CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 36-2024: A 16-Year-Old Girl with Abdominal Pain

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PRESENTATION OF CASE

Dr. Mallory C. Mandel (Pediatrics): A 16-year-old girl was admitted to this hospital because of abdominal pain.

The patient had been in her usual state of health until 4 weeks before the current presentation, when intermittent abdominal pain developed. During the next 2 weeks, the episodes of pain became more frequent, increasing from every few days to two times daily and lasting approximately 2 minutes. Two weeks before the current presentation, the patient had new nausea, and she vomited three times. She was not able to go to school, and her parents took her to the emergency department of another hospital for evaluation.

In the emergency department, the patient described the pain as sharp and rated it at 8 on a scale of 0 to 10 (with 10 indicating the most severe pain). The pain was worst in the epigastrium and in the upper quadrants of the abdomen. The temporal temperature was 36.6°C, the heart rate 105 beats per minute, the blood pressure 125/66 mm Hg, the respiratory rate 18 breaths per minute, and the oxygen saturation 99% while the patient was breathing ambient air. The mucous membranes were dry. She had diffuse abdominal tenderness with guarding on palpation of the epigastrium, left upper quadrant, and right upper quadrant. The white-cell count was 14,900 per microliter (reference range, 4500 to 13,000) and the hemoglobin level 11.3 g per deciliter (reference range, 12.0 to 16.0). Other laboratory test results are shown in Table 1. Qualitative urine testing for human chorionic gonadotropin was negative, and urinalysis was normal. Ultrasonography of the abdomen and pelvis showed no abnormalities, although the appendix was not visualized. Oral acetaminophen was administered, as were intravenous ketorolac, famotidine, and ondansetron. The patient was discharged home with instructions to start treatment with oral famotidine, ondansetron, and ibuprofen.

During the next week, the patient continued to have intermittent episodes of abdominal pain and nausea, and she left early from school on two days because of pain. She took ibuprofen on one occasion, but the pain subsequently worsened;

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Table 1. Laboratory Data.

Variable	Reference Range, Adults, Other Hospital	2 Wk before Current Presentation, Other Hospital	Reference Range, Adults, This Hospital*	On Presentation, This Hospital
Hemoglobin (g/dl)	11.3–15.0	11.3	12.0–16.0	10.8
Hematocrit (%)	33.9–45.0	36.1	36.0–46.0	34.9
White-cell count (per μl)	4000–10,800	14,900	4500–13,000	15,720
Differential count (per μl)				
Neutrophils	1900–8200	12,300	1800–8100	12,940
Lymphocytes	300–4200	1400	1200–5200	1780
Monocytes	200–800	700	200–1400	500
Eosinophils	100–300	500	0–1000	350
Basophils	0–200	100	0–400	80
Platelet count (per μl)	189,000–400,000	577,000	150,000–450,000	620,000
Mean corpuscular volume (fl)	82.1–87.7	71.1	78.0–98.0	69.5
Erythrocyte sedimentation rate (mm/hr)	—	—	0–19	32
C-reactive protein (mg/liter)	—	—	0.0–8.0	4.6

* Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

she decided not to take any additional doses. She did not take any doses of famotidine or ondansetron.

One week before the current presentation, abdominal pain woke the patient from sleep, and her parents took her to the emergency department at the other hospital. The mucous membranes were dry, and no abdominal distention or tenderness was noted. The white-cell count was 13,000 per microliter and the hemoglobin level 11.4 g per deciliter. Repeat ultrasonography of the abdomen and repeat transabdominal ultrasonography of the pelvis again showed no abnormalities. Screening tests for gonorrhea and chlamydia were negative. The patient was discharged home with instructions to start treatment with famotidine, ondansetron, and acetaminophen.

During the subsequent week, episodes of abdominal pain and nausea continued to occur. On the day of the current presentation, the episodes of pain lasted 15 minutes, and the patient vomited five times. Her parents brought her to the emergency department of this hospital for evaluation.

The patient described the episodes of abdominal pain as severe, occurring abruptly, and followed by nausea; when vomiting occurred, the severity of pain decreased. The pain was not related to menstruation, body position, time of day, or eating or drinking, although she noted that she had had decreased appetite. She had had fatigue, dizziness, and generalized weakness, as well as constipation that had worsened during the previous 2 weeks; the last bowel movement had occurred 3 days before the current presentation. There was no fever, diarrhea, or rash.

The patient's medical history included iron-deficiency anemia and anxiety disorder; she had had pica as a toddler. She had normal growth and development and had received all childhood vaccinations. Medications included iron supplementation, oral contraception, and famotidine, as well as acetaminophen as needed for abdominal pain; she had not started taking ondansetron. She had no known drug allergies. The patient lived with her mother, father, sister, and dog in an urban area of New England. She attended high school. She was sexually active and had had two male partners; she used condoms consistently. She did not drink alcohol, use illicit drugs, or smoke tobacco; she had last smoked marijuana 4 weeks before the current presentation. Her maternal grandmother had peptic ulcer disease, her maternal grandfather had heart disease, and her father had depression and anxiety.

On examination, the temporal temperature was 36.7°C, the heart rate 84 beats per minute, and the blood pressure 113/78 mm Hg. The body-mass index (the weight in kilograms divided by the square of the height in meters) was 25.8. The patient was actively retching during the examination. The mucous membranes were moist, and bowel sounds were present. The abdomen was nondistended, soft, and diffusely tender (predominantly in the epigastrium), and there was guarding without rebound. The rectal examination was normal, with no palpable stool in the rectal vault. The white-cell count was 15,720 per microliter and the hemoglobin level 10.8 g per deciliter. Blood levels of electrolytes, glucose, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and bilirubin were normal, as were the results of kidney-function tests. The urine level of human chorionic gonadotropin was less than 6 IU per milliliter (reference range in nonpregnant adults, 0 to 6), and urinalysis was normal. Other laboratory test results are shown in Table 1.

Dr. Ali Pourvaziri: Computed tomography (CT) of the abdomen, performed after the administration of intravenous contrast material and without the administration of oral contrast material, showed the gastric contents, a normal appendix, and no evidence of bowel obstruction. There was a small amount of solid stool in the ascending colon and sigmoid colon.

A mixture of aluminum hydroxide, diphenhydramine, lidocaine, magnesium hydroxide, and magnesium citrate was administered, but the patient vomited after taking these medications.

A diagnostic test was performed.

DIFFERENTIAL DIAGNOSIS

Dr. Garrett C. Zella: This 16-year-old girl presented after 4 weeks of worsening intermittent sudden-onset abdominal pain and 2 weeks of nausea and vomiting. Her heart rate was elevated. She had abdominal tenderness with guarding, and she vomited after taking oral medications used to treat common causes of abdominal pain. Abdominal pain, nausea, and vomiting are all nonspecific symptoms in a teenager; the list of possible causes is long. In working toward the most likely diagnosis, I will begin by considering common conditions and then move to the rarer possibilities.

CONSTIPATION

Constipation is an extremely common cause of abdominal pain and can occur with nausea and even intermittent vomiting. This patient had constipation, and the intermittent nature of her symptoms could be explained by peristalsis and high-amplitude propagating colonic contractions. However, it would be unusual for constipation to lead to severe vomiting unless the patient was severely affected. This patient's abdomen was nondistended with no palpable abdominal mass, and the rectal examination showed no stool in the rectal vault. Constipation alone is unlikely to explain her presentation.

DISORDER OF GUT–BRAIN INTERACTION

Disorders of gut–brain interaction (previously known as functional gastrointestinal disorders), such as functional dyspepsia and functional abdominal pain, often occur in combination with mental health conditions and are an important consideration in this patient with anxiety and a family history of anxiety and depression. If the patient had sleeping difficulties in the context of anxiety, she may have awoken and then had abdominal symptoms. However, symptoms associated with a disorder of gut–brain interaction would be unlikely to awaken a patient from sleep. In addition, symptoms related to such a disorder are not acute and do not worsen over a period of weeks. Finally, this patient's abdominal tenderness with guarding would be atypical for a disorder of gut–brain interaction.

GASTRITIS

Gastritis can lead to nausea and then vomiting because of slowed gastric emptying. This patient had no fever or diarrhea — characteristics that would have suggested gastritis caused by a viral infection. In addition, the symptoms of viral gastritis are usually more severe initially, with slow clinical improvement as healing occurs. Other common causes of gastritis include nonsteroidal antiinflammatory drugs (NSAIDs) and alcohol use; this patient took NSAIDs only briefly, and she had no history of alcohol use. Iron supplements can very rarely cause gastritis.[1]

POSTVIRAL GASTROPARESIS

Postviral gastroparesis is a common condition. Typically, a patient may have loss of appetite for a few days after a viral infection. However, severe

cases often include abdominal pain, nausea, and vomiting and may takes years to resolve.[2] This patient had no preceding viral syndrome. In addition, instead of the slow clinical improvement that typically occurs with postviral gastroparesis, this patient's condition continued to worsen week by week.

OTHER COMMON CONDITIONS

Lactose intolerance is an unlikely diagnosis in this case, given the absence of diarrhea and bloating. Gallbladder disease is also unlikely in this patient, since she had diffuse tenderness beyond the right upper quadrant and had normal findings on ultrasonography of the gallbladder. Pancreatitis cannot be ruled out in this patient, since the pain was primarily in the epigastrium and a blood level of lipase was not provided. However, the pancreas appeared normal on abdominal ultrasonography and CT. Celiac disease is a possible diagnosis in any patient with abdominal pain and vomiting, but the pace of this patient's clinical worsening would not be consistent with this diagnosis.

MECHANICAL CAUSES OF ABDOMINAL PAIN

A key feature of this patient's presentation was the sudden nature of her pain; the episodes occurred abruptly and were followed by vomiting that resulted in relief of the pain. Her symptoms worsened dramatically over a short period of time. There was focal tenderness and guarding on examination. Taken together, these aspects of her presentation point toward a mechanical cause of pain.

Malrotation

Intestinal malrotation may manifest at any age, but 21% of cases are diagnosed between 1 and 18 years of age.[3] Malrotation may result in the development of abnormal peritoneal tissue known as Ladd's bands, which can constrain the malpositioned cecum and compress the duodenum and can in turn lead to bouts of pain, nausea, and vomiting. Malrotation can also result in midgut volvulus, a condition in which the venous drainage is impaired and leads to edema; eventually, the arterial flow becomes compromised and results in ischemia. It is possible that subtle radiologic signs of malrotation were missed on this patient's abdominal CT (which was performed without the administration of oral contrast material). However, there was no evidence that the cecum was high-riding or that the small bowel was shifted to the right, and the results of two abdominal ultrasonographic studies were normal. Therefore, malrotation with intermittent volvulus is possible but unlikely.

Intussusception

Intussusception of the intestine can also manifest at any age. The invaginated bowel may cause venous congestion, followed by bowel-wall edema, which eventually impairs the arterial flow and results in ischemia. Intermittent ischemia could explain this patient's episodes of pain, nausea, and vomiting, as well as the abdominal tenderness and guarding. Although it is possible that evidence of intussusception was missed on this patient's abdominal CT, the two normal abdominal ultrasonographic studies make intussusception unlikely in this case.

Crohn's Disease

Crohn's disease involving the ileum can cause partial obstruction from inflammation, and this patient did have an elevated erythrocyte sedimentation rate and mild microcytic anemia. Although her CT images were obtained without the administration of oral contrast material, which could have helped to delineate bowel-wall edema, there were no obvious bowel-wall abnormalities. More importantly, the time course of her presentation does not fit with a diagnosis of Crohn's disease leading to partial obstruction because I would expect a longer history of worsening symptoms associated with Crohn's disease.

Other Causes of Small-Bowel Obstruction

Multiple causes of small-bowel obstruction could explain the progressively worsening symptoms in this patient. Although adhesions can worsen over time, this patient had no history of abdominal surgery. An obstructing small-bowel lymphoma may grow larger, but this patient had no weight loss, fever, or night sweats. A polyposis syndrome with edematous and inflamed polyps may worsen obstruction of the small bowel over time. However, this patient had no mucocutaneous hyperpigmentation that would be suggestive of a syndrome such as the Peutz–Jeghers syndrome. A bezoar lodged in the small

bowel can enlarge over time and cause intermittent obstruction. This patient could have had a small-bowel phytobezoar resulting from dietary fiber or a pharmacobezoar resulting from oral iron supplementation.

Gastric-Outlet Obstruction

Gastric-outlet obstruction could explain not only the patient's worsening pain, nausea, vomiting, and leukocytosis but also the resolution of severe symptoms immediately after vomiting. Gastric-outlet obstruction can occur with a trichobezoar, and it is notable that this patient had a history of pica as a toddler. There is no mention of alopecia on her scalp — a finding that would suggest trichotillomania — but this condition is often a surprise to the clinician, and the loss of hair may not be noticed on initial examination. A pharmacobezoar resulting from oral iron supplementation can rarely cause gastric-outlet obstruction.

This patient's progressive episodes of abrupt abdominal pain, nausea, and vomiting — with relief of abdominal pain after vomiting — are most likely due to gastric-outlet obstruction caused by a gastric bezoar. Any type of bezoar in the stomach could be interpreted as food on CT of the abdomen when performed without the administration of oral contrast material. To establish the diagnosis of gastric-outlet obstruction caused by a bezoar, I would perform esophagogastroduodenoscopy to detect the foreign body that was probably causing gastric-outlet obstruction.

DR. GARRETT C. ZELLA'S DIAGNOSIS

Gastric bezoar.

DISCUSSION OF CLINICAL MANAGEMENT

Dr. Maureen M. Leonard: Esophagogastroduodenoscopy was performed. The esophagus appeared normal (Fig. 1A). In the stomach, a large trichobezoar that extended into the duodenum was identified (Fig. 1B and 1C).

A bezoar is a foreign body resulting from the accumulation of ingested material and is most commonly found as a hard mass in the stomach. Bezoars are classified according to their composition and include phytobezoars (composed of fruits and vegetables), trichobezoars (composed of hair), and pharmacobezoars (composed of extended-release capsules, enteric-coated aspirin, or iron[4]). In rare cases, bezoars are composed of ingested styrofoam, cement, or other materials.[5]

Trichobezoars are the most common bezoars in humans and are found in the stomach. Hair is resistant to digestion and peristalsis and thus becomes lodged in the gastric folds, where it becomes denatured by gastric acid and combines with food and other materials to form a mass. Once formed, the trichobezoar continues to collect food and indigestible materials, growing and becoming matted. Affected patients are initially asymptomatic, but as the bezoar grows larger, abdominal pain, nausea, vomiting, early satiety, anorexia, and weight loss often develop.[6] Halitosis occurs if the bezoar becomes colonized by bacteria. In rare instances, as in this patient's case, a trichobezoar extends into the small bowel — a condition called the Rapunzel syndrome. Complications of trichobezoars include obstruction, gastrointestinal bleeding due to increased pressure, perforation of the stomach or intestine, intussusception, acute pancreatitis, and cholangitis.[6,7] The risk of complications is increased in patients with a trichobezoar that extends into the small bowel.

Dr. Pourvaziri: From an imaging perspective, the presence of a bezoar is considered when mottled lucencies within solid matter are identified on plain radiography. However, because both the splenic flexure and the stomach project over the left upper quadrant, differentiating a bezoar from fecal material on radiographs can be challenging without a high clinical suspicion. On ultrasonography, a bezoar may be identified by the presence of an intraluminal mass with internal hyperechoic foci, sometimes in an arch-like pattern. CT is the most reliable method for detecting bezoars, which appear as a low-density material with internal air bubbles, sometimes with a characteristic mottled appearance.[8,9] On further review of this patient's CT images, the material in the gastric lumen was found to have a mottled appearance that was consistent with that of a bezoar (Fig. 2A and 2B).

Dr. Leonard: In patients with a bezoar, enzymatic digestion, endoscopy, and surgical laparoscopy or laparotomy are all options to consider for

removal of the bezoar. In patients with a trichobezoar in particular, enzymatic digestion is not effective and the trichobezoar must be removed endoscopically or surgically.[10] Endoscopy is less invasive and more cost-effective than surgery, but it is often not feasible owing to the shape and size of the trichobezoar.[7,11] In symptomatic patients with a large trichobezoar, the trichobezoar must be removed surgically, especially if it extends into the small bowel.

Laparoscopic surgery for removal of a trichobezoar is often unsuccessful because of the long operative time that is needed to remove the bezoar in pieces and because this procedure confers an increased risk of infection.[12] Laparotomy with gastrostomy is the preferred technique for removal. A literature review in 2010 showed that 93% of the patients who had been identified as having a trichobezoar underwent laparotomy; 99% of those procedures were considered to be successful, and 12% led to complications.[11]

In this patient, given the size of the trichobezoar and the fact that it extended into the duodenum, we consulted our surgical colleagues to discuss removal of the trichobezoar. An exploratory laparotomy and gastrostomy was performed. During the procedure, a portion of the trichobezoar was confirmed to extend through the pylorus of the stomach and into the duodenal bulb. The hair in the duodenal bulb was extracted

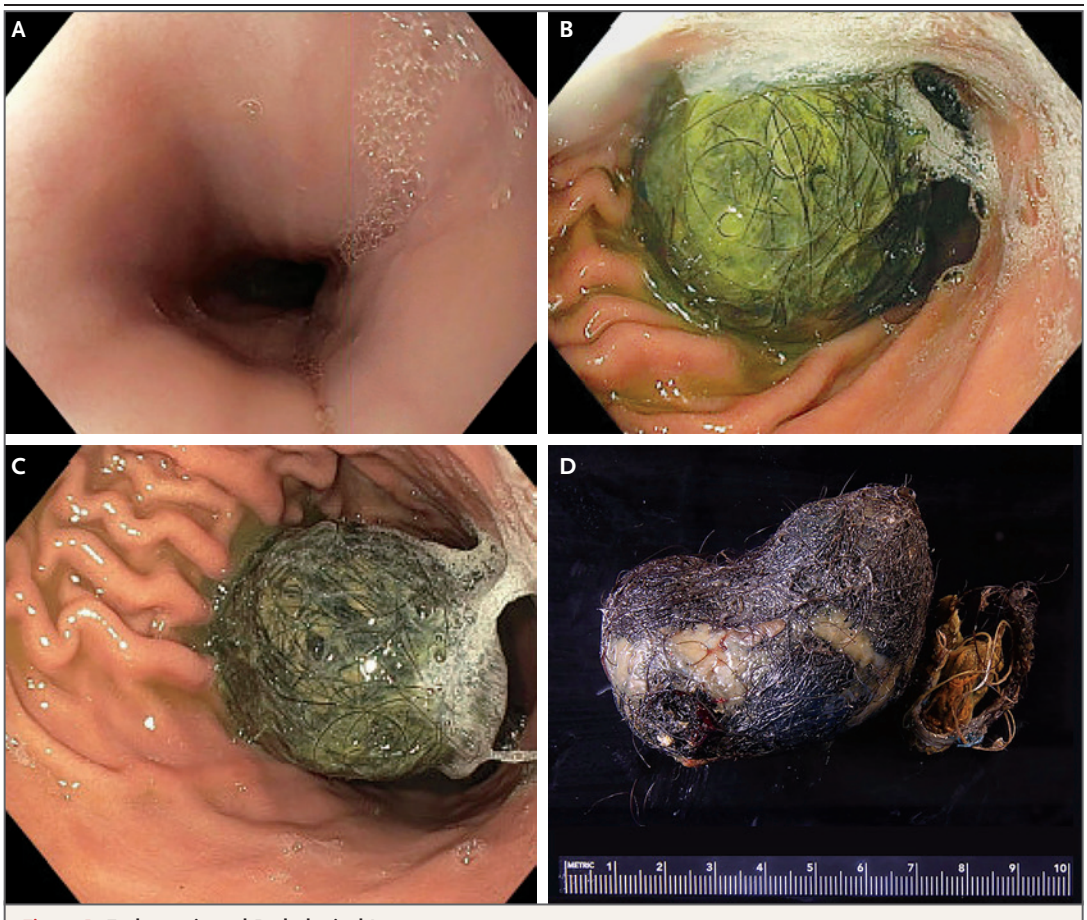

Figure 1. Endoscopic and Pathological Images.

An endoscopic image of the esophagus (Panel A) shows normal esophageal mucosa. Endoscopic images of the stomach (Panels B and C) show normal mucosa and a trichobezoar with visible hair located in the gastric body. The surgically removed gastric and duodenal trichobezoar is shown (Panel D). The tail of the trichobezoar, which extended into the duodenum, can be seen to the right of the trichobezoar.

with gentle pressure, and the trichobezoar was removed through the gastrostomy tube (Fig. 1D).

Once a bezoar is removed, the most important aspect of clinical management is to prevent the recurrence of the bezoar by addressing coexisting psychiatric disorders.

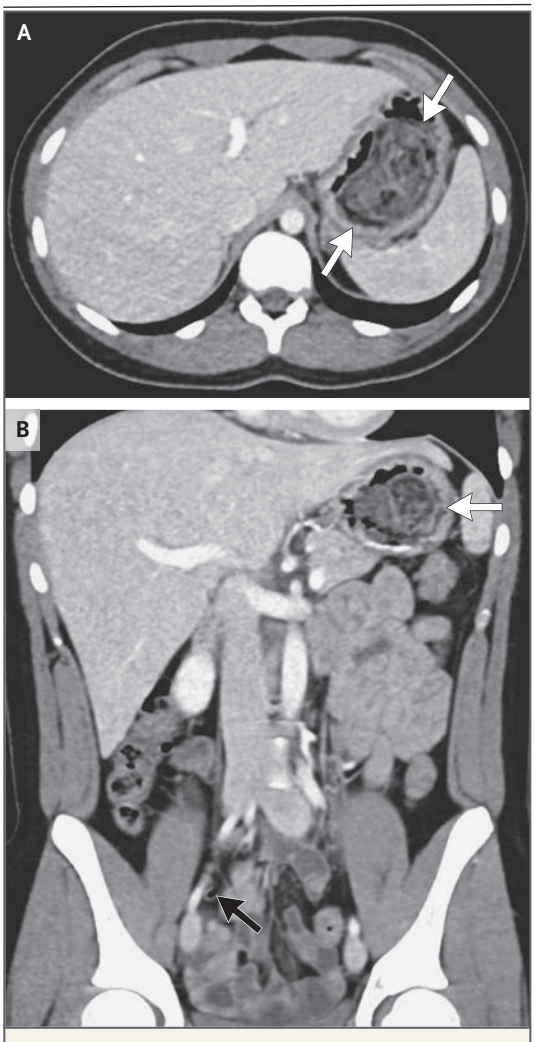

Figure 2. CT of the Abdomen and Pelvis.

CT of the abdomen and pelvis was performed after the administration of intravenous contrast material. An axial image (Panel A) shows no abnormalities in the liver and spleen; heterogeneous and predominantly hypodense mottled materials are present within the gastric lumen (arrows). A coronal reconstruction image (Panel B) shows no abnormalities in the appendix (black arrow) and no evidence of bowel obstruction; the heterogeneous intraluminal gastric contents are again visible (white arrow).

DISCUSSION OF PSYCHIATRIC MANAGEMENT

Dr. Erica L. Greenberg: Trichophagia is most often associated with trichotillomania, a body-focused repetitive-behavior disorder that is described in the Obsessive–Compulsive and Related Disorders chapter of the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5). Trichotillomania has a worldwide prevalence of 1 to 2% and is characterized by recurrent pulling out of the hair with resultant hair loss, repeated attempts to stop the behavior, and clinically significant associated distress or impairment in functioning.[13]

When interviewed, this patient did not report that she had trichotillomania. Case studies have shown that, although rare, it is possible for a trichobezoar to develop without trichotillomania through ingestion of others' hair or of discarded hair strands.[14-16]

Trichotillomania is most commonly seen in women and girls (typically beginning in early adolescence). Given this patient's demographic profile and the strong correlation between trichophagia and trichotillomania, it is important to maintain a high index of suspicion for trichotillomania. This patient also had a history of anxiety disorder. Anxiety and trichotillomania are distinct disorders, although anxiety commonly occurs in conjunction with trichotillomania. Obsessive–compulsive disorder, attention deficit–hyperactivity disorder, and depression are also common among patients with trichotillomania, although none of these conditions were present in this patient.[17]

Among patients with trichotillomania, 50 to 70% have oral ritualistic behaviors associated with hair pulling (e.g., rubbing hair across the lips or biting the roots of hair). The prevalence of trichophagia among patients with trichotillomania ranges from 5 to 20%, and among those with trichophagia, the reported prevalence of trichobezoars is approximately 1%, although this may be an underestimate.[15,18,19] Given the potential serious medical consequences of trichobezoars, it is crucial to ask any patient with trichotillomania about hair ingestion.[19]

Unfortunately, trichotillomania is associated with substantial shame and embarrassment,[20] and this commonly impedes a patient's willingness

to disclose such behaviors. When trichophagia is involved, patients with trichotillomania are even less likely to disclose this information.[19] Thus, the first sign of a patient's trichotillomania is likely to be the presence of a trichobezoar. Assessing for trichotillomania is also challenging because affected patients will go to great lengths to hide their hair pulling and signs of hair loss. Signs of trichotillomania on examination may include patches of hair loss, hairs of various lengths and growth stages, and redness or inflammation on the scalp resulting from frequent hair pulling or manipulation.

Hair pulling in patients with trichotillomania is believed to provide a mechanism for self-regulation, including regulation of mood or affect, sensory experience, environmental stressors, or negative thought patterns.[21] Patients with trichotillomania often report feeling a sense of relief from tension when pulling their hair, along with a positive satisfaction-type feeling.

Trichotillomania tends to be a chronic condition with symptoms that wax and wane over time. To date, no medications have been approved by the Food and Drug Administration for the treatment of trichotillomania; however, meta-analyses have shown a modest benefit with N-acetylcysteine — a natural supplement with antioxidant properties. Although selective serotonin reuptake inhibitors have not shown benefit over placebo among patients with trichotillomania, such medications may be indicated if a patient has clinically significant concurrent anxiety that may be exacerbating the hair pulling.[22] The standard behavioral therapy for trichotillomania is habit-reversal therapy with stimulus control. If, on further interviewing, this patient ultimately reported that she had trichotillomania, she would be referred for this behavioral therapy.

Pica is also on the differential diagnosis for trichophagia and resultant trichobezoars. Pica, which is described in the Feeding and Eating Disorders chapter of the DSM-5, is characterized by the persistent eating of nonfood, nonnutritive substances that is not consistent with cultural practice or social norms.[13] Pica can occur in youths in whom there is no evidence of developmental delays, although it is more commonly observed in those with intellectual impairment or neurodevelopmental disorders, autism spectrum disorder, and nutritional deficiencies (e.g., low iron), as well as in those coping with severe psychosocial stressors.[23] This patient was noted to have mild anemia, and iron-deficiency anemia can lead to pica. Alternatively, trichobezoars (due to trichophagia) can lead to iron-deficiency anemia, rather than the other way around.[18] Given that the only material found in the bezoar was hair, it is unlikely that pica was the primary diagnosis.

Ultimately, for this patient, it would be important to clarify whether she did indeed have trichotillomania and to further understand her motivations and experience regarding the trichophagia. Was there a component of stress reduction? Was she aware of the behavior? Did she find it satisfying or tension-relieving? We would also want to confirm whether she ingests any other nonnutritive substances. A better understanding of these aspects of the patient's case will help to determine a personalized treatment approach that will most likely involve a combination of psychoeducation, behavioral therapy, and possible psychopharmacologic therapy.

Dr. Leonard: The patient had an uneventful postoperative course. She had no complications from oral intake on postoperative day 3 and was discharged home on postoperative day 5. At a surgical follow-up visit 1 month after discharge, the incision was found to be healing as expected. At that time, she reported that her appetite had improved and that her abdominal pain had resolved. She was referred to a psychiatrist, but she did not present for follow-up and reports that she plans to follow up with a hypnotherapist that was recommended by her friends.

FINAL DIAGNOSIS

Trichobezoar.

This case was presented at the Harvard Medical School 2022 postgraduate course "Primary Care Pediatrics," directed by Peter T. Greenspan, M.D., Ronni L. Goldsmith, M.D., Benjamin A. Nelson, M.D., John Patrick T. Co, M.D., M.P.H., and Janice A. Lowe, M.D., and administered by Jennifer Timulty, M.B.A.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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