Chronic Vomiting and Diarrhea in a Young Adult Female: A Case Report

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ABSTRACT
Rumination syndrome is a behavioral disorder that consists of an effortless regurgitation of undigested food that is subsequently either re-swallowed or ejected within minutes of meal ingestion occurring with liquids and solids. It was first described in children that are mentally disadvantaged but has gained more attention to also occur among both adolescents and adults of normal mental capacity. The prevalence has never accurately been assessed due to its rarity and frequent misdiagnosis. Rumination syndrome is frequently confused with bulimia nervosa, gastroesophageal reflux disease, and upper gastrointestinal motility disorders such as gastroparesis and chronic intestinal pseudo-obstruction. Our patient illustrates the possible overlap of Rumination syndrome with an eating disorder, thus creating a challenge of diagnosis and management. Due to its elusive nature rumination syndrome is both underdiagnosed and a misdiagnosed condition. One of the reasons for a delayed diagnosis in patients with rumination syndrome is that many physicians are unaware of it, or are even reluctant to make this diagnosis as it could easily be confused with an eating disorder or with other gastrointestinal motility disorders. Because this syndrome may be left undiagnosed for months to years, patients often undergo many expensive and invasive procedures.

BACKGROUND INFORMATION
Rumination syndrome is a behavioral disorder that consists of an effortless regurgitation of undigested food that is subsequently re-swallowed or ejected within minutes of meal ingestion. It is a chronic condition that typically occurs on a daily basis after every meal, occurring with both liquids and solids. The repetitive regurgitation of gastric contents starts typically within minutes and can persist for up to one to two hours afterwards. The regurgitation can be preceded by a sensation of belching immediately before the regurgitation. The act of regurgitation in this condition is a reflex response, not a conscious decision. It was first described in children that are mentally disadvantaged, but has gained more attention because of recent diagnosis among adolescence and adults of normal mental capacity with a higher prevalence in females. Rumination syndrome is frequently confused with bulimia nervosa, gastroesophageal reflux disease, and upper gastrointestinal motility disorders such as gastroparesis and chronic intestinal pseudo-obstruction.1 The etiology and physiological mechanisms of this syndrome are poorly understood. Complications from rumination syndrome include weight loss, malnutrition, dental erosions, halitosis, electrolyte abnormalities and significant functional disability.

The act of rumination is a consequence of a learned, voluntary relaxation of the lower esophageal sphincter or diaphragmatic crura. This allows the increased intragastric hydrostatic pressure in the postprandial period and tonic contractions of the proximal stomach to overcome the resistance to regurgitation usually provided by the antireflux mechanisms at the esophageal junction, leading to the retrograde movement of the gastric contents.2 The current strategy to diagnosis rumination syndrome is based on the Rome III clinical diagnostic criteria. The criteria must be fulfilled for the last three months with symptom onset at least six months before diagnosis. The required criteria must include both persistent and recurrent regurgitation of recently digested food and that the regurgitation was not preceded by retching. Additional supportive criteria that are not necessarily required include regurgitation events not usually preceded by nausea; cessation of the process with acidification of the regurgitated material; and regurgitant contents containing recognizable food with a pleasant taste. In the case of diagnostic uncertainty, a manometric evaluation may confirm the diagnosis of rumination syndrome by revealing tall R waves in gastric manometry tracings.3

CASE PRESENTATION
A 25 year old Caucasian female presented to our care with a chronic history of nausea, vomiting and diarrhea. For the past four years, she has vomited immediately during the postprandial period, occurring within seven to twenty-five minutes after per oral (PO) intake and six hours after each jejunal tube feeding. The vomiting would not occur during the night when asleep. During the nocturnal jejunal tube feeding, she would have several bouts of diarrhea, amounting to as much as ten episodes of loose stool per day. Within the past year, she lost 25 pounds unintentionally, likely due to her poor food intake and chronic diarrhea. She also complained of mid-epigastric abdominal pain, described as an intermittent, cramping sensation of moderate intensity with no radiation. Other complaints included frequent migraines, a tingling sensation in her hands and feet, occasional episodes of hyperterventing, and five episodes of syncope within the past year with the most recent occurrence being one week prior to admission. Due to her illness, she has

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lost her job and has had to move in with her parents due to financial instability. She has feelings of guilt and depression due to the financial burden that her condition has placed upon her family, but does not have any suicidal ideations. She denied symptoms of fever, chills, palpitations, dyspnea, or dysphagia.

Her past medical history includes asthma and anxiety. She specifically recalled an isolated event of severe food poisoning that occurred approximately six years prior to admission and a few months prior to the start of her symptoms of rumination. Due to her persistent abdominal pain, an abdominal ultrasound was obtained at another facility that revealed biliary sludge in the gallbladder. A HIDA scan was subsequently obtained and revealed an ejection fraction of 20%, leading to a laparoscopic cholecystectomy in March of 2010. Her diagnosis was initially confused with gastroparesis, which led to the placement of a gastric stimulator in October of 2010. There was a less than 10% response to this device. As a result, the patient underwent a pyloroplasty in January of 2011. This surgery initially improved her symptoms for 2-3 weeks. Due to concerns of her nutritional status, she had a J-tube placed in August of 2010 that later had to be replaced in August of 2012 due to infection. A trial of total parenteral nutrition was initiated in October of 2012 but had to be discontinued a few days later due to a PICC line infection. Social history was only significant for an occasional glass of wine on social occasions but no recent use was reported. The father claimed to have found bisacodyl in her apartment when he was moving her into his home. There is no definitive evidence of chronic laxative use or self-induced vomiting. She has previously tried working with a relaxation therapist to help control the episodes of vomiting to no avail. Relevant past history includes the death of her grandfather at the age of fourteen years old for which she received counseling to improve the grieving process.

Physical examination findings revealed a cachectic female patient with a body mass index (BMI) of 16, and vital signs that were only significant for hypotension. She was in no acute distress with a normal mood, affect, attention span, and concentration. Abdominal examination was soft, not distended, and not tender to palpation. A J-tube was located in the left upper quadrant and an enteral device in the right upper quadrant, both with no apparent signs of infection. The remainder of the physical examination did not reveal any significant findings. Inpatient psychiatric evaluation confirmed that the patient had multiple psychiatric symptoms of depression, anxiety, and obsessive compulsive disorder. The initial laboratory findings revealed a white blood cell count of 11.05, hemoglobin of 14.2, hematocrit of 42.6, platelet count of 603,000, sodium of 140, potassium of 3.8, chloride of 103, blood urea nitrogen level of 13, serum creatinine of 0.58, and calcium of 10.5. Iron panel findings were consistent with iron deficiency anemia. Electrocardiogram on admission showed normal sinus rhythm, and normal axis with non-specific T wave abnormality.

Our working diagnosis upon admission was rumination syndrome with conditioned vomiting. The initial hospital course consisted of keeping the patient per os (NPO); intravenous fluid hydration; turning off the gastric stimulator; starting ciprofloxacin and metronidazole for suspected small bowel bacterial overgrowth; and octreotide to decrease gastric, pancreatic, and small bowel secretions, and for reduction of diarrhea. Other medications included loperamide, dicyclomine, nortriptiline, and scopolamine for nausea and pain control. The primary goal of our management plan was to decrease the frequency of vomiting and diarrhea. Once the nausea, vomiting, and diarrhea were better controlled, we gradually increased the rate of J-tube feeding to enhance her nutritional status.

We took a conservative approach in our management and had to determine the functional status of the stomach. On the tenth hospital day, the general surgery team performed removal of the gastric stimulator, revision of the feeding jejunostomy tube by replacement with a Mic-Key button, and full thickness gastric biopsies to study the smooth muscle. For two to three days after the operation, she suffered from severe abdominal pain and post ileus that eventually resolved with the return of bowel function.

The surgical pathology results from the gastric biopsies revealed no evidence of inflammation, necrosis, intestinal metaplasia, dysplasia, or malignancy. The nerve bundles and ganglion cells (myenteric plexus) were positive for S100 immunostain and were normal in number. An adequate population of cells of Cajal within the muscularis propria was indicated by positive e-kit staining. Ultimately these findings confirmed that the stomach was functionally normal. Afterwards, the patient understood the meaning of the results and agreed to put forth a very dedicated effort towards relaxation behavioral techniques with a psychologist in an outpatient setting. She was subsequently discharged on the 17th hospital day with all necessary discharge instructions, medications, and nutrition recommendations for nocturnal tube feedings.

DISCUSSION
Approximately 17% of female patients diagnosed with rumination syndrome have been reported to have a history of bulimia. One could speculate that it may be a learned behavior in which patients are able to purge themselves without digitally inducing frank vomiting. Alternatively, rumination might be thought of as a variant of bulimia nervosa or an atypical eating disorder.

It is hypothesized that patients with rumination syndrome use neural pathways to induce voluntary lower esophageal sphincter relaxation by abdominal wall contractions. Antroduodenal manometry (ADM) shows brief simultaneous pressure increases (R waves) associated with regurgitation, representing an involuntary act to increase the intra-abdominal pressure to induce regurgitation. The timing of rumination can serve as a differentiating point from gastroparesis, in which vomiting is delayed for greater than one hour after eating. Several diagnostic procedures that patients with rumination syndrome commonly undergo include: esophageal motility, upper gastrointestinal motility, and gastric myoelectrical activity and gastric emptying studies. The results of these diagnostic procedures as established by a research study demonstrated that patients with rumination syndrome had a normal esophageal motility study, upper gastrointestinal motility with normal fasting and fed motility patterns, a gastric myoelectrical activity that had normal 2-4 cycles/min slow waves with no dysrhythmia, and gastric emptying normal for a solid meal.

Currently, treatment of rumination syndrome is unsatisfactory in part because the pathophysiological mechanism is unclear.
Medications for rumination disease include acid blocking agents, prokinetic medications, antiemetics, anticholinergics, anxiolytics, and antidepressants. These medications are for symptomatic relief and are not aimed at the underlying issues. Nissen fundoplication has been tried but has been reported to lead to complications like retching, bloating, and gastroparesis. Reassurance and behavioral therapy is currently the mainstay of treatment, with a reported success of 80%. This involves habit reversal by using several strategies such as positive encouragement not to vomit, biofeedback relaxation, and diaphragmatic breathing. Diaphragmatic breathing is the main technique taught to patients to reverse the habit of abdominal wall contraction and possibly alleviate symptoms of regurgitation associated with rumination syndrome. The breathing approach distracts the patient from the urge or temptation to regurgitate.

In summary, rumination syndrome is a commonly underdiagnosed and misdiagnosed condition. One of the reasons for a delayed diagnosis in patients with rumination syndrome is that many physicians are unaware of it, or are even reluctant to make the diagnosis of rumination syndrome because it could be easily confused with an eating disorder or with other gastrointestinal motility disorders. Due to frequent misunderstanding and misdiagnosis, these patients are exposed to many unnecessary diagnostic tests and procedures. It is essential for physicians to be aware and knowledgeable of the syndrome to help prevent delayed diagnosis and secure healthcare cost containment. Our patient illustrates the possible overlap of rumination syndrome with an eating disorder, thus creating a challenge with diagnosis and management.

REFERENCES


