Bulimia nervosa and binge eating disorder in adults: Medical complications and their management

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INTRODUCTION

Medical complications and symptoms of poor health are common in patients with bulimia nervosa and binge eating disorder [1,2]. As an example, somatic symptoms such as shortness of breath, chest pain, joint pain, gastrointestinal problems, menstrual problems, and headache occur in more individuals with either bulimia nervosa or binge eating disorder compared with individuals who have no psychiatric disorder.

The medical complications of bulimia nervosa and binge eating disorder and their management are reviewed here. The epidemiology, clinical features, diagnosis, and treatment are discussed separately, as are the medical complications of anorexia nervosa.

- (See "Eating disorders: Overview of epidemiology, clinical features, and diagnosis".)
- (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis".)
- (See "Eating disorders: Overview of prevention and treatment".)
- (See "Binge eating disorder in adults: Overview of treatment".)
- (See "Anorexia nervosa in adults and adolescents: Medical complications and their management".)

DEFINITIONS

Bulimia nervosa — Bulimia nervosa is marked by episodes of binge eating (eating an amount food in a discrete period of time that is definitely larger than what most people would eat in a similar period of time). In addition, patients use inappropriate compensatory behaviors to prevent weight gain, including self-induced vomiting; misuse of laxatives, diuretics, or enemas; excessive exercise; fasting; and strict diets (table 1) [3]. The diagnosis of bulimia nervosa is discussed separately. (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Diagnosis'.)

Binge eating disorder — Binge eating disorder is characterized by episodes of binge eating, without the inappropriate compensatory behaviors that are seen in bulimia nervosa [3]. The diagnosis of binge eating disorder is discussed separately. (See "Eating disorders: Overview of epidemiology, clinical features, and diagnosis", section on 'Diagnosis'.)

MEDICAL EVALUATION

The medical evaluation should pursue symptoms and signs of the medical complications described below. In addition, all patients with bulimia nervosa should receive a basic panel of laboratory tests, and additional tests as indicated by findings from the history and physical examination. Patients with medical complaints due to bulimia nervosa often attempt to hide their eating disorder from clinicians [4].

The medical evaluation of patients with binge eating disorder is the same as it is for patients in the general population, and is guided by the medical status of the patient.

Medical history — The most common medical symptoms of bulimia nervosa are lethargy, irregular menses, abdominal pain and bloating, and constipation [4,5].

Physical examination — Within the context of a complete physical examination, key portions include weight and height; vital signs including heart rate, blood pressure both supine and standing, and temperature; skin; oropharyngeal; abdominal and neurologic examination (to look for other causes of weight loss or vomiting, eg, abdominal or central nervous system mass).

Common signs of bulimia nervosa are [2,6,7]:

- Tachycardia
- Hypotension (<90 mmHg systolic)
- Xerosis (dry skin)
- Parotid gland swelling (sialadenosis)

- Erosion of dental enamel

Other signs that are often present include hair loss, edema, and scarring or calluses on the dorsum of the hand (see 'Skin' below). In addition, self-induced vomiting may lead to unexplained subconjunctival hemorrhage and epistaxis [7].

Laboratory assessment — Tests should be guided by the symptoms and physical findings. Laboratory tests indicated for all patients with bulimia nervosa include [8]:

- Serum electrolytes
- Blood urea nitrogen
- Serum creatinine
- Complete blood count including differential
- Liver function tests
- Urinalysis

Severely ill patients with bulimia nervosa warrant additional tests [8]:

- Serum calcium, magnesium, and phosphorous
- Electrocardiogram (ECG)

In addition, all female patients should get a pregnancy test.

For patients with suspected pancreatitis, clinicians should check serum amylase, fractionated for salivary gland isoenzyme [8]. Persistent amenorrhea should be investigated with luteinizing hormone, follicle-stimulating hormone, prolactin, and beta-human chorionic gonadotropin. Suspected laxative abuse can be assessed by checking stool or urine for bisacodyl, emodin, aloe-emodin, and rhein.

No specific laboratory tests are indicated for patients with binge eating disorder, unless they are obese and obesity-associated comorbidity is suspected.

MEDICAL COMPLICATIONS OF BULIMIA NERVOSA

The medical complications that occur in patients with bulimia nervosa affect many organ systems, and depend upon the method and frequency of purging (ie, self-induced vomiting or misuse of laxatives, diuretics, or enemas) [9]. Treatment for each complication includes discontinuation of purging [7]. However, abrupt discontinuation itself can be associated with complications, including pseudo-Bartter syndrome, which can predispose to significant edema [10].

Gastrointestinal — Gastrointestinal complications of bulimia nervosa can include [2,5,7,9,11-15]:

- Parotid and submandibular (salivary) gland hypertrophy, with puffy or swollen cheeks
- Laryngopharyngeal reflux
- Loss of gag reflex
- Esophageal dysmotility
- Abdominal pain and bloating
- Heme-stained emesis
- Mallory-Weiss syndrome (esophageal tears)
- Esophageal rupture (Boerhaave syndrome)
- Gastroesophageal reflux disease (GERD)
- Barrett's esophagus
- Gastric dilation
- Diarrhea and malabsorption
- Steatorrhea
- Protein-losing gastroenteropathy
- Hypokalemic ileus
- Colonic dysmotility
- Constipation
- Irritable bowel syndrome
- Melanosis coli
- Cathartic colon
- Rectal prolapse
- Pancreatitis

The differential diagnosis of vomiting and right upper quadrant pain includes biliary disease [2]. An ultrasound can exclude the presence of gallstones. (See "Acute calculous cholecystitis: Clinical features and diagnosis" and "Approach to the management of gallstones".)

Parotid gland hypertrophy (sialadenosis) is typically bilateral and painless, and occurs in approximately 10 to 25 percent of patients with bulimia nervosa [7,15]. Hypertrophy of the parotid gland is the result of self-induced vomiting and develops within a week of cessation of vomiting [9,15]. Swollen parotid and submandibular glands usually do not require specific treatment beyond cessation of self-induced vomiting, and should decrease in size over several months. For patients with pain due to swollen glands, treatment includes applying hot packs to the glands, anti-inflammatory medication, and sucking on hard, tart candy to promote saliva production [2,9,15]. This approach usually resolves the problem within one to two weeks [15]. If

this is not successful, then oral pilocarpine should be prescribed to stimulate salivary flow, at a dose of 5 mg two to three times per day [2]. Bilateral gland enlargement due to purging should be distinguished from unilateral parotitis, which involves a bacterial infection and requires antibiotic medication.

Laryngopharyngeal reflux is the retrograde movement of gastric contents (acid and enzymes such as pepsin) into the laryngopharynx [7,9]. Symptoms include hoarseness, cough, sore throat, throat clearing, and difficulty swallowing. Additional information about laryngopharyngeal reflux is discussed separately. (See "Laryngopharyngeal reflux in adults: Evaluation, diagnosis, and management".)

Esophageal dysmotility may take the form of achalasia (failure of the lower esophageal muscles to relax) or esophageal spasm (irregular contractions) [9,16]. (See "Achalasia: Pathogenesis, clinical manifestations, and diagnosis" and "Distal esophageal spasm and hypercontractile esophagus".)

Diagnosis and management of GERD are discussed separately. (See "Clinical manifestations and diagnosis of gastroesophageal reflux in adults" and "Medical management of gastroesophageal reflux disease in adults".)

Diagnosis and management of Barrett's esophagus are discussed separately. (See "Barrett's esophagus: Epidemiology, clinical manifestations, and diagnosis" and "Barrett's esophagus: Surveillance and management".)

Management of diarrhea and malabsorption are discussed separately. (See "Overview of the treatment of malabsorption in adults".)

Diagnosis and treatment of protein-losing gastroenteropathy are discussed separately. (See "Protein-losing gastroenteropathy".)

Management of ileus secondary to hypokalemia is discussed separately. (See 'Renal and electrolytes' below and "Clinical manifestations and treatment of hypokalemia in adults".)

Treatment options for constipation include six to eight glasses of water per day; exercise; and 10 gram of fiber per day, one to three tablespoons of polyethylene glycol powder per day, or lactulose [2,5,14]. Patients should avoid high-dose, bulking, fiber-containing laxatives or stimulant laxatives that contain senna, cascara, or bisacodyl, which will worsen constipation. In addition, patients who misuse laxatives in an effort to lose weight should be educated that laxatives act upon the colon, after caloric absorption has occurred. Restoring normal bowel function can take a few weeks [14]. Additional information about the evaluation and

management of chronic constipation is discussed separately. (See "Etiology and evaluation of chronic constipation in adults" and "Management of chronic constipation in adults".)

Irritable bowel syndrome is characterized by chronic abdominal pain and altered bowel habits in the absence of an identified cause. A prospective observational study of patients with bulimia nervosa (n = 64) found that irritable bowel syndrome was present in 69 percent, and was associated with self-induced vomiting [17]. (See "Clinical manifestations and diagnosis of irritable bowel syndrome in adults" and "Treatment of irritable bowel syndrome in adults".)

Melanosis coli appears as a dark brown discoloration of the colon; the functional importance is not clear [9]. (See "Clinical pathological cases in gastroenterology: Small intestine and colon", section on 'Melanosis coli' and "Factitious diarrhea: Clinical manifestations, diagnosis, and management", section on 'Endoscopy'.)

Cathartic colon is a severe manifestation of prolonged laxative use. Chronic use of stimulant laxatives may cause permanent harm and render the colon inert and incapable of propagating fecal material [15]. If stopping abuse of laxatives does not resolve cathartic colon, a colectomy may be necessary. (See "Factitious diarrhea: Clinical manifestations, diagnosis, and management", section on 'Imaging findings'.)

Rectal prolapse (rectal procidentia) has been described in patients with bulimia nervosa, perhaps due to intra-abdominal pressure from vomiting or in the context of constipation [9]. (See "Overview of rectal procidentia (rectal prolapse)".)

Acute pancreatitis may develop in patients who abuse alcohol [2]. A review of 25 studies found that among patients with bulimia nervosa, 23 percent met criteria for alcohol abuse and/or dependence [18]. In addition, pancreatitis can occur in patients with bulimia nervosa who do not abuse alcohol or who have been abstinent from alcohol for many years; the etiology is unclear and not necessarily related to vomiting [19]. Treatment of pancreatitis is discussed separately. (See "Management of acute pancreatitis".)

Mallory-Weiss syndrome — Forceful vomiting may cause longitudinal mucosal lacerations in the distal esophagus and proximal stomach [7]. Patients with the Mallory-Weiss syndrome may vomit bright red blood along with gastric contents. A complete rupture of the esophageal wall (Boerhaave syndrome) is very rare, but extremely dangerous. The rupture usually appears in the lower chest, causes intense pain and contamination of the mediastinum and lungs, and requires immediate emergency care [2]. The diagnosis and treatment of the Mallory-Weiss syndrome and Boerhaave syndrome are discussed separately. (See "Mallory-Weiss syndrome" and "Boerhaave syndrome: Effort rupture of the esophagus".)

Renal and electrolytes — The most common complications of bulimia nervosa related to electrolyte imbalances include (table 2) [2,8,20]:

- Dehydration
- Hypokalemia
- Hypochloremia
- Hyponatremia
- Metabolic alkalosis

Other complications include hypomagnesemia and hypophosphatemia [5].

Routine screening detects an electrolyte abnormality in 10 percent of patients with bulimia nervosa [5]. Self-induced vomiting may be the most common method of purging; vomiting leads to loss of potassium and acid, causing hypokalemia and metabolic alkalosis [15]. Hypokalemia in turn may cause muscle weakness, cardiac arrhythmias, and impair renal function [5,11,14]. Hypokalemia in otherwise healthy young adults is highly specific for covert bulimia nervosa, and cardiac arrhythmias secondary to hypokalemia may explain the increased mortality observed in bulimia nervosa [15]. (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Mortality'.)

The laboratory values that are found in patients with bulimia nervosa, including hypokalemia and metabolic alkalosis (both are secondary to volume depletion), can mimic Bartter syndrome, a rare genetic kidney disease [14]. In addition, abrupt discontinuation of purging may lead to pseudo-Bartter syndrome, which can predispose to fluid retention and edema [10]. (See "Inherited hypokalemic salt-losing tubulopathies: Pathophysiology and overview of clinical manifestations", section on 'Clinical manifestations'.)

To correct an electrolyte abnormality, it is usually sufficient to discontinue the purging behavior [5]. It is worth informing patients that after they stop purging, they are at risk for edema and weight gain for one to two weeks, which may persist for three weeks or longer [14]. The risk is greater for patients who purge frequently (eg, three or more times per week). Edema during the at-risk period may be prevented with dietary salt restriction and leg elevation for 10 to 15 minutes a few (eg, three) times per day. Treatment of severe edema is discussed separately. (See "General principles of the treatment of edema in adults".)

Most patients with bulimia nervosa who develop metabolic alkalosis do so as a result of dehydration and volume depletion; these patients will respond to fluid repletion, usually with isotonic saline (in addition to discontinuing the purging behavior) [20]. In other patients with bulimia nervosa, alkalemia can occur in a state of normal or expanded fluid volume; administration of sodium chloride is not indicated for these patients. The evaluation of

metabolic alkalosis (eg, determining the spot urine chloride concentration) to guide treatment is discussed separately. (See "Clinical manifestations and evaluation of metabolic alkalosis".)

Additional information about managing electrolyte abnormalities is discussed separately:

- (See "Evaluation of the adult patient with hypokalemia".)
- (See "Clinical manifestations and treatment of hypokalemia in adults".)
- (See "Hypomagnesemia: Evaluation and treatment".)
- (See "Hypophosphatemia: Evaluation and treatment".)
- (See "Treatment of metabolic alkalosis".)

Dehydration — Dehydration can cause volume depletion, with symptoms of dizziness and orthostasis. Treatment includes replenishing intravascular fluid with saline [2]. For inpatients, intravenous normal saline should be used. Caution must be exercised to avoid edema [2,14]. Rapid infusion of excessive amounts of intravenous saline (eg, >2 to 3 liters per day) may cause volume overload and marked edema [14].

For outpatients, salt cubes in water or chicken broth should be ingested, starting with three cups per day and increasing as needed [2]. The goal is to increase output of lighter-colored urine, increase venous jugular pressure, and eliminate postural changes in pulse and blood pressure.

Additional information about rehydration is discussed separately. (See "Maintenance and replacement fluid therapy in adults", section on 'Replacement fluid therapy'.)

Cardiovascular

Short-term — Short-term cardiac complications in patients with bulimia nervosa appear to be rare [2,9]. Complications include [7,8]:

- Hypotension and orthostasis (see "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock" and "Mechanisms, causes, and evaluation of orthostatic hypotension")
- Sinus tachycardia (see "Sinus tachycardia: Evaluation and management")
- Palpitations (see "Evaluation of palpitations in adults")
- Edema (see "Clinical manifestations and evaluation of edema in adults")
- Electrocardiogram (ECG) changes (see "ECG tutorial: Basic principles of ECG analysis")

- Depressed ST segment
- QT prolongation
- Widened QRS complex
- Increased P-wave amplitude
- Increased PR interval

- Arrhythmia
  - Supraventricular and ventricular ectopic rhythm (see "ECG tutorial: Atrial and atrioventricular nodal (supraventricular) arrhythmias" and "ECG tutorial: Ventricular arrhythmias")
  - Torsade de pointes (see "Acquired long QT syndrome: Clinical manifestations, diagnosis, and management")

If QT prolongation is present, clinicians should avoid medications that prolong the QT_c interval (table 3) [8]. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

Treatment of short-term cardiovascular complications includes correcting any associated cause, eg, hypokalemia or volume depletion. If complications persist despite corrective measures, or if complications are accompanied by chest pain, shortness of breath, dizziness, or loss of consciousness, a cardiology consult should be obtained.

Longer-term — Bulimia nervosa may be associated with substantial longer-term cardiac complications. A registry study identified women who were hospitalized for bulimia nervosa (n >800) and a comparison group of women who were hospitalized for pregnancy-related events (n >415,000); the women were followed for up to 12 years [21]. After adjusting for potential confounding factors such as sociodemographic characteristics and preexisting psychiatric comorbidity including tobacco use, the analyses found that the risk of future cardiovascular disease was four times greater in patients with bulimia nervosa than controls (hazard ratio 4, 95% CI 3-6). Specific cardiovascular complications that occurred more often with bulimia nervosa included myocardial infarction, conduction disorders, cerebrovascular disease, pulmonary vascular disease, and atherosclerosis. As the significance of this finding is unclear, patients with bulimia nervosa should be assessed for risk of cardiovascular disease using standard risk assessment tools. (See "Atherosclerotic cardiovascular disease risk assessment for primary prevention in adults: Our approach".)

Ipecac-induced myopathy — Ipecac syrup is an emetic that patients with bulimia nervosa may ingest to induce vomiting [7]. Chronic use damages muscle cells including the myocardium, which may lead to cardiomyopathy. Manifestations of cardiac myocyte toxicity include tachycardia, bradycardia, T wave abnormalities on electrocardiogram, interventricular conduction delays, ventricular arrhythmia, and congestive heart failure. Cardiomyopathy may be irreversible [15].

Retrospective studies have reported that approximately 10 to 20 percent of patients have used ipecac [22,23]. Although use of ipecac may have declined because it is no longer available over the counter or as a prescription drug in several countries (including the United States and Canada), it appears that it is possible to obtain the drug through mail order or internet purchases.

Cardiac — One component of ipecac is emetine, which accumulates in cardiac muscle cells and is toxic [24,25]. Emetine is eliminated from the body slowly, and has been detected in urine 60 days after chronic use. Signs and symptoms of cardiotoxicity include precordial chest pain, dyspnea, hypotension, supraventricular tachycardia, premature atrial complexes (also referred to as a premature atrial beats, premature supraventricular complexes, or premature supraventricular beats), flattened or inverted T waves, prolonged QT and PR intervals, ventricular tachycardia and fibrillation, cardiac failure, pericardial effusion, pulmonary congestion, and cardiac arrest.

Small, chronic doses have caused cumulative, fatal toxicity in a few patients. The lethal chronic dose is not known. Absorption of emetine is enhanced in patients who become refractory to the emetic effects of the drug during chronic misuse.

Patients suspected of chronic ipecac abuse should receive a cardiology consult. Treatment includes discontinuation of ipecac and supportive care [25]. No specific antidote or pharmacologic antagonist for the cardiotoxic effects of emetine exists, and patients may die despite intensive care. Recovery may be prolonged (eg, months) because emetine is slowly eliminated from the body.

Skeletal muscle — Chronic abuse of ipecac may damage skeletal muscle [8,22,25]. Symptoms include generalized weakness, especially in the neck and proximal muscles of the extremities; myalgia and tenderness; hyporeflexia; slurred speech; dysphagia; and difficulty with tasks requiring muscular activity (eg, climbing stairs). Ipecac-induced myopathy is associated with elevated liver function tests, nonspecific abnormalities on an electromyographic (EMG) recording, and abnormal muscle biopsy. Normal muscular activity is slowly restored following

discontinuation of ipecac. Chronic ipecac abuse and subsequent cardiac myopathy are both discussed separately. (See 'Ipecac-induced myopathy' above.)

Endocrine — Endocrine complications of bulimia nervosa involve the reproductive and skeletal systems, and there may be an association between bulimia nervosa and diabetes. Severe complications can occur in patients with bulimia nervosa and comorbid diabetes [5].

The most common endocrine complication involves the reproductive system. Among 82 women treated for bulimia nervosa, menstrual irregularities were present in 45 percent at pretreatment and in 31 percent at 12 month follow-up [26]. By contrast, an epidemiologic study of 403 healthy, premenopausal women aged 18 to 39 years found that 5 percent had anovulatory menstrual cycles [27]. Signs and symptoms of reproductive system complications include impaired fertility, spotty and scanty menstrual periods, oligomenorrhea, or amenorrhea [8].

In addition, bulimia nervosa is associated with diabetes mellitus [28], such that the risk is approximately two to three times greater in patients with bulimia nervosa than controls:

- Type 1 diabetes
  - A meta-analysis of eight case-controlled studies (748 female cases with type 1 diabetes and 1587 nondiabetic female controls) found that the prevalence of bulimia nervosa was higher in the diabetic patients compared with the nondiabetic patients (2 versus 1 percent) [29].
  - A national hospital registry study found that the risk of type 1 diabetes mellitus was greater in patients with bulimia nervosa (n >4700) than controls (n >1,000,000) (relative risk 3, 95% CI 2-5) [30].
- Type 2 diabetes
  - A national registry study identified patients with bulimia nervosa (n >1200) and controls matched for age and sex (n >4900), and found that the lifetime prevalence of type 2 diabetes mellitus was greater in patients with bulimia nervosa than controls (4 versus 2 percent) [31].
  - Nationally representative surveys in 19 countries (n >52,000 adults) found that the risk of a self-reported diagnosis of adult onset diabetes (presumably type II) was twofold greater among individuals with a lifetime history of bulimia nervosa, compared with individuals without a history of bulimia nervosa [32].

- A meta-analysis of three cross-sectional studies (sample size not reported) found that type 2 diabetes was three times more likely to occur in patients with bulimia nervosa than controls (odds ratio 3, 95% CI 2-6) [33].

Diagnosis and treatment of diabetes are discussed separately. (See "Overview of general medical care in nonpregnant adults with diabetes mellitus".)

Osteopenia and osteoporosis may be more common in bulimia nervosa, particularly in patients who have had previous episodes of anorexia nervosa [8]. Management of osteoporosis is discussed separately. (See "Evaluation and treatment of premenopausal osteoporosis".)

Thyroid function is usually normal in bulimia nervosa [2]. A study compared 75 bulimic women with 64 healthy control women and found similar values for free T4 and free T3 [34].

Dental — Gastric acid in vomitus softens and erodes dental enamel, particularly on the lingual surfaces of the maxillary anterior teeth [7,15]. Erosion may cause sensitivity to hot and cold temperatures in food and drinks [2]. Some patients exhibit decalcification of the teeth (perimylolysis), particularly on the lingual, palatal, and posterior occlusal surfaces [4]. Teeth may also become discolored, and caries and gum disease may occur. Bulimia nervosa is also associated with exfoliative cheilitis, labial erythema, orange-yellow palate, hemorrhagic lesions, and nonspecific oral atrophies [35].

Studies of the frequency of dental complications in bulimia nervosa include the following:

- A survey of patients with bulimia nervosa (n >200) found that at least one dental problem was present in more than 90 percent, including sensitive teeth or gingivae (69 percent), erosion of enamel (64 percent), tooth pain (43 percent), gingival recession (39 percent), and caries (37 percent) [36].

- A meta-analysis of three studies of patients with eating disorders (number of patients not reported) found that tooth erosion was 20 times greater among patients with self-induced vomiting than patients who did not self-induce vomiting [37].

  For patients who are unable to stop vomiting, enamel loss can be reduced by instructing patients to wash out their mouth with water or fluoride solution after vomiting, gargle with baking soda in water to alkalinize their mouth, wait at least 30 minutes before brushing their teeth, and use alkaline toothpaste [2,9]. Brushing one's teeth immediately after purging may increase dental erosion [36]. Hot and cold foods and liquids should be limited as needed to avoid pain [2]. Acidic foods (eg, fruit and yogurt) should also be limited [5]. Chewing sugarless gum is recommended to increase salivary flow. Outside of

vomiting episodes, patients should brush their teeth with fluoride toothpaste and floss on a daily basis [9]. In addition, a dentist should be consulted for restorative procedures after purging is controlled.

Skin — A nearly pathognomonic dermatologic complication is scarring or calluses on the dorsum of the hand (Russell's sign), due to pressure of the teeth against the skin while stimulating the gag reflex to induce vomiting (picture 1) [7,8]. A study of 122 patients with bulimia nervosa detected this sign in 30 percent [38]. Xerosis, poor skin turgor, petechia, telogen effluvium, and acne may also be seen [39].

Self-injurious behavior is common in bulimia nervosa, and patients may show acute or chronic signs of trauma from cuts or burns [5].

Other — Bulimia nervosa is associated with other general medical conditions beyond those described above. In a national hospital registry study that identified patients with bulimia nervosa (n >4700) and controls with minor medical conditions (n >1,000,000), the risk of the following medical conditions was greater in patients with bulimia nervosa than controls [30]:

- **Adrenal insufficiency (Addison disease)** – Relative risk (in bulimia nervosa) 7, 95% CI 2-16
- **Celiac disease** – Relative risk 2, 95% CI 1-4
- **Psoriasis** – Relative risk 2, 95% CI 1-3
- **Vitamin B12 deficiency** – Relative risk 4, 95% CI 2-8

Other medical complications of bulimia nervosa include fluctuation of body weight and aspiration pneumonitis [2,8,25].

MEDICAL COMPLICATIONS OF BINGE EATING DISORDER

Patients with binge eating disorder often describe somatic symptoms and dissatisfaction with health. However, there is little compelling evidence that binge-eating symptoms directly cause medical complications [28,40,41]. Studies suggesting that binge eating disorder may be associated with medical complications include the following:

- Reviews found that after controlling for body mass index, patients with binge eating disorder may possibly be at increased risk for developing components of the metabolic syndrome, such as dyslipidemia and glucose dysregulation [42,43]. However, the literature is limited and not definitive.

- A prospective observational study of 59 obese women with a lifetime history of binge eating and 107 obese women without binge eating compared the prevalence of self-reported hypertension, cardiac problems, diabetes (type 1 or 2), osteoarthritis, asthma, visual impairment, or any major medical disorder [41]. Patients with binge eating reported higher rates of each disorder, but the differences did not reach statistical significance.

- An epidemiologic study found that joint pain, headache, gastrointestinal problems, menstrual problems, shortness of breath, and chest pain each occurred in significantly more patients with binge eating disorder than patients without psychiatric disorders [28]. However, somatic symptoms do not necessarily equate with medical complications. Another finding was that more patients with binge eating disorder described their general health as poor or very poor, compared with patients free of psychiatric disorders (7 versus 1 percent), but the association of binge eating disorder with poor health was not statistically significant when the analyses controlled for comorbid mood disorders.

Obesity — Many patients with binge eating disorder are obese and suffer the medical complications associated with obesity [4,11]. The complications of obesity are discussed separately. (See "Overweight and obesity in adults: Health consequences".)

It is not clear if binge eating disorder is associated with medical complications beyond those observed in obese patients. Several studies show higher rates of gastrointestinal, cardiovascular, pulmonary, and rheumatologic disorders or symptoms in obese patients with binge eating disorder compared to obese patients without binge eating disorder, but the differences are generally not statistically significant [2,4,41].

Diabetes — Binge eating disorder appears to be associated with diabetes:

- A national registry study identified patients with binge eating disorder (n = 113) and controls matched for age and sex (n = 656), and found that the lifetime prevalence of type 2 diabetes mellitus was greater in patients with binge eating disorder than controls (34 versus 4 percent) [31].

- A study of female patients (n >4600) treated in primary care and obstetric gynecology settings found that diabetes was present in more patients with binge eating disorder compared with patients who had no eating disorder (6 versus 3 percent) [28].

- A meta-analysis of five cross-sectional studies (sample size not reported) found that type 2 diabetes was approximately four times more likely to occur in patients with bulimia nervosa than controls (odds ratio 3.7, 95% CI 1.1-12.1) [33].

However, most studies indicate that binge eating disorder or binge eating do not significantly affect glycemic control in type 2 diabetes [2].

Diagnosis and treatment of diabetes are discussed separately. (See "Overview of general medical care in nonpregnant adults with diabetes mellitus".)

Hypercholesterolemia — A five-year prospective observational study included patients with binge eating disorder (n = 134) who were matched for age, sex, and body mass index to individuals with no eating disorder, and found that hypercholesterolemia occurred in more patients with binge eating disorder (30 versus 17 percent) [40].

HOSPITALIZATION

Certain medical complications require inpatient treatment on an internal medicine, psychiatric, or combined ward. The choice depends upon the patient's medical and psychiatric status, and available resources. Criteria for inpatient treatment and principles of hospital management are discussed separately in the context of anorexia nervosa. (See "Anorexia nervosa in adults: Evaluation for medical complications and criteria for hospitalization to manage these complications".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Bulimia nervosa (The Basics)")

SUMMARY

- **Clinical features and diagnosis**
  - **Bulimia nervosa** – Bulimia nervosa is marked by episodes of binge eating (eating an amount food in a discrete period of time that is definitely larger than what most people would eat in a similar period of time). In addition, patients use inappropriate compensatory behaviors to prevent weight gain, including self-induced vomiting; misuse of laxatives, diuretics, or enemas; excessive exercise; fasting; or strict diets (table 1). (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis".)
  - **Binge eating disorder** – Binge eating disorder is characterized by episodes of binge eating, without the inappropriate compensatory behaviors that are seen in bulimia nervosa. (See "Eating disorders: Overview of epidemiology, clinical features, and diagnosis", section on 'Binge eating disorder'.)
- **Evaluation**
  - **General medical symptoms** – The most common general medical symptoms of bulimia nervosa are lethargy, irregular menses, abdominal pain and bloating, and constipation. (See 'Medical history' above.)
  - **Physical examination and common findings** – Within the context of a complete physical examination, key portions include weight and height; vital signs including heart rate, blood pressure both supine and standing, and temperature; skin; oropharyngeal; abdominal and neurologic examination to evaluate for other causes of weight loss or vomiting. Common signs of bulimia nervosa are tachycardia, hypotension, xerosis, parotid gland swelling, and erosion of dental enamel. (See 'Physical examination' above.)
- **General medical complications of bulimia nervosa**
  - **Gastrointestinal** – Gastrointestinal complications include parotid and submandibular gland hypertrophy; loss of gag reflex; abdominal pain, bloating and dilatation; Mallory-Weiss syndrome, gastroesophageal reflux disease (GERD); diarrhea and malabsorption; constipation; and colonic dysmotility. (See 'Gastrointestinal' above.)
  - **Renal and electrolyte** – The most common renal and electrolyte complications include dehydration, hypokalemia, hypochloremia, and metabolic alkalosis. (See 'Renal and

electrolytes' above.)

- **Cardiac** – Cardiac complications are rare. Complications observed in patients with bulimia nervosa include hypotension and orthostasis, sinus tachycardia, palpitations, edema, electrocardiogram changes, and arrhythmia. (See 'Cardiovascular' above.)

- **Ipecac-induced myopathy** – Ipecac is used by some patients with bulimia nervosa to induce vomiting, and chronic abuse may cause cardiomyopathy and damage skeletal muscle. (See 'Ipecac-induced myopathy' above and 'Skeletal muscle' above.)

- **Endocrine** – Endocrine complications of bulimia nervosa involve the reproductive and skeletal systems, and there may be an association between bulimia nervosa and diabetes. (See 'Endocrine' above.)

- **Dental** – Dental complications of bulimia nervosa include erosion of dental enamel, decalcification and discoloration of the teeth, caries, and gum disease. (See 'Dental' above.)

- **Dermatologic** – Dermatologic complications include scarring or calluses on the dorsum of the hand (Russell's sign) (picture 1), xerosis, poor skin turgor, petechia, telogen effluvium, and acne. In addition, patients with self-injurious behavior will show acute or chronic signs of trauma from cuts or burns. (See 'Skin' above.)

- **General medical complications of binge eating disorder** – Patients with binge eating disorder often describe somatic symptoms and dissatisfaction with health. However, there is little evidence of general medical complications that can be directly attributed to binge-eating symptoms. (See 'Medical complications of binge eating disorder' above.)

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