezoars have been diagnosed in premature neonates. We report a case of 71-year-old male referred to our department with an acute cholecystitis. A postoperative respiratory breakdown required mechanical ventilation support and high caloric nutrition which possibly evoked the formation of a gastric lactobezoar. We report the first case of a gastric lactobezoar in an adult and suggest that the regulation of gastric acid may be a possible strategy of the treatment.

Keywords: Fresubin HP energy, Gastric lactobezoar, Proton pump inhibitor

How to cite this article

Birkner L, Dudziak T. Unexpected finding of a gastric lactobezoar in a 71-year-old. *Edorium J Gastroenterol* 2017;4:1–4.

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Received: 27 November 2016

Accepted: 23 January 2017

Published: 10 February 2017

Article ID: 100007G01LB2017

doi:10.5348/G01-2017-7-CR-1

INTRODUCTION

Bezoars are indigestible conglomerations located in the gastrointestinal tract. Although a bezoar can be found in any part of gastrointestinal tract, they are most commonly located in the stomach. Depending on the material constituting the indigestible mass, bezoars are classified into four types. These are phytobezoars (i.e., plant material), trichobezoars (i.e., ingested hair), pharmacobezoars (i.e. medication) and lactobezoars (i.e., milk products) [1–3]. The most common type is the gastric lactobezoar. It is a pathological conglomeration of milk and mucus components [4]. The clinical presentation includes abdominal distension, vomiting, diarrhea, a palpable abdominal mass, respiratory and cardiocirculatory symptoms. Abdominal ultrasound, showing echogenic intrabezoaric air trapping, is often used for diagnosis. However, it is a rarely reported disease thus challenging to diagnose correctly for inexperienced investigators [5]. Gastric lactobezoar is characterized by its occurrence during early age. The majority of lactobezoars have been diagnosed in premature neonates [6].

CASE REPORT

We report the case of a 71-year-old male patient referred to us with an acute cholecystitis, accompanied by somnolence and a beginning disorder of consciousness. He did not display typical symptoms such as pain in right upper abdomen, fever, jaundice or nausea. Preexisting conditions included diabetes mellitus, hypercholesterolaemia, a chronic obstructive pulmonary disease (COPD) worsened by extensive smoking (40 pack

years) and an unknown gastrointestinal disease which was permanently treated with pantoprazol. Although the risks were explicitly discussed with the patient, surgery commenced. A laparoscopic cholecystectomy was performed. Due to an acute cholecystitis in addition to the preexisting COPD the patient's respiratory condition worsened considerably after the surgery. Mechanical ventilation support and endotracheal intubation were started. Additionally a nasogastric feeding tube was installed. The surgery and concurrent medication, namely sufentanil, affected the patient's intestinal activity negatively. He suffered from constipation due to reduced intestinal motility, a typical adverse reaction of the administered medication. Thus high-caloric nutrition was administered. The patient received enteral Fresubin HP Energy (1,5 kcal/ml) from Fresenius Kabi. Thereafter, the nasogastric feeding tube became clogged and was pulled out. A replacement could not be performed due to a cheesy substance obstructing the implementation. Contrast examination showed an unclear stomach obstruction (Figures 1 and 2) and the subsequent gastroscopy (Figure 3) revealed a pasty substance located in the generalized stomach later identified as gastric lacto bezoar. During the course of the early treatment gastroscopy was performed daily, firstly to remove the cheesy substance which was not successful and secondly to extract samples. A serial dilution (Figure 4), which was observed semiquantitatively, showed that the gastric lacto bezoar was dissolved only by commercial citric acid.



Figure 2: Abdomen X-ray showing evidence of a gastric obstruction after administration of 100 ml gastrografin 2.5 h ago.

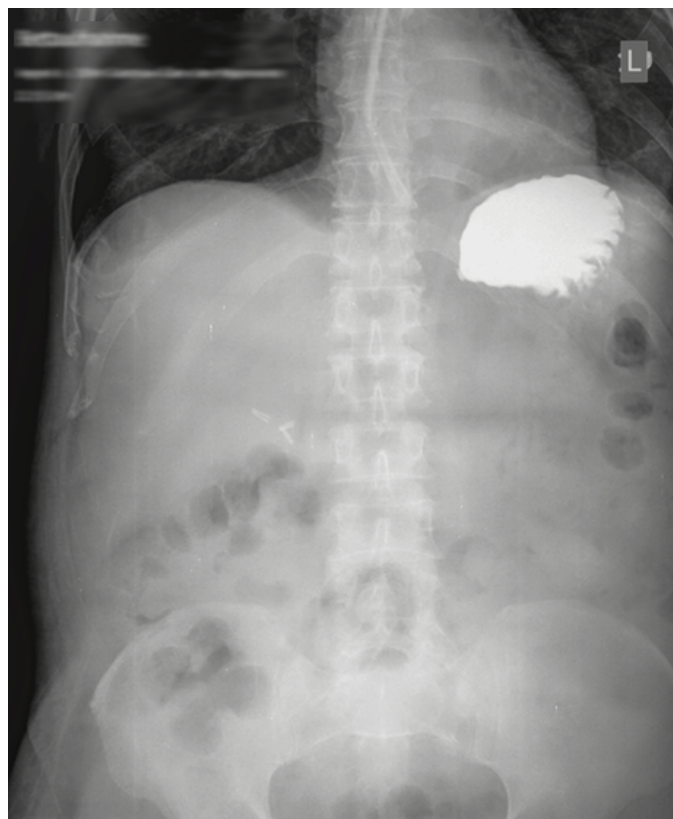


Figure 1: Abdomen X-ray shortly after the administration of 100 ml gastrografin.

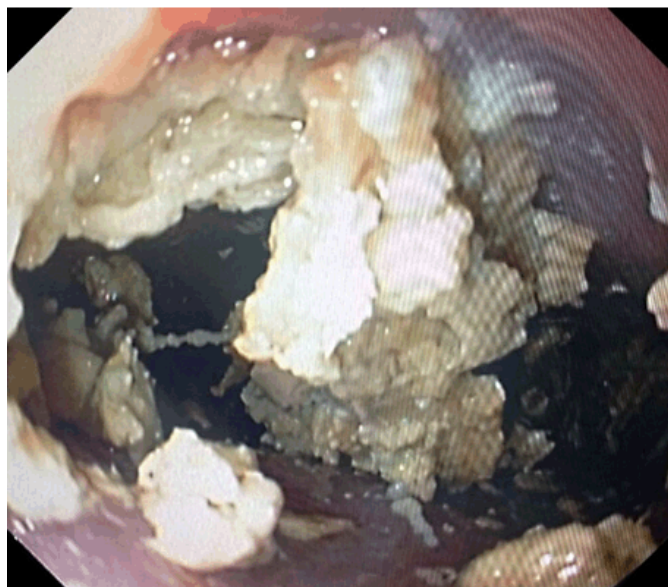


Figure 3: Endoscopic picture showing the cheesy substance on the gastric lacto bezoar in the upper gastrointestinal tract.

DISCUSSION

First described by Wolf and Bruce in 1959, a gastric lacto bezoar is a mass constituted of milk and mucus components. It is a rare condition with less than 100 cases



Figure 4: In vitro serial dilution concerning the semiquantitative analysis of dissolution strategies for gastric lactobezoars showing citric acid and parts of the gastric lactobezoar in question after 48 h at room temperature.

reported since 1975 [7, 8]. Gastric lactobezoars differ from other bezoars most notably by the age of presentation. The majority of gastric lactobezoars have been reported in infants and small children. Most cases deal with age-inadequate malnutrition often associated with cow's milk [9]. Dehydration and formulas high in medium chain triglycerides, casein and caloric density are common contributing factors towards the pathogenesis of gastric lactobezoars [10].

The patient displayed some of the above risk factors including consuming a high caloric density formula. Clinicians need to be aware that these factors, especially the consumption of Fresubin HP Energy, may result in the development of a gastric lactobezoar.

Gastric lactobezoars can be associated with a variety of clinical symptoms including vomiting diarrhea, bloody or tarry stool and abdominal fullness. Additionally, respiratory and cardiovascular symptoms are sometimes observed [11,12]. Due to the timing of events our patient's respiratory condition cannot be linked to the development of the gastric lactobezoar nor can any symptoms be documented since the patient was not responsive. A result of the nonspecific symptoms associated with gastric lactobezoar is that the condition is often not taken into consideration. Also considering the rarity of the disease misinterpretation of the symptoms is common and may pose risk factors for the patient.

There are various treatment strategies suggested in the literature including conservative treatment, treatment with N-acetylcysteine and surgical and endoscopic removal [7, 11]. Conservative treatment, a trial of bowel rests and intravenous fluids, is the preferred method of treatment. This conservative regimen was reported to be successful in over 85% of treated patients [12] because this method may take up to six weeks other treatment strategies should be considered for patients with high risk factors. Surgical or endoscopic removal of the gastric lactobezoar has also been reported. However, endoscopic removal via basket catheter proved unsuccessful in our case.

In want of dissolution strategies, we performed a serial dilution, which was observed semiquantitatively and subjectively in vitro, with samples extracted

gastroscopically. Among the tested substances only citric acid purchased at the local supermarket proved useful due to a nearly complete dissolution of the sample after 72 h. The following substances, pancreatin, N-acetylcystein, wine, orange juice and Coca-Cola, did not achieve the dissolution of the gastric lactobezoar. Transferring these findings we discontinued pantoprazol, a proton pump inhibitor, hoping that the production of gastric acid might dissolve the gastric lactobezoar just as the citric acid had.

What this all amounts to is that in our case the reason for the gastric lactobezoar was primarily the consumption of a high caloric density formula in addition to restricted functioning of the digestive tract. This case shows that gastric lactobezoars can originate from the dysfunctional production of gastric acid. Moreover it must be noted that the regulation of gastric acid could be a possible method for the treatment of gastric lactobezoar.

CONCLUSION

Clinicians need to be aware that the misinterpretation of the non-specific symptoms accompanying gastric lactobezoars can result in high risk factors for the patient. Additionally, the regulation of the production of gastric acid and the adverse effect medication poses should be considered when deciding on a treatment strategy.

Author Contributions

Lukas Birkner – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Tim Dudziak – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

The authors declare no conflicts of interest in this work. We would like to thank Abass Dehzad, Ralf v. d. Boom and Mario Iasevoli for their excellent active support during the endoscopic surgery and their commitment concerning this case.

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