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Review

Bulimia: Medical Complications

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ABSTRACT

Bulimia nervosa is a common eating disorder that predominantly affects young women. There are three main models of purging in bulimia. Resulting medical complications are related to the particular mode and frequency of purging. Commonly, there are oral and gastrointestinal complications along with serious electrolyte and endocrine complications. The majority of the medical complications of bulimia nervosa are treatable if diagnosed in a timely fashion. Some of these patients require inpatient hospitalization, and others can be managed along a continuum of outpatient care. The American Psychiatric Association has comprehensive treatment guidelines for the management of bulimia. Primary care physicians and gynecologists need to be familiar with this disorder and its medical implications.

INTRODUCTION

BULIMIA NERVOSA WAS FIRST IDENTIFIED as a distinct disorder by Gerald Russell, M.D., in the late 1970s.[1] As with anorexia nervosa, bulimia nervosa is an illness that afflicts primarily young females ages 12–35, although recently there has been an increased incidence in women in midlife. The reported lifetime prevalence of anorexia nervosa in females ranges form 0.5% to 3.7%.[2,3] In comparison, bulimia nervosa has a lifetime prevalence of 1%–4% and may be as high as 19% in certain select groups of female college students.[4–6] These numbers may be misleading, however, because many more persons are afflicted with eating disorders that fall short of the formal diagnostic criteria for bulimia nervosa or anorexia nervosa but are no less debilitating. Binge eating (similar to bulimia nervosa except it lacks the purging component), for example, has been reported in close to 80% of female college undergraduates. The ratio of female/male patients is in the range of 10:1–20:1.

DIAGNOSTIC CRITERIA

Although there are a few physical and laboratory findings that are strongly suggestive of bulimia nervosa, the diagnosis is based on specific behavioral criteria (Table 1). A high index of suspicion in the right clinical setting, together with sensitivity and respect for the patient, will aid the clinician in eliciting the historical features necessary for making the diagnosis. The high prevalence of this disorder in young women would seem to mandate routine screening in this population, although this has not yet been established

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TABLE 1. DSM-IV CRITERIA FOR BULIMIA NERVOSA[a]

Binge eating (≥2 times / week for 3 months)
Purging or other compensatory weight loss measures (≥2 times / week for 3 months)
Regular self-induced vomiting or misuse of laxatives or diuretics in the purging type; other inappropriate compensatory behavior, such as fasting or excessive exercise, in the nonpurging type
Self-image unduly influenced by body weight or shape
Absence of anorexia nervosa

[a]American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed. Washington, DC: APA, 1994.

as the standard of care. There are, however, a few recently developed screening questionnaires that seem to have reasonable utility to diagnose bulimia.[7] According to the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV), bulimia nervosa is characterized by recurrent episodes of binge eating and purging or other compensatory weight loss behaviors occurring at least twice a week for 3 months, an undue influence of body shape and weight on self-image, and the occurrence of these behaviors at times other than during episodes of anorexia nervosa.[8]

There are two subtypes of bulimia nervosa, purging and nonpurging. The purging type is characterized by self-induced vomiting or the use of laxatives or diuretics to induce weight loss and counteract the sequelae of binge eating. The nonpurging type, in contrast, is characterized by fasting or engaging in excessive exercise and the compensatory behaviors for avoiding weight gain from binge eating episodes. To meet clinical criteria according to DSM-IV, binges and the resulting compensatory behavior must occur at a minimum of two times a week for a period of 3 months. Variants that do not clearly fall into either category include the use of diet pills or thyroid hormone, chewing and spitting out food, and in the case of patients with diabetes mellitus, skipping insulin doses to induce an osmotic diuresis and weight loss.

Bulimia nervosa frequently shares company with other psychiatric disorders, including substance abuse, depression, bipolar disorder, anxiety disorders, and posttraumatic stress disorder (PTSD) (up to 37%).[9–12] In fact, their inherent compulsiveness predisposes these patients to other addictive psychosocial behaviors, such as stealing, drug abuse, and alcohol dependence.[13] When present, these comorbid conditions need to be diagnosed and treated effectively to maximize treatment outcomes in individuals suffering from an eating disorder. The complexity of these patients is best served by a multidisciplinary treatment team.[14]

Weight preoccupation is a primary symptom in both anorexia nervosa and bulimia nervosa, and many patients demonstrate a mixture of both anorexic and bulimic symptoms. Up to 50% of patients with anorexia nervosa develop bulimic symptoms, and some patients who are initially bulimic develop anorexic symptoms.[15] The risk of death in bulimia is substantially lower than that in anorexia nervosa, but it is still higher than in age-matched females in the population as a whole.[16]

The pathogenesis of bulimia nervosa can be best understood using a biopsychosocial model. Genetic studies have identified a clear biological basis for eating disorders, and, in fact, anorexia nervosa is as inheritable as schizophrenia. If a woman's mother or sister has anorexia nervosa, her lifetime incidence is 12 times greater than the norm for developing anorexia nervosa and 4 times greater than the norm for developing bulimia nervosa. However, it is clear that although genetics may load the gun, life—that is, family, trauma, dieting, social pressures, western media / advertising—pulls the trigger. Many patients have been exposed to teasing about their body shape, and often these patients have the trait of excessive perfectionism and the negative self-evaluation with which it is associated.[17]

MEDICAL COMPLICATIONS

There are three main modes of purging: self-induced vomiting, abuse of laxatives, and misuse of diuretics. Most patients with bulimia induce vomiting with their finger, but some use ipecac. As the illness progresses, many can simply turn their heads and vomit reflexively without mechanical stimulation. The laxatives abused are the stimulant type containing bisacodyl, cascara, or senna. Diuretic preparations and diet pills, such as those containing ephedrine, are used less frequently. The medical complications of bulimia nervosa are related to the mode and frequency of purging, whereas in anorexia nervosa, they arise as a result of starvation (restricting) and weight loss (Table 2).

TABLE 2. MEDICAL COMPLICATIONS OF BULIMIA

Renal	Cardiovascular
Hypokalemia	Hypotension
Hyperchloremic metabolic acidosis	Syncope
Hyperamylasemia	Arrhythmias
Nonanion gap metabolic acidosis	Cardiomyopathy
Pseudo-Bartter's syndrome	Mitral valve prolapse
Idiopathic edema	
Oral	Endocrine
Perimolysis	Irregular menses
Sialadenosis	False positive dexamethasone suppression test
Xerostomia	Hypoglycemia
Oral mucosal erythema	Diabetes mellitus
Cheilitis	
Gastrointestinal	
Esophagitis	
Dyspepsia	
Gastroesophageal reflux disease	
Esophageal rupture	
Melanosis coli	
Atonic colon	
Cathartic colon	
Steatorrhea	
Protein-losing gastroenteropathy	
Gastrointestinal bleeding	

In a study involving 275 women with bulimia nervosa, the most common symptoms were weakness (83.6%), feeling bloated (75.2%), puffy cheeks (50.1%), dental problems (36.5%), and finger calluses (27.4%).[18]

Renal and electrolyte abnormalities

A number of electrolyte and acid-base abnormalities may result from bulimia nervosa, depending on whether the method of purging used is self-induced vomiting, diuretic misuse, or laxative abuse. Hypokalemia is the most serious abnormality, as it may cause cardiac arrhythmias, rhabdomyolysis, muscle weakness, hypokalemic cardiomyopathy, and tetany. The prevalence of hypokalemia is low and occurs in 4.6% of bulimic patients; it is primarily found in lower-weight bulimics who vomit or use laxatives or both.[19] Given its low sensitivity, screening for hypokalemia cannot be recommended as a means of detecting bulimia nervosa because its absence does not exclude the diagnosis. On the other hand, finding hypokalemia in an otherwise healthy young woman is very specific for bulimia nervosa and suggests daily vomiting or diuretic abuse.

Several mechanisms contribute to the development of hypokalemia in bulimia nervosa. There is a direct loss of potassium as a result of vomiting. The concomitant loss of chloride ions and gastric acid leads to a hypokalemic-hypochloremic metabolic alkalosis. Laxative abuse causes loss of potassium and bicarbonate in the stool, resulting in hypokalemia and a nonanion gap metabolic acidosis. Some diuretics cause renal wasting of potassium. The more significant cause of hypokalemia is activated when purging of any type results in a significant degree of volume depletion. Then, the renal renin-angiotensin hormonal system is activated, leading to high ambient levels of these hormones. These, in turn, drive the renal retention of sodium in exchange for loss of hydrogen and potassium ions, which are excreted in the urine. This results in a metabolic alkalosis in bulimic patients who purge excessively through either self-induced vomiting or diuretic abuse. The most severe cases of metabolic alkalosis are seen with self-induced vomiting (Table 3).

The normotensive, hypokalemic, hypochloremic metabolic alkalosis seen in many patients with bulimia is known as pseudo-Bartter's syndrome and has significant therapeutic implications. Specifically, the efficacy of potassium repletion is abrogated unless there is concomitant normalization of the hypovolemic state.[20,21] On occasion, a bulimic patient will seek care in an emergency room and be found to have severe hypokalemia. Despite massive amounts of potassium repletion, such patients remain hypokalemic because their fluid status has not been

TABLE 3. ELECTROLYTE VALUES USUALLY ASSOCIATED WITH PURGING

	Serum				*Urine*			
Purging method	*Sodium (138–147 mmol/L)*	*Potassium (3.7–5.2 mmol/L)*	*Chloride (101–112 mmol/L)*	*Bicarbonate (22–28 mmol/L)*	*pH (7.38–7.42)*	*Sodium*	*Potassium*	*Chloride*
Vomiting	Increased, decreased, normal	Decreased	Decreased	Increased	Increased	Decreased	Increased	Decreased
Laxative use	Increased, normal	Decreased	Increased, normal	Decreased, normal	Decreased	Decreased	Decreased	Normal
Diuretic use	Decreased, normal	Decreased	Decreased	Increased	Increased	Increased	Increased	Increased

normalized as well. Correcting the volume status improves the metabolic alkalosis and inactivates the renin-angiotensin axis, allowing successful potassium repletion.

A good rule of thumb is that every decrease of 1 mmol/L in the serum potassium level represents a loss of 100–150 mmol in total body potassium. For repletion, oral potassium chloride is generally preferred. Typically, potassium is administered in a split dose of 40–80 mEq/day for a few days, with subsequent measurements of the potassium level to ensure normalization.

Another manifestation of pseudo-Bartter's syndrome is the development of lower extremity edema in those who purge excessively via vomiting, laxatives, or diuretics and then abruptly stop. The unopposed high levels of aldosterone, caused by the abrupt cessation of purging, can cause marked salt retention and edema. Attempts to self-treat this condition with diuretics will exacerbate the problem. Salt restriction, lower extremity elevation, and patience are usually all that is necessary to resolve this problem. Occasionally, it is prudent to prescribe a short, 2-week course of a potassium-sparing diuretic, such as spironolactone, to negate the effects of aldosterone and treat the edema, particularly when the distress over edema threatens a relapse in the patient's bulimic behaviors.

Oral complications

The most obvious oral health disorder seen in bulimia is tooth enamel erosion (perimyolysis). This erosion occurs primarily on the lingual and occlusal surfaces of the maxillary dentition but may also be seen on the facial surfaces in later stages of disease. The erosion is caused by regurgitation of acidic gastric contents on a repetitive basis during self-induced vomiting. The low pH of gastric contents is below the critical threshold for enamel mineralization.[22] Enamel erosion is usually seen after 6 months of self-induced vomiting and is ubiquitous in those who have had the disorder for ≥5 years.[23] Erosion may cause thermal tooth hypersensitivity from dentin exposure. Unfortunately, once tooth enamel is eroded, it will not regenerate, and restorative dental procedures may be necessary. It remains controversial whether the mechanical abrasion from toothbrushing immediately after vomiting worsens this erosion, and several experts recommend mouthwashing with neutralizing solutions, such as sodium bicarbonate.[22]

Despite commonly bingeing with foods high in carbohydrate and fat content, patients with bulimia have lower rates of dental caries, plaque, and periodontal disease. This may be the result of compulsive oral hygiene practices in this population.[24]

Parotid gland enlargement (sialadenosis) is a particularly bothersome disorder seen in patients with bulimia. Enlargement occurs not immediately but after a patient has been in the binge-purge cycle for some time. Once enlargement occurs, however, it can reappear quickly when vomiting resumes. The prevalence of parotid enlargement is estimated to be between 10% and 50% in bulimic patients and occasionally involves swelling of the submandibular glands. Clinically, the swelling develops 3–6 days after a binge-purge episode has stopped, and its severity is thought to be related to the frequency of vomiting behavior. The swelling is usually painless but can be quite disfiguring and, therefore, emotionally disturbing to a patient who is already self-conscious about her physical appearance.

Histologically, parotid gland enlargement is noninflammatory, but the glands exhibit an increase in acinar size and in the number of secretory granules. The mechanism of this enlargement is not entirely clear, but theories include:

1. Work hypertrophy, where repeated chewing and intake of carbohydrate-rich food hyperstimulate glands
2. Autonomic stimulation of salivary glands by pancreatic proteolytic enzymes brought into the mouth during emesis, which stimulate lingual taste receptors
3. Cholinergic stimulation of salivary glands by vomitus, increasing saliva production and secretion, which causes acinar hypertrophy

Treatment of parotid gland enlargement begins with cessation of self-induced vomiting, heat applications, and the use of sialagogues, such as tart candy. For patients unresponsive to these measures, pilocarpine hydrochloride tablets (5 mg three times per day) may be useful.[25] In unresponsive cases, parotidectomy is an option but may cause worsened cosmetic deformity and is rarely indicated.

Erythema of the oral mucosa occurs as a consequence of chronic irritation from vomited gastric contents. Frequently, patients will complain of pharyngeal, palatal, and gingival soreness. Cheilosis is characterized by pallor, maceration, and in severe cases, painful fissures involving the angles of the mouth. Vitamin B_{12} deficiency should be excluded, and efforts should be made toward correction of the primary oral abnormality to maintain lip hygiene and prevent leakage of saliva through the corners of the mouth.

Gastrointestinal effects

The two main sites in the gastrointestinal tract affected by bulimia are the esophagus and colon. Esophageal involvement is found in patients who purge through self-induced vomiting. Depending on the frequency of vomiting, which can range from once to more than 10 times a day, the result may be esophagitis, esophageal ulcers, strictures, or rupture.

Although rare, esophageal rupture (Boerhaave's syndrome) in bulimia is life threatening; the overall mortality is approximately 20%. Early surgical intervention, however, is generally curative. The presentation includes severe chest pain, mediastinitis, and pleural effusions; if the diagnosis is delayed, tachycardia and hypotension may develop.

Barrett's esophagus, in which columnar epithelium replaces normal squamous epithelium as a result of chronic irritation from recurrent reflux or vomiting of acidic stomach contents, has also been described in bulimia. Esophageal adenocarcinoma develops in approximately 0.5% of patients with Barrett's esophagus per year.[26]

Esophageal complications give rise to frequent complaints of sore throat, dyspepsia, dysphagia, or hematemesis.[27] The last is usually the result of Mallory-Weiss tears or esophagitis. Spontaneous reflux of gastric contents into the lower esophagus can also occur and causes significant heartburn as a result of laxity of the lower esophageal sphincter after months of repeated vomiting.

Treatment for esophageal complications of bulimia is similar to that for gastroesophageal reflux disease. Acid suppression with a proton pump inhibitor is of value in this regard. In the patient who tends to vomit spontaneously, a prokinetic agent, such as metoclopramide, is worth considering. It acts by increasing lower esophageal sphincter tone and hastening gastric emptying and, hence, can be a useful adjunct in severely affected patients.

Patients with bulimia nervosa who purge through laxatives may also experience complaints referable to the colon.[28] Some patients may ingest up to 50 laxative pills per day to achieve their desired result. A review of 73 studies of laxative abuse revealed a lifetime incidence of 27.2% among patients with bulimia nervosa compared with 4.18% in the general population. Chronic abuse of stimulant cathartics (compounds containing senna or cascara) can cause melanosis coli (a reversible brown-black discoloration of the colonic mucosa and submucosa), a benign finding. In addition, these patients can develop an atonic colon, resulting in chronic constipation and laxative dependence, or a cathartic colon, a permanently flaccid and dilated colon that cannot propagate fecal material, resulting from damage to the neurological innervation of the intestine. Cathartic colon may cause refractory constipation requiring bowel resection.[29] A previously abused stimulant laxative, phenolphthalein, was removed from the market a few years ago because of concern about its association with colon cancer.

Constipation is particularly problematic during attempts to withdraw stimulant laxatives in

a bulimic patient.[30] Laxative withdrawal is best managed by anticipatory counseling, including setting the expectation that constipation and possibly rebound edema may occur but typically resolve within 10 days to 3 weeks. Patients should also be advised that regular exercise, adequate fluid intake, the addition of such bulk-forming agents as fiber or psyllium to the diet, and in some cases, an osmotic laxative, such as lactulose, will be necessary. Stool softeners are of little clinical value.[31]

Patients' misconceptions about stimulant laxatives should be addressed. Moreover, they should be told that laxatives are an ineffective means of weight loss because their site of action is the large intestine, where they cause temporary fluid loss without concomitant caloric losses. Relapse to stimulant laxative abuse will worsen the dependency on them and increase the risk of more refractory constipation. Steatorrhea, protein-losing gastroenteropathy, and gastrointestinal bleeding have also been reported as a result of laxative abuse.

Cardiovascular complications

Cardiovascular complications occasionally result from the purging behaviors used by bulimics. Excessive purging of any type may cause volume depletion, which can cause dizziness, hypotension, or even syncope. Purging may also cause hypokalemia, which can lead to arrhythmias. The repeated use of ipecac to induce vomiting can cause an irreversible and potentially fatal cardiomyopathy. Ipecac is usually sold in bottles containing 30 ml of syrup of ipecac, the equivalent of 21 mg of emetine base, which is the toxic component. An estimated total dose of 1.25 g of emetine base may be fatal,[32] and lesser doses may cause skeletal and cardiac muscle myopathy. The drug is excreted slowly, so accumulation over time is a major concern. Mitral valve prolapse may be more prevalent among bulimic women and usually resolves with weight gain. On occasion, these patients may complain of palpitations or even chest pain as a result thereof and, similarly, if ephedrine-type diet supplements are abused.

Endocrine effects

There appears to be an increased prevalence of bulimia in patients with insulin-dependent diabetes.[33] Such patients may deliberately omit or reduce their insulin injections to induce diuresis and weight loss. In this regard, some of the behaviors that are important in dietary management of diabetes may set the stage for an eating disorder. Specifically, traditional dietary regimens that limit sweets teach patients to avoid certain foods and may thus promote abnormal eating habits. Although the causal relationship between bulimia and diabetes is debated, the detrimental effects of this combination are clear. In diabetic patients with bulimia, end-organ damage develops at a younger age than in age-matched nonbulimic diabetic patients.[34] Glycosylated hemoglobin values are elevated in many diabetic patients with bulimia because of the metabolic effects of binge eating, noncompliance with dietary regimens, and deliberate omission of insulin in order to produce an osmotic diuresis and weight loss.

In contrast to anorexia nervosa, other endocrine manifestations are not prominent in bulimia.[35] Compared with the general population, persons with bulimia nervosa appear to have a higher prevalence of nonsuppression on the dexamethasone suppression test, ranging between 20% and 67% in different studies.[18] This lack of suppression remains unexplained. Oligomenorrhea may occur in bulimia nervosa during episodes of active bulimia, but amenorrhea is infrequent.[14] This is in marked contrast to anorexia nervosa, where amenorrhea is a universal finding. The future ability of bulimic patients to conceive does not appear to be impaired.[36] Of note, although many women with bulimia will have reduced symptomatology during pregnancy, this is often followed by an exacerbation of symptoms after delivery.[37]

Similarly, in contrast to anorexia, wherein the prevalence of osteoporosis is very high,[38] bone density is generally normal in bulimic patients unless there has been a prior history of anorexia nervosa and marked weight loss. In this instance, bone densitometry would be warranted. Russell's sign, that is, erosions covering the dorsum of the hands, is a result of using one's finger to self-induce vomiting.[39]

SITE OF TREATMENT

Most medical complications of bulimia can be treated in the outpatient setting by a primary care doctor or provider with expertise in dealing with

these patients. Primary care providers should use a multidisciplinary approach when managing these patients, especially the more severe cases. The team should include a psychologist / psychiatrist and a nutritionist. Patients with severe bulimia nervosa may benefit from a referral to a center specializing in eating disorders. The main role of the primary care provider is to screen for and diagnose the disorder and then manage any medical complications that arise while the patient is engaged in therapy.

Factors that suggest a need for hospitalization include severe depression, marked dehydration and disabling symptoms, purging that is rapidly worsening and has proved refractory to outpatient treatment, severe hypokalemia (plasma potassium level <2.0–3.0 mmol / L), and major orthostatic changes in blood pressure (>30 mm Hg) and pulse (>30 beats per minute). Data are lacking on how these factors affect the ultimate outcome.[40]

SUMMARY

Bulimia nervosa is an increasingly common disorder among young women, especially in industrialized countries. Not only can it have adverse physical effects, but it can also wreak havoc on the emotional and social lives of these patients. The physical signs of this disorder can be subtle and easily overlooked unless specifically sought. Likewise, normal laboratory abnormalities are unreliable for excluding the disorder. The art of medicine comes into play in the evaluation and treatment of persons with bulimia nervosa. Adopting an accepting rather than a judgmental attitude will aid the clinician in establishing trust with the patient, eliciting the historical details necessary to make the diagnosis, and offering the necessary medical treatment to negate the litany of medical complications that can occur with bulimia.

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