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Gastrointestinal complications and refeeding guidelines in patients with anorexia nervosa

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Summary

Anorexia nervosa (AN) is the third most common disorder, after obesity and asthma, in the population of adolescents between 13–18 years of age. Food intake reduction is associated with whole body dysfunction, affecting its physical, psychological and social spheres. As a result of starvation, dysfunction develops in virtually all systems and organs. However, most frequently patients with AN complain of digestive symptoms, such as a feeling of fullness after meals, pain in the upper abdomen, dysphagia, nausea, bloating and constipation. They can have mild functional character, but may also reflect serious complications, including diseases requiring urgent surgical intervention. In addition, gastric complaints may hinder nutritional management of AN. Care of AN patients requires cooperation of many specialists in the field of psychiatry, psychology, pediatrics, internal medicine and nutrition. However, it is often difficult to organize such a team. Therefore, we decided to approach the issues of gastrointestinal symptoms and complications in the course of AN, and the rules of nutritional therapy.

Key words: anorexia nervosa, gastroenterological complications, refeeding syndrome

Introduction

Anorexia nervosa (AN) is a chronic syndrome involving deliberate quest to lose weight through a variety of behaviors such as limiting food intake, the use of low-calorie diet, excessive exercise, usage of laxatives, self-induced vomiting or following other methods of weight loss [1].

There are two types of AN: the restrictive form, in which food intake reduction and/or intense physical exercise predominate, and the bulimic-purging form associated with the occurrence of binge eating episodes and simultaneous purging behaviors (i.e., induced vomiting, use of diuretics or laxatives).

In highly developed countries, the incidence of AN is estimated at 0.3–2%, and the ratio in men and women ranges between 10:1 and 4: 1 [1]. It is the third disease in terms of incidence after obesity and asthma in young people between 13–18 years of age [2].

Gastrointestinal complications in AN

Food intake reduction is associated with impaired functioning of the whole body, concerning the physical, psychological and social spheres [1]. Starvation leads to the dysfunction of virtually all systems and organs, but usually AN patients complain of digestive ailments, such as postprandial fullness, pain in the epigastric region, dysphagia, nausea, bloating and constipation [1]. The symptoms can have mild functional character, but also display signs of serious complications, including disorders requiring urgent surgical intervention. Moreover, even mild gastric complaints may hinder the dietary treatment of anorexia nervosa, as patients, looking for an organic explanation of their low body weight, tend to use the symptoms as an argument to justify their aversion to eating.

It needs to be emphasized that, gastrointestinal tract symptoms can be the first physical signs of an eating disorder. In adult patients suffering from late-onset anorexia nervosa correct diagnosis is frequently preceded by many years of gastroenterological diagnostics and therapy and sometimes unnecessary surgical procedures [3].

Literature provides numerous data on the changes in the functioning of the digestive tract in the course of AN.

Changes in the mouth

Patients with AN frequently complain of dry mouth. They may experience erosion of the lining of the gums and palate, angular cheilitis, gingivitis and periodontitis [4]. These lesions are due to reduced resting and stimulated salivary flow, and reduction in salivary pH [4].

In the purging form of the illness, damage caused by self-induced vomiting appears in the oral mucosa, especially in the soft palate region [5]. In the course of anorexia nervosa, unilateral or bilateral painless swelling of the parotid glands may also occur [5, 6]. According to Gross et al. [6], enlarged salivary glands are found in 10–50% of cases, mainly in the purging form of AN [5]. The etiology of these abnormalities has not been fully explained. Hypertrophy of the parotid gland may result from repeated binge eating episodes, during which the glands are stimulated, and from persistent vomiting, in which gastric acid gets into the salivary ducts irritating their lining [5].

The action of gastric acid leads to irreversible dental enamel erosion [4, 5], characterized by demineralization of the outer layers and gradual involvement of deeper tissues of the tooth. At first, shallow, crater-like cavities, clearly demarcated from the

healthy enamel are formed. As the disease progresses the lesions become severe, and dentin and even dental pulp get exposed [4].

Gieorgijewska et al. [7] examined a group of 30 female patients, who were diagnosed with anorexia nervosa (AN) and bulimia nervosa (BN). Among the observed changes in the oral cavity in patients with AN the most common was angular cheilitis (71%), followed by post-traumatic lesions (35%) and inflammation of the tongue (43%). The authors also confirmed higher incidence of enamel erosion in regularly vomiting patients with purging AN [7].

Disorders of the upper gastrointestinal tract

The first reports on esophageal dysfunction in AN were published in 1986. Stacher et al. [8] examined 30 female patients aged 14–43 years, finding esophageal motility disorder in half of them. They found achalasia in 7 patients, distal dysphagia in 6 patients, gastroesophageal reflux in 1 patient, and diffuse esophageal spasm also in 1 patient [8].

Reports on patients with esophageal achalasia, who were initially misdiagnosed with anorexia nervosa have been published [9]. Literature data indicate a relatively high prevalence of motility disorders of the esophagus and stomach in the course of AN, which may be responsible for gastric symptoms reported by patients [10–13].

Benini et al. [11] performed manometry of the esophagus in 8 patients with purging AN and in 9 with the restrictive form, finding higher than normal, but still within the norm, resting pressure of the lower esophageal sphincter (LES) in patients with the restrictive type, which was stabilized after 22 weeks of treatment. The authors revealed no significant differences regarding the resting LES pressure or its relaxation disturbances in patients with purging AN as compared to the control group [11].

In our center, we studied esophageal function in AN girls using a manometry technique with a 4-channel probe flow [10]. We found incomplete relaxation of the upper esophageal sphincter during swallowing of the liquid in 40% of the patients and incomplete relaxation of the lower sphincter in 5 patients. None of the examined girls showed esophageal achalasia [10].

The rate of stomach emptying in patients with AN is much lower than in healthy subjects [12]. According to some authors, the stomach emptying delay occurs only after solid food intake [13], whereas other researchers have shown that it concerns both solid and liquid foods [12].

It has not been yet established whether gastric motility disorders in anorexia nervosa are transient – associated with emaciation, or permanent. According to Dubois [14], the abnormalities persist even after gaining proper weight. Benini et al. [15] suggest that they disappear when mental state of the patients, and not their BMI, improves. In contrast, other researchers have shown that the rate of stomach emptying in AN elevates significantly with increasing body weight [16].

Anorexia nervosa and intestinal malabsorption

The available literature provides data on the coexistence of anorexia nervosa and celiac disease (CD), although the exact pathomechanism of this phenomenon has not been elucidated yet [17, 18].

Ricca et al. [17] presented a case of two female patients suffering from AN and CD. The diagnosis of CD preceded the diagnosis of eating disorders in one patient, whereas the other patient was first diagnosed with AN. According to the authors, the adherence to gluten-free diet and paying close attention to the selected food products may initiate eating disorders. On the other hand, the diagnosis of celiac disease in the patient already suffering from AN may aggravate the illness due to the need for strict adherence to the diet.

In the study by Basso et al. [18], the prevalence of CD in AN patients is not significantly different from that found in the general population. However, in our department serological diagnosis of celiac disease are routinely performed, taking into account other literature reports, our own experience and the fact that most cases of CD are not full-blown or have unusual nature.

Lower gastrointestinal tract

Motility disorders in the course of anorexia nervosa may also affect the distal part of the gastrointestinal tract. They may be responsible for constipation occurring in approximately 60% of patients with AN [1].

The bowel transit time, radiologically evaluated using shading signs, is prolonged in patients with anorexia nervosa as compared to healthy subjects [1, 19]. Some authors have also found rectal and anal motor weakness, as well as abnormal sensation of rectal filling in anorectal manometry, which does not resolve after nutritional rehabilitation [1, 19].

Liver dysfunction

Negative energy balance leads to liver dysfunction. The most frequently reported irregularities include hyperbilirubinemia and elevated serum levels of transaminase, observed in approximately 1/3 of hospitalized patients [20]. These disorders are usually mild and become normalized after weight gain [21].

The pathogenesis of elevated serum levels of transaminases in AN has not been fully explained. They may result from ischemia or liver steatosis, low concentration of glutathione or starvation-induced hepatocytes autophagy [21]. According to Hanachi et al. [22] risk factors for hypertransaminasemia in AN include age <30 years, BMI <12 kg/m^2, male gender and restrictive form of the illness.

Acute liver failure is a very rare complication of anorexia nervosa. Furuta et al. [23] reported a case of a 20-year-old woman suffering from AN who developed hepatic encephalopathy. Despite plasmapheresis, the patient developed acute renal

failure, pulmonary edema and disseminated intravascular coagulation syndrome. In turn, Di Pascoli et al. [24] reported a case of a 26-year-old female patient with acute liver damage in the course of multiple organ failure caused by dehydration and malnutrition.

Pancreatic dysfunction

Severe malnutrition can cause significant impairment in the secretion of pancreatic enzymes. However, it normalizes after successful nutritional treatment. The pathogenesis of this phenomenon is still widely debated [25].

A study conducted on malnourished monkeys has shown that chronic energy deficit leads to structural changes in the pancreas, including follicular cell atrophy [26]. These observations have also been confirmed in humans who in the course of moderate to severe malnutrition show an increase in serum trypsinogen, which may indicate damage to the follicular cells or pancreatic duct obstruction [27].

Studies concerning serum concentrations of amylase and lipase as markers of pancreatic exocrine dysfunction in anorexia nervosa are not numerous [28–30]. Most frequently reported is an isolated increase in amylase activity in serum and urine, which is usually observed in purging AN as a result of parotid gland stimulation [28]. However, some literature reports have also shown the increased activity of this enzyme in restrictive AN. However, the pathomechanism of this phenomenon has not been fully explained yet [28].

A few studies have evaluated both amylase and lipase activity. The increased serum levels of these enzymes were not associated with any clinical symptoms or ultrasound scan abnormalities and were normalized after weight gain [29].

There are several reports of cases of pancreatitis in patients with AN. However, the pathomechanism of this complication is not fully understood [30]. Parenchymal organ damage may occur due to increased pressure in the pancreatic duct. Stasis of pancreatic juice may in fact cause premature conversion of inactive trypsinogen into trypsin still within the pancreas, which leads to the activation of other proteases destroying the gland tissue. The factor predisposing to the development of pancreatitis may also include an increase in duodenal pressure and retrograde reflux of the duodenal contents into the pancreatic duct. It is usually caused by distension, resulting from both chronic starvation and rapid re-alimentation [31].

Pancreatitis in the course of AN may also be associated with applied pharmacotherapy. It has been shown that tricyclic antidepressants, unlike selective serotonin reuptake inhibitors (SSRIs), may increase the risk of this complication [32]. However, cases of pancreatitis in patients with anorexia nervosa during therapy with sertraline have been reported [33].

Treatment of gastrointestinal complications in AN

Attempts have been made to use prokinetic drugs in the treatment of motility disorders of the gastrointestinal tract in patients with AN. Some patients were treated with metoclopramide due to its stimulating effect on peristalsis and stomach empting. The therapy was found to accelerate stomach emptying and reduce such symptoms as epigastric pain, belching, vomiting, lack of appetite and early satiety [34].

Cisapride was also administered to treat stomach emptying in the course of AN. Its effectiveness was evaluated in a randomized, placebo-controlled study. All patients receiving the drug showed accelerated stomach emptying, and in most patients severity of symptoms associated with retention of gastric contents was reduced. In the control group, stomach emptying was improved after 6 weeks of nutritional therapy only in half of the subjects [35].

In another study, Szmukler et al. [36] did not confirm the beneficial effect of cisapride on the rate of stomach emptying assessed with scintigraphy in patients with AN. However, the therapy resulted in reduced severity of subjective symptoms related to food intake and appetite improvement [36].

Based on the observations presented above, it can be concluded, that both metoclopramide and cisapride can alleviate the symptoms of postprandial fullness by increasing the motor activity of the stomach, and thus contribute to changing eating behaviors in patients with AN. These drugs should be considered adjunctive therapy in patients with severe somatic symptoms resulting from motility disorders of the gastrointestinal tract.

Gastrointestinal complications of AN requiring surgical intervention

Literature reports describe several cases of stomach perforation as a result of hyperinflation caused by episodes of binge eating in patients with the purging form of anorexia nervosa [37] and a case of perforation of the sigmoid colon due to intestinal wall ulceration caused by fecal impaction [38].

Superior mesenteric artery syndrome, i.e., incarceration of the lower part of the duodenum between the superior mesenteric artery and the aorta, may also occur in the course of AN, which may result in bowel obstruction [39].

Yamada et al. [40] presented a case of a 41-year-old woman suffering from AN for many years, who developed necrosis of the ileum and the cecum. The patient died from septic shock three days after surgery. The autopsy showed no obstruction of the mesenteric artery and no atherosclerotic lesions. This case suggests that intestinal necrosis may be caused by ischemia due to severe malnutrition and liquid deficiency [40].

In our center, a 17-year-old patient with AN had bowel obstruction requiring urgent surgical intervention. The obstruction was due to birth defect – imperfective return of the intestines, and was induced by constipation, typical for the underlying illness, and the use of risperidone [41].

Refeeding syndrome

Too rapid refeeding carried out both orally and parenterally in malnourished patients may result in refeeding syndrome (RS) [42]. It is characterized by diselectrolitemia, especially hypophosphatemia, with accompanying fluid retention and impaired carbohydrate metabolism [42, 43].

Systemic phosphorous resources decrease during starvation in order to maintain its normal serum concentration. Sudden return to normal feeding after a long period of malnutrition, especially increased intake of carbohydrates, leads to a rise in insulin secretion and enhanced penetration of phosphorus from the extracellular to intracellular space, where it is used for the production of adenosine triphosphate (ATP) and 2,3-diphosphoglicerol (2,3-DPG) [42–44]. Secondary reduction in the production of high-energy phosphates due to phosphorus deficiency in the body leads to serious, life-threatening consequences: heart failure, arrhythmia and sudden cardiac arrest, respiratory insufficiency due to diaphragm weakening, peripheral edema and neurological disorders (paraesthesia, paralysis, seizures and impaired consciousness). During RS there may also appear gastrointestinal disturbances such as nausea and vomiting, dysphagia, constipation and intestinal paralytic ileus [42].

The main risk factor for the development of RS is the degree of malnutrition and the severity of symptoms depends on the serum level of phosphates. The clinical symptoms may not be found in phosphatemia above 1.5 mg/dl, and the decrease in serum concentration of phosphorus below 1.0 mg/dl can lead to severe RS [42–45].

Guidelines on nutritional therapy in AN

In order to avoid RS in patients with AN, it is necessary to gradually increase the number of introduced calories [44]. However, lack of coherent recommendations concerning the number of calories provided at the initial stage of nutritional treatment and the rate of calorie delivery increase in time poses a problem. The European guidelines recommend starting refeeding from 5–20 kcal/kg b.w., whereas the US ones from 30–40 kcal/kg. According to the Royal Australian and New Zealand College of Psychiatrists nutritional therapy should be launched with the supply of 1,400 kcal/day, increasing the caloric content of meals by approximately 400–500 kcal every three days [20]. Meals should be eaten under supervision, and in specific cases administration of high-protein supplements (100 kcal/100 ml), enteral nutrition through a nasogastric tube or parenteral nutrition should be considered [20, 43]. The expected weight gain in hospital conditions should oscillate between 0.5 and 1 kg/week. [43, 44].

However, most recent publications suggest that limiting the amount of energy supplied at the beginning of treatment does not reduce the risk of RS [43]. This syndrome may occur during nutritional treatment of different caloricity, which casts doubt on the direct relationship between the total amount of energy consumed and the likelihood of RS [44, 46].

It seems that RS prevention does not depend so much on the limitation of total caloricity of meals, but rather on a decrease in carbohydrate intake to 40% of daily caloric intake [47] which prevents a sudden increase in insulin secretion responsible for electrolyte imbalance, especially hypophosphatemia [44].

According to Ornstein et al., the occurrence of RS is associated only with hypophosphatemia, especially when diagnosed before the start of refeeding [48]. Therefore, routine monitoring of this parameter during the initial stages of nutritional therapy is recommended. Phosphate supplementation at a suggested dose of 15–30 mg/kg b.w. (1 ml of a phosphate mixture contains 36 mg of phosphorus) can be considered [43].

The treatment of purging AN should also involve the control of serum levels of potassium. Hypokalemia is usually observed in patients taking laxatives or provoking vomiting [47]. Due to the increased risk of osteopenia and osteoporosis, all patients with anorexia nervosa should receive calcium and vitamin D_3 [49, 50].

To conclude, patients with AN may develop non-specific gastrointestinal symptoms of usually unknown cause, both in the course of the illness itself and during refeeding. In most cases, they are functional in nature and resolve spontaneously with the improvement in body weight. The differential diagnosis of these disorders should, however, take into account the organic changes that might lead to serious consequences, particularly in extremely malnourished patients.

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