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Gastrointestinal Aspects of Bulimia Nervosa

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1. Introduction

Eating disorders are an important cause of physical and psychosocial morbidity in adolescent girls, young adult women, and to lesser extent in men. In the diagnostic and statistical manual of mental disorders fourth edition (DSM-IV), three broad categories of eating disorders are delineated: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified (American Psychiatric Association, 1994). The international classification of diseases tenth revision (ICD-10) also reported three categories of eating disorders: anorexia nervosa, bulimia nervosa, and atypical eating disorder (World Health Organization, 1992). In detail, anorexia nervosa is characterised by extremely low bodyweight and a fear of its increase; bulimia nervosa comprises repeated binge eating, followed by behaviour to counteract it. The category of eating disorder not otherwise specified encompasses variants of these disorders, but with sub-threshold symptoms (e.g., menstruation still present despite clinically significant weight loss, purging without objective binging) (Treasure et al., 2010).

The main feature that distinguishes bulimia nervosa from anorexia nervosa is that attempts to restrict food intake are punctuated by repeated binges (episodes of eating during which there is an aversive sense of loss of control and an unusually large amount of food is eaten). The amount consumed in these binges varies, but is typically between 4.2 MJ (1000 kcals) and 8.4 MJ (2000 kcals) (Fairburn & Harrison, 2003). In order to prevent weight gain, self-induced vomiting and excessive exercise, as well as the misuse of laxatives, diuretics, thyroxine, amphetamine or other medication, may occur. The combination of under-eating and binge eating results in bodyweight being generally unremarkable, providing the other obvious difference from anorexia nervosa. There is some controversy concerning whether those who binge eat but do not purge should be included within this diagnostic category. The ICD-10 criteria stress the importance of purging behaviour on the grounds that vomiting and laxative misuse are considered pathological behaviours in our society in comparison to dieting and exercise. The DSM-IV criteria agree about the importance of compensatory behaviour but distinguish between the purging type of bulimia nervosa in which the person regularly engages in self-induced vomiting or the misuse of laxatives, diuretics or enemas, from the non-purging type in which other inappropriate compensatory behaviours such as fasting or excessive exercise occur but not vomiting or laxative misuse (National Collaborating Centre for Mental Health, 2004).

It is noteworthy that bulimia may be suspected also in patients undergoing bariatric surgery. Indeed, this kind of surgery, also named weight loss surgery, includes a variety of procedures performed on people who are obese. Weight loss is achieved by reducing the size of the stomach with an implanted medical device (gastric banding) or through removal of a portion of the stomach (sleeve gastrectomy or biliopancreatic diversion with duodenal switch) or by resecting and re-routing the small intestines to a small stomach pouch (gastric bypass surgery). When determining eligibility for bariatric surgery, psychiatric screening is critical.

Bulimia nervosa may lead to significant morbidity and mortality. The diagnosis depends on obtaining a history supported, as appropriate, by the corroborative account of a parent or relative. This will require an empathic, supportive, non-judgemental interview style in which the person is enabled to reveal the extent of his or her symptoms and behaviours. Although those with bulimia nervosa generally have fewer serious physical complications than those with anorexia nervosa, they commonly report more physical complaints when first seen (National Collaborating Centre for Mental Health, 2004). The gastrointestinal tract is the site of most acute and chronic medical complications of the disease (Table 1). Identification of any of the gastrointestinal aspects may aid in establishing an early diagnosis, which has been shown to increase the likelihood of recovery.

Effects of bulimia
Dental erosion
Dental caries
Oral dryness
Parotid and salivary glands enlargement (raised serum amylase)
Dysphagia
Esophagitis/oesophageal ulcers
Vomiting
Hematemesis (rare)
Increased gastric capacity
Delayed gastric empting
Gastric rupture (rare, but high letality)
Bloating
Abdominal pain
Diarrhoea
Constipation
Volvulus (rare)
Rectal prolapse (rare)

Table 1. Common adverse effects of bulimia nervosa on the gastro-intestinal tract.

Many changes in gastrointestinal physiology are associated with bulimia nervosa. Some of these are particularly interesting because they may favour the maintenance or even an increase in eating disorder symptoms and so militate against recovery. Some gastrointestinal complications are due to unrelenting abuse of the alimentary canal occurring over the course of years. Others occur in an acute form in a severely ill patient and may require urgent attention.

In this chapter we provide a systematic review of the existing literature on the gastrointestinal involvement in patients affected by bulimia nervosa. We searched the Medline databases for articles on bulimia nervosa published since 1980. The key words used were eating disorders, bulimia nervosa, bulimia, binge eating, gastro-intestinal tract, oesophagus, stomach, oral cavity, and bowels. Only articles written in English were reviewed.

2. Oral cavity

Oral pathology plays a crucial role in the diagnosis of bulimia, often providing the vital link between the patient and medical intervention. The acidic contents of the regurgitated in patients with bulimia nervosa causes erosion of the deciduous and permanent dentition. The erosion particularly affects the posterior teeth and the palatal aspects of the upper anterior teeth; both the deciduous and the permanent dentitions can be affected. Erosions generally occur after six months of vomiting behaviour, and severity of erosions increases with time. The erosion of enamel of the teeth exposes the underlying dentin, producing acute contact and thermal hypersensitivity (pain on eating hot or cold meals).

Dental caries are a further oral feature of bulimia nervosa, and are related either to the cariogenicity of the diet or to the lowering pH sustained by vomiting. To protect teeth from the effects of chronic vomiting, regular dental review is highly recommended; patients should be given appropriate advice on dental hygiene, which should include avoid brushing teeth after vomiting as it may increase tooth damage, mouth rinsing after vomiting with water and sodium bicarbonate (or other non-acid mouth wash) in order to neutralise the acid environment, use of fluoride mouth rinses and toothpastes which may be helpful for desensitisation, reduce intake of acidic foods (fruit, fruit juice, carbonated drinks, pickled products, yoghurt and some alcoholic drinks), finishing meals with alkaline foods (e.g. milk or cheese), avoiding habits such as prolonged sipping, holding acidic beverages in the mouth and "frothing" prior to swallowing, chewing sugar-free gum after meals to stimulate salivary flow (although this may cause increased gastric secretions).

Painless bilateral enlargement of salivary glands (especially the parotid gland, but occasionally the submandibular salivary glands) are a frequent finding in bulimia nervosa, and are a useful indicator in diagnosing and monitoring the disease, avoiding unnecessary tests. Possible explanations for enlarged parotid glands include nutritional deficiencies, excessive starch consumption, re-feeding after starvation, and functional hypertrophy associated with repeated episodes of binge eating. Therefore, increase amylase secretion is characteristic of bulimia nervosa, and it may be useful in monitoring the degree of compliance to therapeutic programs (Anderson et al., 1997).

Oral dryness may also be a sign of bulimia nervosa; although no differences between patients with bulimia nervosa and controls has been found in the salivary flow rates and fluid secretory capacity for parotid and submandibular glands, oral dryness may be related to surface mucosal alteration and to a change in ability to perceive moisture adequately (Anderson et al., 1997).

3. Oesophagus

Pharyngeal and velar gag reflex may be impaired in bulimia nervosa. This was believed to be a learned response and a form of desensitization from years of gastric purging. Loss of the gag reflex facilitates self-induced vomiting by making it less aversive. Abnormal oesophageal motility has also been found frequently in patients with bulimia. Initially, patients need to provoke the gag reflex using their fingers or another object, whereas in advanced stages, physical means may not be necessary. Reflux of gastric contents into the lower oesophagus may cause relaxation of the lower oesophageal sphincter; loss of sphincter control may be sufficient to induce vomiting.

Vomiting is the main symptom suggestive of bulimia. It may be induced by medications such as ipecac, hypertonic saline, or other emetogenic substances. It may be a result of self-induced gagging, as mentioned above; therefore, calluses on the back of the hand may be found (Russell's sign), suggesting the use of the hand to stimulate the gag reflex and induce vomiting. It may also be promoted by forceful abdominal muscle contraction during spontaneous lower oesophageal sphincter relaxations associated with belching. This characteristic is sometimes useful in manometrically discriminating the patient with bulimia from the patient with gastro-oesophageal reflux. These patients often vomit surreptitiously. Vomiting and subsequent electrolyte disturbances may lead to cardiac and metabolic effects out side the alimentary canal, such as dehydration, hypokalaemia and cardiac dysrhythmias and hypochloremic alkalosis. When electrolyte disturbance is detected, it is usually sufficient to focus on eliminating the behaviour responsible. In the small proportion of cases where supplementation is required to restore the patient's electrolyte balance, oral rather than intravenous administration is recommended, unless there are problems with gastrointestinal absorption (National Collaborating Centre for Mental Health, 2004).

Dysphagia, esophagitis, oesophageal erosions, ulcers and bleeding occur frequently in patients who practise self-induced vomiting. However, it has been suggested that psychiatric diagnosis is often mistaken and that many patients thought to have eating disorders may well be suffering primarily from oesophageal disorders such as achalasia. Therefore, bulimia nervosa should be considered in the diagnostic work-up of patients referred for suspect gastro-oesophageal reflux disease, but a 24-h oesophageal multichannel intraluminal impedance, endoscopy and/or manometry should be always performed to excluded an organic cause of symptoms.

Hematemesis may result from a mucosal tear of the lower end of the oesophagus during vomiting (Mallory-Weiss Syndrome) or from haemorrhagic esophagitis due to acid reflux. The initial evaluation of any patient with upper gastrointestinal bleeding must include a brief history followed by a rapid assessment of the physical condition, with particular attention to the vital signs and the patient's level of consciousness. If the bleeding is severe, therapy may need to begin before the location of the bleeding can be ascertained. Significant gastrointestinal bleeding will be initially manifest by tachycardia, whereas hypotension occurs later, an ominous signal of impending cardiovascular collapse. Immediate therapy is aimed at correction of volume loss and anaemia, which should include aggressive fluid and blood resuscitation. If the patient remains unstable after receiving a blood transfusion of approximately 85 mL/kg or greater, emergency exploratory surgery is indicated. Surgical consultation is mandatory in any case of severe upper gastrointestinal bleeding.

Small amounts of red blood in vomitus may be due to the fingernails injuring the pharynx and the history and examination will generally clarify this.

4. Stomach

Gastric capacity increases in bulimia nervosa, presumably as a result of repeated large volume binge-eating episodes. This may be associated with an absence of satiety signals until a large amount of food has been ingested. A decrease in gastric emptying rate is also been found in bulimic patients, as well as lower amplitude of antral contractions.
Acute gastric dilation has rarely been reported in bulimia nervosa. It is usually accompanied by pain and discomfort. Stomach (or oesophageal) perforation is a complication, which has a high mortality, and occurs in two situations. Firstly, it may occur in a patient with unrecognized acute gastric dilatation who continues to eat or binge. The thin gastric wall continues to dilate and eventually tears. The result may be an acute abdomen with sub-diaphragmatic air observed on an erect abdominal or chest X-ray. Alternatively, there may be a relatively silent oesophageal tear with air observed in the mediastinum on the chest X-ray. The condition is an acute surgical emergency requiring immediate laparotomy and repair. Secondly, it may occur in a patient, who may be of normal weight, who is unable to vomit following a binge-eating episode and suffers a gastric or oesophageal (Mallory-Weiss) tear. The cause appears to be strenuous attempts to vomit, which expose the stomach to extreme strain and rupture.

5. Bowels

Bulimic patients often abuse laxatives; indeed, patients believe that laxatives prevent absorption, having observed solid food appear in their stools, although it has been showed that laxatives have no detectable effect on the absorption of liquid nutrients. Stimulant laxatives are most frequently used, because they are fast-acting and reliable way to produce watery diarrhoea; therefore the effects of laxative abuse include diarrhoea, steatorrhea, and general malabsorption of nutrients. Irritable bowel syndrome type symptoms may be present. Colonic damage is mainly the result of prolonged abuse of laxatives, which have been shown to cause degeneration of the colonic autonomic nerve supply. The urgent presentations of colonic dysfunction are due to the weak, atonic cathartic colon. This can present as volvulus, prolapse of the rectum through the anus and intractable constipation. Any of these problems, if severe enough, can require surgery and sometimes colectomy. Mouth to caecum and total gut transit times are significantly prolonged, as is colonic transit time. This slowing, in addition to gastric delay, may also contribute to prolonged satiety by producing long-lasting feelings of general abdominal fullness.
Constipation is extremely common, mainly due to dehydration. Recommended treatment to avoid constipation is regular food intake, adequate fluids and exercise.
Laxative abuse carries also the acute complication of electrolyte and fluid disturbances and can be particularly dangerous in low weight individuals. Abrupt cessation of laxatives in those who are taking them regularly can result in reflex fluid and sodium retention, and consequent weight gain, and oedema. This can increase patient anxiety and reluctance to curtail the use of laxatives. To avoid this effect a gradual reduction in laxative use is advised (National Collaborating Centre for Mental Health, 2004).

6. Pancreas

Fasting and binge eating foods high in refined carbohydrates, especially if vomiting follows this, can lead to high levels of insulin release by the pancreas with large fluctuations in

blood sugar levels. This may disrupt the appetite control mechanisms and the utilisation and deposition of energy. Serotonin is implicated in appetite regulation (there may be a particular role in carbohydrate balance); disruptions in serotonin levels may be affected by the impact of insulin on its precursor, tryptophan, and in turn acute tryptophan depletion may lead to an increase in calorie intake and irritability in bulimia nervosa and may be related to decreased mood, increased rating in body image concern and subjective loss of control of eating in people who have recovered from bulimia nervosa (National Collaborating Centre for Mental Health, 2004).

7. Conclusions

Bulimia nervosa is a common health problem in young people, has been reported worldwide both in developed regions and emerging economies, and its prevalence is arising. It can lead to serious medical complications. However, studies from the US and continental Europe suggest that only a fraction of people with bulimia receive specialised treatment for their eating disorder. The alimentary canal is the front line for the eating disorder patient. Therefore, the expression of the disease in the gastrointestinal tract may have a critical role in early diagnosis and management of the disease. New treatment strategies are now available, and evidence-based management of this disorder is possible. A specific form of cognitive behaviour therapy is the most effective treatment, although few patients seem to receive it in practice.

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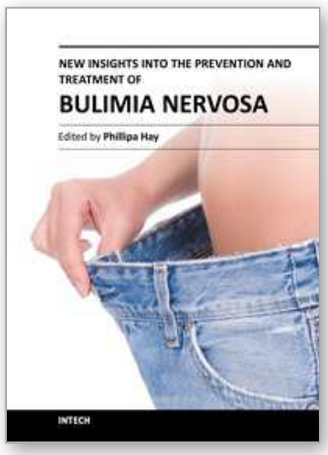

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Bulimia nervosa and eating disorders are common cause of distress and health related burden for young women and men. Despite major advances over the past three decades many patients come late to treatment and find that the therapy is incompletely addressed to the complex psychopathology and co-morbidities of the illness. The present book brings timely and contemporary understandings of bulimia nervosa to aid in current thinking regarding prevention and treatment. It will be read by therapists interested in enhancing their current approaches and those interested in earlier and more effective prevention and closing the gap between illness onset and accessing treatment. They will find practical guidance but also new ideas and ways of thinking about bulimia nervosa and the illness experience in this book.

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