

CASES THAT TEST YOUR SKILLS

Persistent vomiting severely compromised Ms. M's quality of life. Through her four hospitalizations and the loss of 50 lbs across 5 months, clinicians from various disciplines could not pinpoint the cause of her emesis. How would you help this woman?

The patient who couldn't stop vomiting

Case history A sudden GI problem

s. M, 32, was transferred to our university medical center's psychiatric ward for assessment and treatment of persistent vomiting. She had been in good health until 5 months previously, when she was hospitalized with dehydration after a weeklong bout with acute nausea and vomiting. An ultrasonogram of the abdomen and pelvis and oral cholecystography were normal.

Ms. M's diagnosis at that time was gastroenteritis. She received IV rehydration and was released after 5 days, with results of thyroid stimulating hormone (TSH) tests pending. TSH results received 1 week post-hospitalization were 50 mU/ml. (A normal reading is <10 mU/ml.) Ms. M's diagnosis was changed to primary hypothyroidism, and levothyroxine, 0.1 mg/d, was begun. The thyroid supplement did not relieve her GI symptoms, however, so Ms. M also received H-2 blockers and antiemetics.

Symptoms re-emerged 3 weeks later, and the patient was hospitalized again for persistent fatigue, nausea, vomiting, and dehydration. Additional lab test results were normal. The patient was discharged tolerating a regular diet following 2 days of IV rehydration and an increase in antiemetic chemotherapy.

The patient's emesis continued despite treatment,

and she was losing weight. A general surgeon examined Ms. M 3 months after she became ill. He recommended no further work-up, citing the absence of abdominal pain or stool changes and an unclear etiology.

Three weeks later, Ms. M was referred to a dermatologist for evaluation of pigmentation changes of her lips and gums. Hyperpigmentation of the oral mucosa had developed over several weeks. A dermatologic evaluation indicated hypervitaminosis, although vitamin supplementation was denied.

Severe continuous vomiting resulting in dehydration precipitated the third hospitalization in 4 months. Ms. M continued to lose weight, dropping 25 lbs since illness onset. Diagnostic studies, including a head CT, urine drug screen, electrolyte assessment, and thyroid function tests were normal. Again no specific etiology was identified, and a psychologist was consulted. Results of the Minnesota Multiphasic Personality Inventory test demonstrated a strong tendency toward somatization. Significant depression also was noted, and Ms. M was diagnosed with dysthymia.

Ms. M's clinical status improved slightly after 3 more days of IV rehydration. She was discharged tolerating a regular diet, and the treatment plan included psychological counseling. Three days later, the patient was admitted to a hospital psychiatric ward with continuous vomiting. She was noted to be confused, lethargic, and hysterical.

At this point, would you suspect anorexia nervosa or bulimia? If not, how else would you explain Ms. M's persistent vomiting?

Dr. Wiseman's and Dr. Dunlop's observations

Psychogenic vomiting is a form of chronic emesis where the etiology is unrelated to a primary anatomic or physiologic defect. It differs from anorexia nervosa or bulimorexia because it lacks the characteristic features of body image distortion, abnormal fear of weight gain, or self-induced vomiting.¹ Symptoms include absence of nausea, insignificant weight loss, occurrence usually after meals, and limited control over emesis.²

Out of 24 patients with psychogenic vomiting examined by Rosenthal, 18 received psychological evaluation and testing.³ The illness was found to be chronic but not debilitating in these patients. Most were without major psychiatric diagnoses or personality disorders, although stressful life events were temporally related with acute exacerbations of vomiting.

Muraoka, in a study of 59 patients admitted with psychogenic vomiting, concluded that most subjects had previous structural or functional GI problems and a history of emesis.⁴

Ms. M did not exhibit body image distortion, nor was she causing herself to vomit or attempting to lose weight. Instead, there appeared to be no medical reason for her symptoms. It was therefore assumed she had psychogenic vomiting.

Patient history A 'death-bed' promise

ccording to the social history obtained by the hospital's psychiatric team. Ms. M is a married mother

of three who works part-time. She denied using alcohol or other substances and had recently stopped smoking

with the aid of hypnosis. She did not follow a special diet, took no medications, and was quite active when healthy. Her father had been treated for hyperthyroidism; otherwise her medical, psychiatric, and family history were noncontributory.

The patient described her relationship with her husband and her children as good, without significant interpersonal conflicts. The psychiatric team, however, viewed her husband as domineering and her as dependent upon him to make decisions.

The patient reported the death of a friend from complications of anorexia nervosa 6

months before the onset of her current symptoms. Ms. M had made a "death-bed promise" to care for her friend's elderly mother. A contract was written, and Ms. M was remunerated for providing companionship and housekeeping. Before her symptoms developed, the patient's time commitment to this task increased to the point that she felt "she was neglecting her own family." Upon becoming ill, however, she willingly terminated this agreement.

About 1 week before the onset of vomiting, Ms. M, who was approximately 60 lbs overweight at the onset of symptoms, received a subliminal message tape from her father pertaining to weight loss. She had listened to the tape for several days and wondered if this could have bearing on her current symptoms, since the tape represented the only change in her routine before the vomiting started.

The attending psychiatrist surmised that the escalating demands of the patient's elderly ward created a psychological conflict between caring for her family and for her deceased friend's mother. Although the recurrent hospitalizations added to her stress, the vomiting relieved her internal conflict by rendering her unable to care for anyone, including herself.

Because physical examination findings, blood chemistries and counts, and imaging studies did not indicate a physical disorder, Ms. M's diagnosis now was psychogenic vomiting and eating disorder not otherwise specified (NOS).

continued

How would you treat this apparent case of psychogenic vomiting? To what extent can psychological factors contribute to excessive vomiting or other gastrointestinal distress?

Dr. Wiseman's and Dr. Dunlop's observations

Clarke described the use of emesis to communicate psychological distress in three cases of female Asian immigrants with psychogenic vomiting.⁵ In each patient the onset of emesis was related to a psychological stressor, and each patient felt she gained a benefit from the chronic vomiting.

In one case, a young woman with unexplained vomiting for 3 weeks was admitted to a psychiatric hospital. During several interviews the patient revealed that she was angry with her parents because she had been relocated against her wishes and required to perform traditional domestic duties. She also felt frightened that her hostile thoughts may have caused her father to have a heart attack. A psychiatric social worker intervened with the patient and her family to address the woman's desire to be independent. The vomiting stopped without recurrence. Continued vomiting would have changed the family's expectations and demands, as the patient would have assumed the role of the chronically ill family member.

Of the patients with persistent psychogenic vomiting reviewed by Rosenthal, most had adjustment disorders, poor assertiveness skills, or lack of limit-setting abilities.³ In this psychological setting, vomiting can best be understood as a mechanism for decreasing anxiety and unconscious conflict. Many patients reported difficulty expressing anger, and vomiting eliminated the negative emotion.³

Further evaluaton Another diagnosis

D uring her fourth hospitalization, this time at our medical center, Ms. M was treated with amitriptyline, 50 mg at bedtime, but this was discontinued when she developed orthostatic hypotension. Following 2 weeks of psychiatric inpatient treatment without improvement, Ms. M was transferred to a tertiary medical center's psychiatric service for further evaluation.

The examination revealed a mildly dehydrated, ill-appearing woman with sallow skin, hyperpigmentation of the oral mucosa, and minimal enlargement of the thyroid gland. Her blood pressure was 88/62 mm Hg, and her pulse was 112/min supine with orthostatic changes. Her weight had decreased from 195 to 144 lbs across 5 months. Serum sodium was 134 mmol/L (135-145); potassium,

5.7 mmol/L (3.5-5.5); chloride, 100 mmol/L (95-105); bicarbonate, 22 mmol/L (22-27); urea, 7 mg/dl (5-20); creatinine, 1.0 mg/dl (0.5-1.5); glucose, 117 mg/dl (70-110); and calcium, 9.6 mg/dl (8.5-10.5). Ms. M's complete blood count was normal.

A mental status exam revealed a cooperative but anxious and ill woman without delusions or distorted body image. Ms. M did exhibit a depressed mood and diminished concentration. Her affect was sad, but she denied hopelessness or worthlessness and did not meet DSM-IV criteria for major depression or personality disorder. Results of the modified Mini Mental State Examination were normal.

As we accumulated and carefully reviewed the patient's medical records, a constellation of signs and symptoms became apparent. These included fatigue, nausea, vomiting, hypotension, hyponatremia, hyperkalemia, weight loss, hyperpigmentation, and the report of subtle episodic psychiatric changes. The index of suspicion for a primary pathophysiologic process was raised. Hypothalamic-pituitary-adrenal axis pathology, specifically adrenal insufficiency, was considered.

An abnormal plasma cortisol level obtained at 8 AM was <1.0 mg/dl (normal range: 4-20 mg/dl) with no response to synthetic adrenocorticotropic hormone (ACTH). An ACTH level was not obtained. An abdominal CT with specific attention to the adrenal glands revealed small and atrophic adrenals without masses or calcifications. Mantoux tuberculin skin test results were negative.

A diagnosis of adrenocortical insufficiency, probably caused by autoimmune adrenalitis, was made. In addicontinued on page 67



continued from page 64

tion, a thyroid function test showed TSH 22.5 IU/ml (0.13-4.6), T4 6.3 mg/dl (4.5-12.0), and T3 176 mg/dl (90-245). Consistent with primary hypothyroidism and adrenal insufficiency, Schmidt's syndrome—an autoimmune disease of the adrenal and thyroid glands that is a subtype of Addison's disease—was established.

Ms. M was transferred to a medical ward, where endocrinologists prescribed hydrocortisone, 30 mg in divided daily doses; fludrocortisone acetate, 0.05 mg as a single daily dose; and levothyroxine, 0.1 mg/d. She responded clinically and was discharged after 2 days. A 6month follow-up found the patient symptom-free with body weight exceeding premorbid levels.

How was the diagnosis of Addison's disease missed in this patient? What symptoms mask adrenal insufficiency to the point that it mimics psychogenic vomiting?

multiple diagnoses. Ms. M was diagnosed with conversion disorder, eating disorder NOS, hypervitaminosis, and hypothyroidism, when only one disorder— Schmidt's syndrome—was present.

Second, conversion disorder was mistaken for the etiology when significant physical signs and symptoms and laboratory abnormalities—including weight loss, severe debilitation, dehydration, hyperpigmentation, hyponatremia, and hyperkalemia—were present.

This case illustrates the imperative to rule out adrenal insufficiency in cases of persistent unexplained vomiting. Gastrointestinal symptoms are present in more than one-half of all cases of Addison's disease.¹¹ Intestinal cramps, diarrhea,

> anorexia, and vomiting are nonspecific abdominal symptoms that may delay appropriate diagnosis and treatment. In a study by Tobin et al, eight cases of Addison's disease with significant GI disturbance had a mean duration of 7.6 months before an accurate diagnosis was made.¹⁰

continued

DIAGNOSING CONVERSION DISORDER

Conversion disorder is not a diagnosis of exclusion but has both negative and positive diagnostic criteria. The DSM-IV guidelines for conversion disorder include:

Positive criteria:

- A change in physical functioning suggests a physical abnormality
- A psychosocial stressor produces a psychological conflict that is believed to help initiate or exacerbate the illness

Negative criteria:

- The symptom is not under conscious control
- The symptom has no cultural sanction and cannot be explained by a discernable physical abnormality
- Isolated chronic pain or sexual dysfunction are excluded.

While functional disabilities are common with conversion disorders, physical and laboratory abnormalities are absent or minor in comparison with the patient's subjective complaints.

Source: DSM-IV. Washington, DC: American Psychiatric Association, 2000.

Dr. Wiseman's and Dr. Dunlop's observations

As this case demonstrates, physiologic vomiting may be misclassified as a conversion disorder because symbolic meaning is given to the emesis, psychological gain is inferred from the symptoms, and clinicians cannot find the true cause of the vomiting. (See *Box* for DSM-IV guidelines for diagnosing conversion disorder.)

Several case reports describe various psychiatric manifestations of Addison's disease as components of presenting symptoms of anorexia nervosa, self-mutilation, bereavement, and schizophrenia.⁶⁻⁹ Often specialists from several disciplines see patients with chronic adrenal insufficiency before an accurate diagnosis is established.¹⁰ The presentation is so diverse and insidious that when unexplained emesis is a symptom, psychogenic vomiting may mistakenly be considered the etiology.

In our patient's case, the correct diagnosis was initially obscured for two reasons:

· First, the lack of definitive medical results led to

The patient who couldn't stop vomiting

References

- Garfinkel PE, Kaplan AS, et al. The differentiation of vomiting/weight loss as a conversion disorder from anorexia nervosa. Am J Psychiatry 1983;140(8)1019-22.
- Wruble LD, et al Psychogenic vomiting: a review. *Am J Gastroenterol* 1982;77:318-21.
 Rosenthal RH, Webb WL, Wruble LD. Diagnosis and management of persistent psy-
- chogenic vomiting. *Psychosomatics* 1980;21(9):722-30.4. Muraoka M, Mine K, Matsumoto K, et al. Psychogenic vomiting: the relation
- between patterns of vomiting and psychiatric diagnosis. *Gut* 1990;31(5):526-8.
 Clarke DJ, Salmons PH, Harrison T. Psychogenic vomiting among female Asian immigrants to the United Kingdom. *Int J Soc Psychiatry* 1988;34(3):221-9.
- Tobin MV, Morris AI. Addison's disease presenting as anorexia nervosa in a young man. Postgrad Med J 1988;64(758):953-5.
- 7. Rajathurai A, Chazan BI, Jeans JE. Self mutilation as a feature of Addison's Disease. Br Med J (Clin Res Ed) 1983;287(6398):1027.
- Demilio L, Dackis CA, Gold MS, Ehrenkranz JRL. Addison's disease initially diagnosed as bereavement and conversion disorder. *Am J Psychiatry* 1984;141(12):1647.
- Persistent vomiting caused by adrenal insufficiency can be mistaken for psychogenic vomiting. Clinicians must consider the possibility of endocrine disease or other medical etiologies before rendering a psychiatric diagnosis for persistent vomiting.
 - Bottom

- Saria K. Case of Addison's disease erroneously diagnosed as schizophrenia. *Clin Endocrinol* 1967;15:84-8.
- Tobin MV, Aldridge SA, Morris AL, Belchetz PE, Gilmore IT. Gastrointestinal manifestations of Addison's disease. *Am J Gastroenterol* 1989;84(10)1302-5.
- Kannan CR. Disease of the adrenal cortex. Diseases of the month. St. Louis: Year Book Publishing Inc. 1988;606-74.

Related resources

- Margulies P. Addison's disease: The facts you need to know. National Adrenal Diseases Foundation. http://www.medhelp.org/nadf/nadf3.htm.
- Family Practice notebook.com: Psychogenic vomiting. http://www.fpnotebook.com/GI191.htm
- National Institute of Diabetes & Digestive & Kidney Diseases: Addison's disease. http://www.niddk.nih.gov/health/endo/pubs/ addison/addison.htm

DRUG BRAND NAMES

Fludrocortisone acetate • Florineff

AUTHOR AFFILIATIONS

William J. Wiseman, MD, Psychiatrist, Stress Center, St. Vincent Hospital, Indianapolis, IN

Stephen Dunlop, MD, Director of Behavioral Care, Saint Francis Hospital Center, Indianapolis, IN

DISCLOSURE

The authors report no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.