Medical complications of bulimia nervosa

ABSTRACT

Bulimia nervosa is a psychiatric disorder with many different medical sequelae. This article reviews the principal medical complications associated with bulimia nervosa, and emphasizes the importance of a timely approach to diagnosis and management.

Bulimia nervosa is characterized by bingeing (consuming an inappropriately large amount of food in a discrete time period), followed by purging (attempts to remove food from the body by self-induced vomiting, or abuse of laxatives and diuretics, or increasing caloric utilization through exercise). The term bulimia nervosa was first used in 1979 (Russell, 1979). Bulimia nervosa most commonly affects young women with a prevalence of approximately 2% (Hay and Claudino, 2012), more than twice the prevalence of anorexia nervosa. The most frequently used purging behaviour is self-induced vomiting, followed by laxative and diuretic abuse (Dalle Grave et al, 2009). Up to 50% of patients with bulimia nervosa may consistently revert to multiple purging behaviours (Edler et al, 2007).

Like individuals with anorexia nervosa, individuals with bulimia nervosa are excessively concerned about body shape and weight. Psychological and environmental factors interact with and influence the expression of genetic risk to cause eating pathology. Individuals with bulimia nervosa restrict their intake (as a result of underlying anxiety and the need to be in control), but then give in to their urges, and engage in bingeing and then purging (Frank, 2016). Mood disorders are often comorbid with bulimia nervosa. Substance use disorders are also more common in patients with bulimia nervosa than in patients with anorexia nervosa.

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Bulimia nervosa is a serious psychiatric illness with substantial morbidity and mortality. Much of the reported mortality rate is attributable to the medical complications inherent to the illness, which will be discussed below (Brown and Mehler, 2013). These patients also have an increased mortality from suicide. Patients who cross over from restricting types of anorexia nervosa to either anorexia nervosa binge–purge subtype or bulimia nervosa are at particularly high risk of attempting suicide (Pisetsky et al, 2013).

Psychiatric treatment

Best care practices for bulimia nervosa include nutritional rehabilitation, psychotherapy, treatment of medical complications, and psychopharmacological management of symptoms and psychiatric comorbidities. Patients are typically treated in outpatient or partial hospital programmes, but hospitalization may be necessary for suicidal ideation or behaviour, or uncontrolled purging with substantial electrolyte disorders. A multidisciplinary approach is ideal, wherein specialists collaborate to support the patient in working towards individualized goals (National Institute for Health and Care Excellence, 2017).

Treatment goals for bulimia nervosa include:

1. Cessation of restricting and binge eating behaviour
2. Interrupting compensatory behaviour (e.g. vomiting, compensatory exercise, misuse of insulin, laxatives or diuretics)
3. Remission of comorbid psychopathology (e.g. depression, bipolar disorder, substance abuse, attention deficit hyperactivity disorder)
4. Treatment of associated medical conditions (e.g. electrolyte disturbance, gastrointestinal problems, dental issues).

Nutritional rehabilitation aims to eliminate bingeing and purging episodes and stop food restriction by implementing a planned and consistent diet. Patients learn to interpret and tolerate the normal physiological experiences of digestion. Cognitive behavioural therapy, 'enhanced' for eating disorders, which incorporates nutritional counselling, typically consists of up to 20 sessions over 20 weeks and has the most robust published support for treatment of bulimia nervosa (Serpell et al, 2013; National Institute for Health and Care Excellence, 2017). Children and adolescents with bulimia nervosa usually benefit from a specialized family therapy approach. This typically consists of 18–20 sessions over 6 months, which encourages the family to help their loved one recover (Le Grange et al, 2015).

Medication is most effective when combined with psychotherapy and is not generally offered as a singular treatment intervention for bulimia nervosa (Aigner et al, 2011; National Institute for Health and Care Excellence, 2017). Patients who have not responded adequately to psychotherapy, or those with comorbid psychiatric or medical disorders, and those with chronic or intractable illness, are especially likely to benefit from the use of psychiatric medicines.

Even when no symptoms of depression are identified, antidepressant medications may be helpful in reducing the frequency of binge eating episodes and purging behaviours in adults with bulimia nervosa. Antidepressant medications are the most rigorously studied class of medication for these patients. To optimize efficacy and tolerability, first-line medication management usually begins with the selective serotonin re-uptake inhibitor fluoxetine at doses higher than are generally used to treat depression or anxiety (Hay and Claudino, 2012).

Medical complications of bulimia nervosa

Electrolyte abnormalities

All purging behaviours if undertaken excessively will cause acid–base and electrolyte abnormalities. Self-induced vomiting typically leads to the most severe electrolyte and acid–base derangements of all the purging behaviours. Hypokalaemia and a chloride-responsive metabolic alkalosis are the hallmarks of this purging behaviour. Gastric fluid has a high concentration of hydrogen chloride; loss of this fluid through emesis therefore directly leads to the generation of a hypochloraemic metabolic alkalosis. This acid–base state would normally be transient; however, when vomiting is habitual and leads to intravascular volume depletion, several compensatory actions in the kidney act to maintain a state of alkalosis, which is often severe. First, in response to volume depletion, the kidney enhances proximal reabsorption of sodium bicarbonate, thereby promoting alkalosis. Second, and more importantly, volume depletion stimulates the renin–angiotensin–aldosterone system in an effort to maintain effective arterial blood volume and prevent fainting. Aldosterone promotes reabsorption of sodium bicarbonate in the distal nephron, while concurrently promoting excretion of potassium in the urine. Indeed, of all the different modes of purging behaviours in which patients with bulimia nervosa engage, the most severe degrees of metabolic alkalosis develop in self-induced vomiting. A serum bicarbonate level of 38 mEq/litre or greater in a patient with bulimia is almost always attributable to this method of purging (Mehler and Walsh, 2016).

Laxative abuse is also associated with a number of electrolyte and acid–base abnormalities. Stool fluid is relatively high in potassium, therefore laxative abuse with recurrent diarrhoea can directly lead to potassium losses and consequent hypokalaemia. Unlike patients who purge through vomiting or diuretic abuse, patients who develop hypokalaemia from laxative abuse, via excessive stool potassium losses, will have a urinary potassium concentration that is low (Brown and Mehler, 2012). Laxative abuse may lead to either metabolic acidosis or metabolic alkalosis. Early on, the acute loss of large volumes of diarrhoea, which has relatively high bicarbonate and relatively low chloride concentrations, can lead to net loss of bicarbonate. Over time, however, chronic laxative abuse leads to volume depletion and hypovolaemia. As discussed above, chronic volume depletion results in upregulation of the renin–angiotensin–aldosterone system. Thus, chronic ongoing laxative abuse may result in a metabolic alkalosis via the actions of aldosterone (Gennari and Weise, 2008). However, the magnitude of the metabolic alkalosis is typically less than that of patients who purge through vomiting (Gennari and Weise, 2008; Mehler and Walsh, 2016).

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Electrolyte and acid–base abnormalities seen in diuretic abuse mimic those of self-induced vomiting, although the degree of abnormality is typically less severe. Over-the-counter diuretics are typically too weak to induce severe electrolyte derangements. However, patients who abuse prescription thiazide and loop diuretics often develop both hypokalaemia and a metabolic alkalosis. When diuretic abuse leads to intravascular volume depletion, the presence of aldosterone will lead to net excretion of hydrogen and potassium ions in the urine, causing chronic hypokalaemia and a metabolic alkalosis (Bartoli et al, 2017).

Lastly, it should also be noted that any of the aforementioned purging behaviours can lead to abnormalities in salt and water balance. The most clinically important abnormality is hypovolaemic hyponatraemia resulting from intravascular volume depletion with relative excess of body water. Abuse of thiazide diuretics carries particular risk of causing dangerous hyponatraemia, as thiazide diuretics do not impair the kidney's ability to concentrate urine and can therefore cause imbalanced loss of sodium with relative water retention (Bartoli et al, 2017).

Medical complications of self-induced vomiting

Multiple oral complications, including cheilosis, perimylolysis and soft tissue trauma, may result from chronic self-induced vomiting. Xerostomia, or dry mouth, is a common symptom among patients with eating disorders, which may be partially attributable to dehydration and medications such as antidepressants. Patients with eating disorders report significantly increased frequency of dry or cracked lips compared to controls (Johansson et al, 2012). Angular cheilosis is a form of stomatitis (inflammation of the mucus membranes of the mouth) seen in patients

with eating disorders, and is characterized by pallor and maceration of the mucosal lining at the corners of the mouth. When examining the oropharynx of patients with bulimia, ulcerations on the palate and oropharynx are often seen as a result of the repeated use of a finger or another object to induce vomiting.

Perimylolysis, or erosion of the teeth, has been reported to occur in up to 64% of people who purge by means of self-induced vomiting. Chronic contact with acidic gastric contents and proteolytic enzymes in the vomitus leads to loss of superficial tooth enamel and the dentine that lies underneath the enamel. Symptoms of severe perimylolysis include tooth sensitivity to hot and cold food as a result of exposed dentine, along with chipping of the edges of the teeth. The dental areas affected first are primarily the lingual surfaces of the upper anterior teeth, followed by the palatal and posterior occlusal surfaces of the maxillary teeth. Early perimylolysis is characterized by a smooth, glossy appearance. As erosion continues, the teeth lose height and enamel surfaces become dull with jagged incisal edges.

Dental care instructions are an important part of the treatment plan for patients with bulimia. The most effective means of preventing and treating dental complications from bulimia is to stop the self-induced vomiting. Apart from that, the practice of rinsing one's mouth with water or a slightly basic solution containing baking soda or sodium fluoride after vomiting may help to neutralize acid and protect tooth surfaces (Steinberg, 2014). The optimal tooth brushing practice for patients with bulimia remains controversial as there is concern that the abrasive mechanical action of brushing after vomiting may accelerate enamel erosion. Most clinicians recommend gentle brushing because of the lack of evidence that tooth brushing, per se, contributes to erosion.

Parotid hypertrophy, or swelling of the salivary glands, occurs in up to two-thirds of patients with bulimia, and is directly related to the frequency of vomiting. The swelling is usually painless, bilateral, and readily apparent on physical examination. Occasionally, the parotid and, rarely, submandibular salivary glands enlarge to two to five times their normal size. This swelling (sialadenosis) begins 2–3 days after cessation of chronic self-induced vomiting, which can be disconcerting for the patient with bulimia who has finally decided to cease purging (Gaudiani and Mehler, 2016). The exact pathophysiology of parotid hypertrophy is undetermined.

Treatment of sialadenosis depends on the severity of the swelling and the patient's concern over his or her cosmetic appearance. Abstinence from vomiting alone will lead to the resolution of swelling in most cases. In addition, hot compresses, non-steroidal anti-inflammatory medication (such as ibuprofen) and sialogogues (such as sour sweets) can aid in the resolution of swelling or, if started at the time of cessation of vomiting, may prevent its development.

Gastrointestinal complications of self-induced vomiting

Voice and throat complaints are commonplace among patients with a history of self-induced vomiting. Laryngopharyngeal reflux describes the syndrome in which gastric acid contents affect and injure the larynx and pharynx. Hoarseness, sore throat, dry cough, chronic throat clearing and difficulty swallowing are all symptoms of laryngopharyngeal reflux. Physical findings of laryngopharyngeal reflux include post-cricoid oedema, thick mucus over the larynx, telangiectasias and polypoid changes (Rothstein, 1998).

Gastro-oesophageal reflux disease is a very common complication of frequent vomiting. Patients with bulimia may be more prone to this condition because of laxity of the lower oesophageal sphincter from recurrent vomiting (Mehler, 2011). Symptomatic patients with gastro-oesophageal reflux disease often complain of heartburn or odynophagia and these symptoms are commonly reported among patients with bulimia. Complications of gastro-oesophageal reflux disease include oesophagitis, oesophageal ulceration and Barrett's oesophagus, which is the replacement of normal squamous epithelium with columnar epithelium as a result of chronic gastric acid exposure. In some cases, Barrett's oesophagus may progress to oesophageal adenocarcinoma. First-line treatment for gastro-oesophageal reflux disease involves proton-pump inhibitors. Patients with bulimia and severe gastro-oesophageal reflux disease symptoms should perhaps undergo endoscopy of the upper gastrointestinal tract in order to evaluate for erosions or for evidence of Barrett's oesophagus.

Vomiting also has other deleterious effects on the oesophagus. In cases of haematemesis, a Mallory–Weiss tear may be to blame, which is normally a self-limited bleed from a submucosal artery following laceration of the mucosa at the lower oesophagus or proximal stomach associated with retching. Typically, bleeding is minimal. Rarely, Boerhaave's syndrome (perforation of the oesophagus) may occur, leading to extrusion of gastric contents into the mediastinum, creating a state of mediastinitis. Boerhaave's syndrome has a very high mortality rate if not diagnosed early and surgically repaired.

Medical complications of laxative abuse

Stimulant laxatives are widely abused by patients with bulimia. Laxative abuse in these patients has been found to range from 18% up to 75%, with stimulant laxatives being the type most frequently abused (Roerig et al, 2010). The stimulant laxatives can be divided into two classes: diphenylmethanes (i.e. bisacodyl) and anthraquinones (with senna being the most popular in this class). These medications likely work by causing low-grade inflammation in the colon, causing accumulation of water and electrolytes as well as directly stimulating intestinal motility via the colonic nerve plexi. Laxative abuse should be suspected in patients with bulimia with

diarrhoea of unclear aetiology and a low stool osmotic gap. The anthraquinones can produce a dark discolouration of the bowel called melanosis coli. This is the result of colonic surface epithelial cells undergoing apoptosis followed by breakdown of these apoptotic cells into lipofuscin pigments within the lysosomes of macrophages. It is currently believed that melanosis coli does not confer any negative health consequences and will usually reverse with discontinuation of the laxatives.

Patients with bulimia will frequently increase the dosing of laxatives under the false presumption that this will increase weight loss, while further increasing the risk of additional medical complications. Indeed, they may increase their intake up to 50–100 laxatives daily to achieve the desired effect. However, studies suggest that this has only a mild effect on causing nutrient malabsorption and weight loss because the site of action of laxatives is in the distal colon, well past the small bowel where most caloric absorption occurs. The excessive defecation and/or straining that may accompany the abuse of this amount of laxatives can also lead to rectal bleeding from haemorrhoids and anal fissures and also lead to the development of rectal prolapse and diverticulosis.

One frequently debated medical complication of stimulant laxatives is the entity known as the cathartic colon syndrome. This is diagnosed via barium enema showing dilated loops of bowel with loss of haustral markings. With this condition, the colon is believed to become incapable of peristalsis and is relegated to being like an inert tube. Early studies suggested that myenteric plexus damage caused this condition, with longer abuse and higher amounts used being associated with greater degenerative changes (Riemann et al, 1980). Other studies found no such histological changes; however, even without myenteric plexus damage, studies have still shown evidence of impaired colonic emptying (Wald, 2003). Indeed, one study found decreased oxidative metabolism in the intestinal smooth muscle and myenteric neurons, offering another possible explanation for the previous discrepancies (Nadal et al, 2003). Adding to the debate over this condition, the disease largely disappeared from the literature for some time making some believe the condition was solely associated with certain formulations of senna that are no longer produced. However, a later study found radiological changes consistent with cathartic colon in those abusing stimulant laxatives that were not seen in the control group (Joo et al, 1998). Thus it seems that cathartic colon is a transient condition if the abuse of the laxatives can be stopped (Mehler and Rylander, 2015).

Pseudo-Bartter's syndrome

Patients with bulimia, who chronically abuse laxatives and diuretics or engage in daily vomiting, fall into the category of pseudo-Bartter's syndrome as they exhibit biochemical abnormalities of hypokalaemia, hypochloraemia and metabolic alkalosis. In pseudo-Bartter's syndrome, the renin–angiotensin–aldosterone system is the key mechanism responsible for maintaining plasma volume. It is over-activated in the eating disorder population with bulimia nervosa, as well as in those with excessive purging behaviours across the various other forms of eating disorders, who engage in chronic and frequent daily vomiting, laxative or diuretic abuse.

In response to a decrease in plasma circulating volume, caused by dehydration from any form of purging, the juxtaglomerular apparatus responds by secreting renin which results in an increase in production of angiotensin I and II. Angiotensin II stimulates the zona glomerulosa in the adrenal gland to produce and secrete increased amounts of aldosterone (Schrier et al, 2010). Aldosterone acts on the renal collecting duct to reabsorb sodium in exchange for potassium and hydrogen. Increased hydrogen excretion leads to an increase in production and reabsorption of bicarbonate which leads to a worsening metabolic alkalosis. Thus, the chronic state of hyperaldosteronism, initially achieved as a defence mechanism to treat dehydration and prevent fainting, leads to worsening hypokalaemia and metabolic alkalosis. Hypokalaemia and hypovolaemia in turn stimulate prostaglandin production, especially PGE2, which stimulate natriuresis and thus more renin and aldosterone secretion (Dahabreh and Najada, 2013).

Given the above-described pathophysiological changes that are activated as a result of the chronic hypovolaemic state of bulimia nervosa, pseudo-Bartter's syndrome becomes a vicious cycle of a continued state of hyperaldosterone, leading to pseudo-Bartter's syndrome's predictable chemical laboratory abnormalities of hypokalaemia and metabolic alkalosis and a propensity toward troublesome oedema formation when the purging behaviours are abruptly ceased.

Thus, when these patients decide to cease purging, the adrenal glands continue to secrete high levels of aldosterone for at least 1–3 more weeks, until the production of aldosterone is reduced to normal levels when the state of continuous dehydration recedes. The body is perhaps trying to prevent dehydration and has not yet reset its exuberant secretion of aldosterone in response to the chronic purging behaviours which were the norm. Therefore, during that period, the fluid balance in the body changes to a positive one because there is no longer the abnormal egress of fluids out of the body via the different modes of purging, but high levels of aldosterone persist as the body's homeostatic mechanism attempts to conserve fluid. As a result, there is a risk of severe oedema formation and fluid retention which can cause marked weight gain and distressing body image issues during the immediate time period after purging has stopped.

Similarly, if during this period this patient needs to go to an emergency room for intravenous saline to treat symptoms which arose from being dehydrated, or from the low potassium levels, he/she can, within just a few hours, gain large amounts of weight and oedema, if the intravenous fluids are administered in the rapid manner that emergency department physicians use to treat typical

KEY POINTS

- Patients with bulimia nervosa have a litany of medical complications as a result of the frequency and the mode of purging behaviours.
- Most medical complications are eminently treatable with timely diagnosis and management.
- There are a few critical electrolyte abnormalities which point to the need for inpatient hospitalization.
- Pseudo-Bartter's syndrome is one of the most vexing and complex complications which are associated with purging behaviours and require specialty eating disorder expertise to expertly manage.

dehydration. Again, this is the result of the markedly elevated state of serum aldosterone levels (Trent et al, 2013) and its action to reabsorb renal sodium. Indeed, it is the very distressing memories of these misguided treatments which may give pause to the patient with bulimia nervosa and prevent him/her from seeking treatment despite feeling ill from hypokalaemia and volume depletion. This delay may contribute to the increased standardized mortality rate in people with bulimia nervosa of nearly twice that of age-matched controls (Smink et al, 2012).

Treatment of pseudo-Bartter's syndrome and its oedema and electrolyte abnormalities in the eating disorder population requires an understanding of the aforementioned underlying pathophysiological changes that ensue. In addition, a key medical treatment strategy in bulimia nervosa patients who abruptly cease purging behaviours is, paradoxically, the use of the diuretic, spironolactone. This mild diuretic competitively inhibits aldosterone, the key cause of the oedema. Because of the blocking action on aldosterone, these diuretics can actually cause hyperkalaemia and need to be used with caution in those patients with decreased renal function (Lainscak et al, 2015).

The benefits of spironolactone in the treatment of pseudo-Bartter's syndrome are actually three-fold given its mechanism of action. First of all, as mentioned above, spironolactone competitively inhibits the action of aldosterone, the key hormone in the initiation and perpetuation of this condition. The state of hyperaldosteronism (from hyperstimulation of the renin–angiotensin–aldosterone system) in pseudo-Bartter's syndrome that results from chronic volume depletion, may take up to 2–3 weeks to be completely downregulated to the normal blood level once the stimulus is removed. The second benefit of spironolactone is that it is a mild diuretic and as mentioned above can alleviate the oedema that ensues upon cessation of purging behaviours. Lastly, spironolactone is a potassium-sparing diuretic and thus helps to correct the hypokalaemia that is so common in this syndrome when these patients initially present (Mascolo et al, 2011). The dosage that is used is 25–200 mg, given in a single morning dose, with the usual practice being to start at the lower range and increase if there is excessive weight gain or worsening oedema which has accompanied efforts to cease purging.

Conclusions

The purging behaviours of bulimia nervosa are associated with a litany of inherent medical complications. In addition, when these behaviours cease, there is often the development of predictable and frustrating sequelae which may impair the patient's ability to sustain his/her 'detox'. This article has described both the complex pathophysiology behind the cascade of troubling events which occur with the abrupt cessation of purging, known as pseudo-Bartter's syndrome and other complications with bulimia nervosa, and outlined which medical treatment strategies are used to help achieve successful extrication from the purging behaviours of bulimia nervosa. **BJHM**

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