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REVIEW ARTICLE

A comprehensive review of the medical complications associated with restricting behaviors in anorexia nervosa

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ABSTRACT

Objective: The purpose of this paper is to comprehensively review the medical complications associated with restricting behaviors in anorexia nervosa and their recommended treatments.

Methods: A thorough review of the literature through February 2026 was completed.

Results: The medical complications associated with restricting behaviors in anorexia nervosa affect every body system and are increasingly common with greater severity of malnutrition.

Conclusion: Medical complications associated with anorexia nervosa are associated with high morbidity and mortality and are generally reversible, or at least associated with reduced symptomatology, with weight restoration.

Introduction

Anorexia nervosa (AN) is a highly lethal mental health illness with physical complications affecting every organ system. The diagnosis is based on the following criteria: 1) restriction of energy intake relative to requirements, 2) an intense fear of gaining weight, and 3) a disturbance in the way in which one's body weight or shape is experienced[1]. Individuals who engage in recurrent episodes of binge eating or purging behaviors during the preceding three months are diagnosed with anorexia nervosa, binge eating/purging subtype (AN-BP), and individuals not engaging in bingeing or purging behaviors are diagnosed with the restricting subtype of AN (AN-R). A severity index also characterizes individuals with a body mass index (BMI) ≥ 17 kg/m^2 as 'mild' disease, while those with a BMI < 15 kg/m^2 qualify as 'extreme' AN.

Individuals with AN have about five times greater mortality than people without AN[2,3]. The increased mortality is the second highest of all psychiatric illnesses, second only to deaths related to opioid abuse. Although much of this increased mortality stems from suicide[4], iatrogenic complications during refeeding and the medical complications resulting directly from starvation also contribute[5]. Low BMI is the single greatest predictor for development of these medical complications discussed herein[6] and is also highly predictive of increased mortality in AN[2].

This mortality is frequently attributed to "natural causes" that often result directly from the malnutrition[2,3]. Thus, a thorough understanding of these consequential medical complications in AN is critical for any provider treating this population. This review will focus on the medical complications that result directly from restrictive behaviors in individuals with AN. Table 1 provides a list of these complications as well as their presenting symptoms and specific treatment(s), although weight restoration is the primary therapy for them.

Table 1. Starvation-related medical complications in anorexia nervosa

Medical complication	Etiology	Clinical presentation	Treatment options
Digestive system			
Oropharyngeal dysphagia	Weakness of the oropharyngeal muscles	Coughing with oral intake	WR, speech pathology consult, NPO
GERD	Gastroparesis	Heartburn, regurgitation, dysphagia, chest pain	WR, Acid suppressants
Functional heartburn	DGBI	Heartburn	WR, Neuromodulators
Functional dyspepsia	DGBI	Nausea, early satiety, epigastric pain, bloating	WR, Neuromodulators, PPIs, H2Bs
SMA syndrome	Duodenal narrowing from mesenteric fat pad atrophy	Postprandial abdominal pain, nausea/vomiting	WR, psychological support
Starvation hepatitis	Hepatic autophagy	Abnormal LFTs	WR
Refeeding hepatitis	Steatosis of the liver	Abnormal LFTs, steatosis on imaging	Consider reduction in carbohydrate intake
Gallstones	Bile stasis and cholesterol turnover	Right upper quadrant abdominal pain	WR, Supportive care
Functional constipation	DGBI	Reduced frequency of defecation, pain, bloating	WR, osmotic laxatives, neuromodulators
Rectal prolapse	Excessive straining, pelvic floor dysfunction	Incomplete bowel evacuation, rectal prolapse	WR, pelvic floor exercises, surgery
Pulmonary system			
Hypercarbia	Diaphragmatic weakness	Asymptomatic	WR

Aspiration pneumonia	Oropharyngeal dysphagia	Variable	Supportive care, antibiotics
Pneumothorax/ pneumomediastinum	Iatrogenic, pulmonary histologic changes	Variable: asymptomatic, hypoxia, dyspnea, chest pain	Supportive care, oxygen, chest tube
Cardiovascular system			
Bradycardia	Vagal predominance	Low heart rate, hypotension	WR
Hypotension	Reduced CO, slowed metabolism, hypovolemia	Orthostasis, low blood pressure, hypotension	WR, hydration, d/c contributing meds
Mitral valve prolapse	Cardiac hypotrophy	Possible mid-sytolic click on auscultation	WR
Pericardial effusion	Unknown	Variable	WR, pericardiocentesis (cardiac tamponade)
Acrocyanosis	Thermoregulatory function	Painless cyanosis of the extremities	WR
Muscular system			
Sarcopenia	Atrophy of the skeletal muscles	Weakness, falls	WR, physical therapy
Pelvic floor dysfunction	Atrophy of the pelvic floor muscles	Organ prolapse, Increased GI symptoms	WR, pelvic floor therapy
Urinary system			
Acute kidney injury	Reduced glomerular filtration	Elevated creatinine	WR, adequate fluid intake
Hyponatremia	Excess water intake, impaired water diuresis	Nonspecific neurological symptoms	WR, appropriate water and solute intake
Endocrine system			
NTIS	Reduced leptin levels	Altered TFTs, slowed metabolism	WR
Amenorrhea	Reduced secretion of GnRH	Absent menses	WR, can consider transdermal estrogen
Hypoglycemia	Lack of metabolic substrates, hormonal changes	Frquently asymptomatic low blood glucose	WR, urgent correction of hypoglycemia
Hypophosphatemia/ refeeding syndrome	Transition from catabolic to anabolic state with introduction of nutrition	Hypophosphatemia, heart failure, , edema, hemolysis, respiratory insufficiency, seizures, rhabdomyolysis	Appropriate monitoring and correction of electrolytes during early refeeding
Refeeding edema	Sodium and water retention due to insulin	Edema, aggressive weight trends	Supportive care, low sodium diet
Hypercholesterolemia	Accelerated metabolism of fatty acid precursors	Elevated total cholesterol, HDL, and LDL	WR
Skeletal system			
Low bone mineral density	Hormonal changes, dietary changes, and reduced mechanical loading	Increased fracture risk, abnormal DXA scans	WR, pharmacologic agents
GMT	Abnormal bone marrow	Leukopenia/anemia >> thrombocytopenia	WR
Nervous system			
Compressive neuropathy	Nerve compression from reduced subcutaneous fat	Localized paresthesias or weakness	WR

Integumentary system			
Lagophthalmos	Atrophy of the orbital fat pad	Blurry vision	WR, lubricating eye drops
Patulous eustachian tube	Atrophy of the eustachian tube adipose tissue	Autophony, aural fullness	WR

Abbreviations:

CO (cardiac output), DGBI (disorder of gut brain interaction), DXA (dual-energy x-ray absorptiometry), GERD (gastroesophageal reflux disease), GI (gastrointestinal), GMT (gelatinous marrow transformation), GnRH (gonadotropin releasing hormone), HDL (high density lipoprotein), H2Bs (histamine-2 blockers), LDL (low density lipoprotein), LFTs (liver function tests), NPO (nothing by mouth), NTIS (non-thyroidal illness syndrome), PPIs (proton pump inhibitors), SMA (superior mesenteric artery), TFTs (thyroid function tests), WR (weight restoration)

Digestive system

Oral complications

Although oral complications are more commonly associated with vomiting behaviors[7], individuals with AN-R are also at increased risk. Frequency of dental caries may be increased in AN due to the associated dietary changes, including increased consumption of carbonated beverages and extended exposure to acidic substances[8]. Xerostomia (dry mouth), related to reduced fluid consumption and adverse effects of many of the medications used to treat comorbid conditions, such as anxiety and depression, may further increase the risk for dental caries. Xerostomia and micronutrient deficiencies also contribute to development of oral mucosal lesions[9,10].

Oropharyngeal dysphagia

Dysphagia, a subjective sensation of difficulty swallowing, is a common complaint in individuals with AN. One study found a high frequency of oropharyngeal dysphagia on video fluoroscopic swallow studies in individuals who complained of dysphagia, resulting from weakness and atrophy of the oropharyngeal muscles[11]. Low BMI is a strong predictor for development of oropharyngeal dysphagia, although long-standing reflux can sometimes trigger pharyngeal irritation and further contribute to oropharyngeal dysphagia. Symptoms frequently include difficulty swallowing, a sensation of food lodged in the throat, and coughing with oral intake. Weight restoration alone tends to improve the function of the swallowing muscles, although consultation with a speech language pathologist should be considered in more severe cases. Use of post-pyloric feeding tubes and nothing by mouth should also be considered during the early stages of refeeding in individuals who have aspirated or are deemed high risk for aspiration[12].

Gastroesophageal reflux disease (GERD) and Functional heartburn

There is an association between gastroparesis and symptoms of reflux[13]. However, individuals with AN-R who report symptoms of reflux, which often includes heartburn, regurgitation, and chest pain, frequently have normal pH impedance testing[14] and lack of findings on endoscopy that are consistent with esophagitis[15], suggesting that symptoms of heartburn are instead most often due to a disorder of gut brain interaction (DGBI) known as functional heartburn[16]. Upper endoscopy is required to make a diagnosis of functional heartburn, but its utility is unclear in those with AN-R who have a high likelihood of functional heartburn, and whose symptoms are expected to improve with weight restoration[16].

Individuals with frequent, bothersome reflux who do not respond to first-line treatment that includes proton pump inhibitors (PPIs) should be considered for pharmacological agents that are beneficial for the treatment of functional heartburn, including neuromodulators, such as tricyclic antidepressants (TCAs), and histamine-2 blockers (H2Bs). Alternative therapies, including acupuncture and

hypnotherapy, have also shown benefit for functional heartburn in the general population[17].

Functional dyspepsia

Gastroparesis, or slowed gastric emptying, is a nearly universal finding in those with AN. However, there is poor correlation between gastric emptying times and associated gastroduodenal symptoms, including early satiety, bloating, nausea, and vomiting[18]. Furthermore, subjects in the Minnesota starvation experiment developed nearly a 30% increase in gastric emptying times from baseline but did not report any GI distress during both starvation and the controlled refeeding portion of the experiment[19]. This would suggest that symptoms historically attributed to gastroparesis are actually better attributed to DGBIs, specifically functional dyspepsia[18]. Indeed, some studies have found that nearly 100% of individuals with AN before treatment meet criteria for functional dyspepsia[16,20], although most studies report a lower incidence.

Gastric emptying scans are generally not warranted in those with AN for the workup of symptoms including satiety, bloating, nausea, and vomiting, given the high likelihood of meeting diagnostic criteria for gastroparesis and their poor predictability for these symptoms. Furthermore, the diagnostic yield of upper endoscopy in the general population with symptoms compatible with functional dyspepsia and without alarm features, such as older age or an upper GI bleed, is very low[21]. It is also the recommendation of these authors that endoscopy is not warranted in those with AN for similar reasons, unless atypical features, such as melena, are present. Testing for Helicobacter pylori in the general population with suspected functional dyspepsia is recommended; however, it remains unknown if those with AN and functional dyspepsia should be tested for this infection.

Weight restoration alone results in improved GI symptoms, although it is common for symptoms to persist and for new DGBIs to appear[16]. Psychological interventions also result in symptom improvement[22], given the high prevalence of comorbid psychological factors that contribute to development of functional dyspepsia[23]. PPIs and H2Bs are first-line treatments for functional dyspepsia in the general population, and they should be trialed in those with AN as well[24]. Currently available prokinetics, such as metoclopramide, have not actually been studied in the eating disorder population, although they are frequently prescribed. Close monitoring is warranted with use given their risk for QT prolongation, extrapyramidal symptoms, and tardive dyskinesia. TCAs, such as amitriptyline and imipramine, are also effective neuromodulators for functional dyspepsia[25].

Superior mesenteric artery syndrome

The duodenum can become compressed between the superior mesenteric artery (SMA) and the aorta with weight loss, due to atrophy of the mesenteric fat pad that normally positions the SMA away from the aorta. The resultant SMA syndrome is associated with symptoms of postprandial abdominal pain along with nausea and occasionally vomiting. A computed tomography (CT) scan with intravenous contrast is considered the gold standard for diagnosing this condition, and finding an aortomesenteric angle $\leq$ 25 degrees, especially with an aortomesenteric distance $\leq$ 8 mm, is highly sensitive for a diagnosis of SMA syndrome[26]. Modest weight gain, whether using oral, enteral, or parenteral nutrition, depending on the severity of the duodenal narrowing and tolerance of oral nutrition, in addition to psychological support, results in normalization of the radiologic findings and resolution of symptoms[27].

Starvation and refeeding hepatitis

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are frequently elevated in AN, a condition known as starvation hepatitis that results from programmed cell death (autophagy) of the hepatocytes[28,29]. Starvation hepatitis is associated with low BMI and increased likelihood

of hypoglycemia, presumably serving as a metabolic response to starvation as the triglycerides, glycogen, and other nutrients stored in the liver are thereby released to the rest of the body[30]. Starvation hepatitis also contributes to the elevated serum B12 levels in those with AN[31], as the liver serves as the main site of storage. Weight restoration results in rapid improvement in AST and ALT, and additional diagnostic testing is not warranted, unless liver function tests fail to improve/normalize with continued weight restoration.

Refeeding hepatitis, on the contrary, develops during the early stages of refeeding as excess carbohydrates stimulate insulin secretion that ultimately leads to deposition of triglycerides and glycogen in the liver[32]. Imaging in refeeding hepatitis often reveals steatotic changes along with hepatomegaly, contrary to the unremarkable imaging in starvation hepatitis. Treatment is generally not warranted as there are no known long-term adverse effects from refeeding hepatitis. However, in more severe cases associated with significant increases in AST and ALT, a small reduction in carbohydrate intake will result in rapid improvement in the AST and ALT.

Gallstones

Increased bile stasis and cholesterol levels resulting from starvation lead to changes in bile composition that can cause gallstone formation[33]. Gallstones may be asymptomatic and incidentally found during workup for other GI complaints, in which case weight restoration is the recommended treatment. However, cholecystectomy is generally recommended for individuals with symptomatic gallstones, although the effectiveness of this approach has not been investigated in AN, and it may be difficult to discern the symptoms of gallstones from the GI symptoms associated with the highly prevalent DGBIs.

Functional constipation

Although several weeks of nutritional rehabilitation will result in normalization of the slowed colonic motility that develops from weight loss, individuals continue to report symptoms of decreased frequency of defecation, abdominal pain, abdominal distension, and bloating[34,35], demonstrating a complex pathophysiology for the symptoms associated with functional constipation. Similarly, many individuals with AN meet criteria for irritable bowel syndrome[20]. Constipation alone is not an indication for colonoscopy, and this should only be sought in individuals with AN when other concerning symptoms are present, such as non-hemorrhoidal bleeding, or if there is concern for other conditions.

Bowel function should be optimized with normalization of electrolytes and glucose, along with adequate hydration. First-line pharmacologic treatment should consist of osmotic laxatives, at least until increased fiber can be introduced into the diet, given the negative effects of fiber on slowed gastric motility and symptoms of fullness. Stimulant laxatives should be used cautiously in those with AN given the increased risk for abuse. Similarly, secretagogues often prescribed for the treatment of irritable bowel syndrome, such as lubiprostone and linaclotide, are unstudied in the AN population and could theoretically be associated with increased risk for abuse. Lactulose may worsen bloating due to intestinal bacterial fermentation of the medication and should be avoided in those with AN. Peppermint oil has numerous beneficial effects on gastrointestinal physiology, with a good safety profile, making it an effective treatment for many of the GI symptoms and associated DGBIs in individuals with AN[36].

Rectal prolapse

Although rectal prolapse is more common in individuals with purging behaviors, individuals with AN-R are also at risk for development of rectal prolapse[37,38]. Frequent straining related to constipation and/or weakness of the pelvic floor musculature can contribute to its development, manifesting as protrusion of the rectum through the anal canal. Weight restoration and exercises to strengthen the pelvic floor muscles are generally

recommended for treatment, although surgery may be required in severe cases[39].

Pulmonary system

Hypercapnia

Hypercapnia, retained carbon dioxide, is a common laboratory finding in individuals with AN, especially at increasingly low body weight, due to diaphragmatic weakness[40,41,42]. This is usually mild and asymptomatic, unless there is increased severity of retained carbon dioxide due to additional pathology. Similarly, it is uncommon for individuals to report dyspnea and to develop hypoxia from diaphragmatic weakness, unless additional pathology is present.

Aspiration pneumonia

Oropharyngeal dysphagia is a risk factor for development of aspiration pneumonia[11]. Chemical pneumonitis, due to aspiration of acidic gastric contents, can result in an inflammatory reaction that typically presents with an abrupt onset of respiratory distress and vital sign abnormalities, including hypoxia and fever, although more severe cases can result in fulminant respiratory failure. Pure chemical pneumonitis tends to resolve quickly and requires only supportive care but can be difficult to differentiate from bacterial pneumonia that results from aspiration of bacteria from the oropharyngeal cavity and which requires antibiotic treatment. Bacterial pneumonia presents with a more subacute clinical presentation than chemical pneumonitis. Aspiration pneumonia is associated with increased mortality in those with AN[43]; therefore, prevention is critical. Preventative care can include use of post-pyloric enteral nutrition during the early stages of refeeding for individuals at high risk for aspiration[12].

Pneumothorax/pneumomediastinum

Pneumothorax (air within the pleural space that surrounds the lung) and pneumomediastinum (air outside the pleural space but within the chest cavity) are known complications associated with AN. Although vomiting and straining during bowel movements increase intra-thoracic pressure, suggesting that individuals with a purging history might be higher risk for development of these complications, many case reports are described in individuals without a purging history[44,45,46]. It is hypothesized that pneumothoraces in this population result from structural abnormalities to the lungs that are directly caused by the malnutrition[47]. Pneumomediastinum is believed to most often result from a transient, self-limited alveolar rupture, with air then dissecting the peribronchovascular sheaths into the mediastinum and subcutaneous tissues (Macklin effect)[48]; no case reports have described development of pneumomediastinum resulting from esophageal rupture (Boerhaave's syndrome), and there are limited case reports in the AN population describing concomitant pneumothorax and pneumomediastinum[44]. Symptoms frequently include acute onset of chest pain and dyspnea, with or without hypoxia, although presentation may also be asymptomatic. Treatment generally depends on the size of the pneumothorax and the stability of the patient but can include supportive care with oxygen and/or a chest tube for lung re-expansion; furthermore, individuals with AN are at risk for prolonged air leaks[49]. Presence of pneumomediastinum due to a transient air leak generally requires only supportive care.

Cardiovascular system

Bradycardia

Bradycardia is a nearly universal finding in individuals with AN that is believed to result from increased parasympathetic (vagal) predominance[50]. Although the ejection fraction, defined as the percentage of blood ejected from the ventricle in relation to the volume of blood pumped into the ventricle, is unaffected in AN[51,52], bradycardia does result in reduced cardiac output (the volume of blood pumped by the heart per minute), which can contribute to the increased risk for syncope in this population[53]. Additional brady-arrhythmias, such as junctional escape rhythms, become increasingly likely with worsening severity

of bradycardia and likely contributes to the increased mortality in this population[54,55]. For this reason, adults presenting with heart rates less than 40 beats per minute (< 45 beats per minute for adolescents) should be monitored on telemetry. Weight restoration is the primary treatment for bradycardia, and pacemakers should be avoided in those with AN as chronotropic response remains intact[56]. In fact, individuals with AN exhibit an exaggerated increase in heart rate with even minor activity, contrary to the insignificant increase in heart rate seen in athletes completing a similar level of activity.

Hypotension

Hypotension is another frequent complication in individuals with AN, due to decreased cardiac output, reduced metabolic rate, decreased triiodothyronine (T3) levels, and hypovolemia. It is common to observe an exaggerated heart rate increase upon standing, which is an expected compensatory physiological response in this population resulting from reduced venous return to the heart from sarcopenia and increased baroreceptor sensitivity[49]. However, orthostatic hypotension, defined as a greater than 20 mm Hg drop in systolic blood pressure and/or greater than 10 mm Hg drop in diastolic blood pressure upon standing, less commonly develops in those with AN, and is more likely to be reported in those who engage in purging rather than restricting behaviors[57,58]. Although postural orthostatic tachycardia syndrome (POTS), defined as a heart rate increase of ≥ 30 beats per minute within 10 minutes of standing and in the absence of orthostatic hypotension, is frequently diagnosed in those with AN, this diagnosis is under-studied in this population. Furthermore, a significant number of individuals with restrictive eating disorders (and an even greater number of individuals with a purging history) will exhibit a heart rate increase greater than 30 beats per minute upon standing from a supine position, with pulse frequently doubling or even tripling with positional changes[58]. Many of the psychiatric medications used to treat comorbidities in this population also likely impact the exaggerated tachycardia, more so than the malnutrition itself[59]. Hypotension is often asymptomatic but can manifest as lightheadedness upon standing and can increase the risk for syncope, especially when orthostasis is present. Adequate hydration improves the exaggerated tachycardia and hypotension, although it is unlikely to fully normalize the vital signs, which occurs once individuals weight restore to about 80% of ideal body weight (IBW)[57,58].

Mitral valve prolapse

The reduced hemodynamic load placed upon the heart, as well as the development of cardiac hypotrophy from malnutrition itself, results in reduced left ventricular mass and thickness[60,61]. This causes a mismatch between the size of the mitral valve and the dimensions of the left ventricle, inducing prolapse of the mitral valve[62,63]. Mitral valve prolapse is most often diagnosed based on results from an echocardiogram, as auscultatory findings of a mid-systolic click and murmur on exam are suggestive, but often absent. It is most often an asymptomatic complication, and a direct association between the prolapse and symptoms of chest pain, palpitations, dizziness, and dyspnea remains unclear given their nonspecific nature. Additional treatment for mitral valve prolapse is not warranted aside from weight restoration, which results in normalization of cardiac hypotrophy and, therefore, the prolapse[62].

Pericardial effusion

Development of fluid within the pericardial sac surrounding the heart is reported in about 25% of individuals with AN[61,64]. Hypotheses for development of the effusion include physiologic changes that result from low T3 versus an enlarged pericardial space, due to reduced pericardial fat, that becomes occupied by a transudative fluid[65]. A large pericardial effusion can rarely result in reduced cardiac filling and contractility, a condition known as cardiac tamponade, although pericardial effusions are most often an asymptomatic and

incidental finding. Clinical symptoms suggesting cardiac tamponade warrant urgent drainage of the effusion via a pericardiocentesis but conservative management with continued weight restoration should be considered if only echocardiographic findings suggestive of cardiac tamponade are present[65].

Acrocyanosis

Acrocyanosis is a painless, cyanotic discoloration of the extremities that develops with cold exposure[66]. The pathophysiology of acrocyanosis remains poorly defined, although it is believed to result from impaired vasodilatation that serves as a protective mechanism to conserve body heat[67]. Anecdotally, improvement is seen with weight restoration.

Muscular system

Sarcopenia

Sarcopenia, the disease of compromised muscle mass, strength, and function, is a common finding in individuals with AN. Although malnutrition is associated with a generalized myopathy, type II (fast-twitch) fibers, which rely on anaerobic metabolism and are important for balance and quick movements, are more affected in AN, compared to type 1 (slow-twitch) muscle fibers, which rely on aerobic metabolism and are important for endurance activities[68,69]. These changes result in an average relative loss of muscle mass of about 25%, which is associated with a mean reduction in muscular strength of about 35%[70].

The greater loss of type II fibers contributes to the high fall risk in this population, often resulting in individuals being placed on strict bedrest during hospitalization[71,72]. However, bedrest results in even greater risk for additional physical harm from progressive deconditioning due to lack of physical activity as well as increased risk for pressure ulcers from the lack of protective subcutaneous tissue. Weight restoration results in improved muscle mass and strength, although this may not reach age-matched controls after weight restoration[69]. Ensuring adequate physical activity and properly supervised exercise can improve treatment outcomes[73].

Pelvic floor dysfunction

Pelvic floor dysfunction results from dyscoordination of the pelvic floor, a group of muscles that support various organs in the pelvis and help to control urination and defecation[34]. Pelvic floor dysfunction not only contributes to increased risk for rectal prolapse, as discussed above, but is also likely associated with increased GI symptoms that are so prevalent in this population[74,75]. Low BMI is a significant risk factor for development of pelvic floor dysfunction, as individuals with AN are increasingly likely to develop pelvic floor dysfunction compared to atypical AN or bulimia nervosa[74], but causes for development of pelvic floor dysfunction in the eating disorder population are otherwise unknown. Active re-training of the pelvic floor musculature is beneficial for symptom improvement[76], and weight restoration is also recommended given its impact on muscle function in sarcopenia, although its impact toward improved pelvic floor dysfunction is unclear[34].

Urinary System

Acute kidney injury

Unlike the relation between purging behaviors and severe electrolyte deficiencies, such as hypokalemia and hypomagnesemia, as well as the increased risk for chronic kidney disease[77,78,79], the kidneys are relatively spared in AN-R. Hypovolemia and decreased cardiac output beget reduced renal perfusion that results in decreased glomerular filtration[80], although this may not be reflected by increased blood urea nitrogen or creatinine levels, given the state of malnutrition. Adequate hydration and refeeding results in rapid improvement[81].

Hyponatremia

Individuals with AN remain at risk for hyponatremia for several reasons: excessive water intake

(polydipsia), dehydration, and impaired water diuresis. Adequate solute intake, including glucose, protein, and sodium, is critical for optimal concentration of the urine and conservation of the body's water, with excessive diuresis developing below a critical point of intake[82,83]. Thus, treatment should focus on nutritional rehabilitation and appropriate fluid intake. Nonspecific neurologic symptoms become more prominent as serum sodium decreases below 125 mEq/L. Similarly, overly aggressive correction of serum sodium <120 mEq/L, that has been present for greater than two days, increases the risk for development of central pontine myelinolysis[84].

Endocrine System

Non-thyroidal illness syndrome

Starvation results in adaptive changes to the hypothalamic-pituitary-thyroid axis, known as non-thyroidal illnesss syndrome (NTIS). NTIS is generally associated with low-normal free thyroxine (T4), low-normal thyroid stimulating hormone (TSH), low total T3, and increased levels of reverse T3, with these changes becoming more pronounced as severity of malnutrition worsens[85]. In individuals with AN, this likely results at least in part from reduced leptin levels, the body's main orexigenic hormone produced by fat cells, which regulates many of the hormonal systems described herein[86,87]. This adaptive reduction in thyroid hormones helps to reduce the resting energy expenditure and conserve limited resources, as T3 is a major determinant of resting metabolic rate[88]. Weight restoration results in normalization of this axis, and thyroid hormone supplementation should not be used in this population given the potential negative impact on bone density, body weight, cardiovascular physiology, and others.

Oligomenorrhea and amenorrhea

Changes to the hypothalamic-pituitary-gonadal axis result from reduced hypothalamic pulsatility of gonadotropin-releasing hormone (GnRH) due to reduced fat mass and leptin levels[87]. Abnormal GnRH pulsatility causes reduced secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the pituitary gland, thereby shutting down production of testosterone and estrogen from the testicles and ovaries, respectively. The resultant hypoestrogenic state in AN is associated with both oligomenorrhea (infrequent menstrual bleeding) and amenorrhea (absence of menstruation for greater than three months)[89]. Onset of amenorrhea, as well as resumption of menses with weight restoration, are variable across individuals, although return of meses is often predicted by the weight at which menstruation stopped[90]. Restoration of estrogen levels via transdermal estrogen is beneficial for bone growth[91]; however, weight restoration is the primary treatment to normalize these hormone levels.

Growth hormone resistance

Growth hormone (GH) resistance, associated with increased levels of circulating GH from the pituitary gland and reduced levels of insulin-like growth factor 1 (IGF-1), produced from the liver, develops with acute starvation[92,93]. Although the mechanism of GH resistance in AN is not fully elucidated, these changes help drive the shift in metabolism from carbohydrates to fats, mediated by GH[94], while minimizing IGF-1 dependent anabolic processes, which unfortunately includes reduced bone mass. Weight restoration normalizes this axis[95], and pharmacologic interventions aimed at normalizing the GH pathway are not warranted and may actually be detrimental[96].

Hypercortisolemia

Increased levels of cortisol, the body's main stress hormone, are associated with AN due to up-regulation of the hypothalamic-pituitary-adrenal (HPA) axis[97]. This likely results from increased production of cortisol-releasing hormone from the hypothalamus[98], as well as increased levels of ghrelin, an orexigenic hormone produced primarily in the stomach[99]. Elevated cortisol levels help to maintain normoglycemia in the setting of decreased caloric intake, but at the cost of multiple

adverse physiologic effects, including reduced bone density, increased production of stomach acid that leads to development of gastritis, and accumulation of visceral fat that contributes to the central adiposity associated with refeeding of individuals with AN[100]. Weight restoration results in normalization of this axis, and cortisol-blocking medications should not be prescribed.

Hypoglycemia/insulin sensitivity

Individuals with AN are at increased risk for sustained, often asymptomatic, hypoglycemia that most frequently develops during times of fasting (e.g., overnight) and between meals, although patients with AN can also develop postprandial hypoglycemia[101,102]. This risk increases with greater severity of starvation and with concomitant starvation hepatitis[30]. The previously described hormonal adaptations, including elevated ghrelin levels, help to maintain normoglycemia, but the lack of glycogen stores and continued restriction reduce the effectiveness of these hormonally-induced metabolic responses. AN is also associated with reduced insulin secretion and increased insulin sensitivity[103,104], due in part to the restricted intake but also from increased adiponectin levels, a hormone secreted mostly from white adipose tissue, that enhances insulin sensitivity[105]. Consistent nutrition and weight restoration normalizes hypoglycemia.

Hypophosphatemia and refeeding syndrome

Refeeding syndrome refers to the metabolic and electrolyte disturbances that may occur following the reintroduction of nutrition in severely malnourished patients[106,107]. Individuals with AN are at particular risk during early nutritional rehabilitation due to prolonged caloric restriction and depletion of total body electrolyte stores. Starvation results in reduced insulin secretion and a shift toward fat and protein catabolism as primary energy sources. During this period, intracellular stores of phosphate, potassium, magnesium, and thiamine become depleted despite frequently normal serum concentrations. With reintroduction of carbohydrate-containing nutrition, insulin secretion increases and promotes cellular uptake of glucose along with phosphate, potassium, and magnesium required for adenosine triphosphate (ATP) production, as well as the transition from catabolic to anabolic metabolism[106,107].

Definitions of refeeding syndrome have varied historically; however, recent consensus recommendations from the American Society for Parenteral and Enteral Nutrition (ASPEN) define refeeding syndrome based on electrolyte changes, including phosphate, magnesium, and potassium, occurring within five days of initiating or substantially increasing caloric intake[108].

Although hypophosphatemia is the most commonly recognized biochemical abnormality during refeeding, refeeding syndrome represents a broader clinical process in which electrolyte and fluid shifts may result in significant cardiopulmonary, neurologic, and hematologic complications, with the full syndrome characterized by hypophosphatemia, diaphragmatic weakness, hemolysis, rhabdomyolysis, seizures, and heart failure[106,107].

Management of refeeding syndrome emphasizes prevention, early identification, and prompt correction of electrolyte abnormalities while continuing nutritional rehabilitation that may be best accomplished in specialized medical stabilization facilities. Thiamine supplementation should be administered prior to and during early refeeding, particularly when carbohydrate intake is increased, although this recommendation is based on the prevalence of thiamine deficiency in alcoholism[109]. Electrolyte replacement should address phosphate, potassium, and magnesium concurrently, as abnormalities frequently coexist.

Refeeding edema

Unlike the aggressive weight gain and edema that can develop in individuals with a purging history (pseudoBartter syndrome), individuals with AN-R are at risk for mild, yet bothersome, edema

formation within the first couple weeks of refeeding[108]. This refeeding edema is believed to result from increased insulin secretion associated with refeeding, as insulin causes sodium, and thereby fluid absorption, in the renal tubules[110]. Treatment for refeeding edema generally includes supportive measures, such as leg elevation and avoidance of excessive sodium intake, but pharmacologic measures are not warranted.

Hypercholesterolemia

Hypercholesterolemia, specifically total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL), is found in AN[111,112]. This results from accelerated metabolism of fatty acid precursors into HDL and LDL, although individuals with increasingly low BMIs have normalization of the hypercholesterolemia due to reduced efficiency of this metabolism[111]. There is no evidence to suggest increased risk for atherosclerosis in those with AN (contrary to BN[113]), and hypercholesterolemia normalizes with weight restoration.

Reproductive system

Pregnancy

Although oligomenorrhea and amenorrhea are very common in individuals with AN, fertility remains intact. Moreover, women with AN are actually at a greater than two-fold risk for unplanned pregnancy with unprotected intercourse, as ovulation can still occur in the absence of menses[114]. Active AN during pregnancy is also associated with increased risk for low birth weight and small-for-gestational age births[115].

Skeletal system

Low bone mineral density

Studies have found that greater than 50% of adults with AN have z-scores less than -2 on dual energy X-ray absorptiometry (DXA)[116,117], which qualifies for osteoporosis in the premenopausal population with AN (the term osteopenia should be avoided in those who are premenopausal). Bone density in this population is negatively affected for many reasons, including not only the aforementioned hormonal changes but also the typical age of onset of AN before peak bone mass is achieved, dietary changes, reduced mechanical loads on the bones from sarcopenia, and the excessive exercise that frequently accompanies AN and that results in further bone density loss[118]. These changes all contribute to the increased long-term fracture risk for those with AN, especially females[119]. Although DXA scans only assess bone density without testing other aspects of bone health, such as bone strength and quality, and scores from the DXA scan poorly correlate with fracture risk[117], DXA scans are nonetheless recommended for any individual with active AN and/or amenorrhea for more than 6-12 months, with repeat testing recommended every 1-2 years thereafter until bone density is stabilized. Weight restoration with resumption of menses, which should be considered the primary treatment, results in stabilization of bone density at one year, with a 3-4% yearly increase in bone density thereafter with continued weight restoration[120]. Transdermal estrogen has also been found to be efficacious in both adolescents and adults[91], while denosumab and teriparatide are beneficial in adults (not studied in adolescents). Bisphosphonates are unlikely to be beneficial in adolescents but are appropriate treatment considerations for adults[118]. Unfortunately, low bone density may be one of the irreversible complications associated with AN.

Gelatinous marrow transformation

Gelatinous marrow transformation (GMT), also known as serous fat atrophy, or colloquially as starvation marrow, is a frequently encountered medical complication in individuals with AN that contributes to variable rates of leukopenia, anemia, and, less commonly, thrombocytopenia. Although the amount of weight gain needed for normalization of peripheral blood counts remains unknown, bone marrow changes may be resolved fairly quickly with nutritional support[121]

Nervous system

Brain atrophy

Changes to the brain in individuals with AN include both a global reduction in grey matter as well as reductions in regional volumes[122,123]. White matter (myelin) volumes seem to be unaffected in acute starvation, although the organization of the myelin is likely disturbed[123,124]. These changes may contribute to some of the cognitive impairments reported in those with AN[125], although it remains unknown if the cognitive changes are related to the malnutrition itself or are trait-dependent changes. Adolescents seem to display more complete normalization of these changes with weight restoration than do adults, especially with regard to grey matter[123], and it remains unknown whether this may be one of the potentially irreversible complications associated with AN (in addition to bone disease).

Compression neuropathy

There is lack of evidence to support B12 and folate deficiencies in individuals with AN[126], and although increased prevalence of copper deficiency is described in this population[127], it is uncommon for patients to present with neurologic symptoms directly related to copper deficiency. However, individuals are at risk for nerve dysfunction due to compression neuropathy (mechanical compression of the nerves) from the loss of subcutaneous adipose tissue at locations where the nerves are located more superficially. For example, a compressive neuropathy of the peroneal nerve, resulting in numbness on the outer leg and foot drop, is commonly described in individuals with AN[128,129], although individuals with AN are also anecdotally at risk for paresthesias in the forearm due to ulnar nerve compression. As the amount of subcutaneous adipose tissue normalizes with weight restoration, and the nerve compression is relieved, nerve healing and regeneration can occur, although this can take weeks to months, depending on the severity of the injury. Physical and occupational therapy may also be beneficial for those with foot drop or other neurologic deficits.

Integumentary system

The skin manifestations associated with AN more commonly include xerosis, hypertrichosis lanuginosa (lanugo), telogen effluvium, nail fragility, and pruritus[130,131]. Xerosis (dry skin) is reported in a high number of individuals with AN and is caused by several factors, including lipid changes directly in the skin, changes to the amount and composition of sebum, hypothyroidism, and excessive hand washing[132]. Excessive hand washing is associated with pruritus, although hormonal changes and sebaceous gland activity also contribute. Lanugo presents as fine, downy hair, typically present on the face, back, abdomen, and forearms. It is not a sign of virilization but is likely related to hypothyroidism and helps to preserve heat[132]. Telogen effluvium is a condition whereby hair growth ceases, and there is increased shedding of hair as it enters the resting (telogen) phase of growth. Brittle nails likely develop due to a combination of excessive hand washing and vitamin deficiencies. On the contrary, excess vitamin A intake (carotenoderma) may be reflected by yellow pigmentation in areas such as the palms, soles, and nasolabial folds[132]. It remains unclear if those with AN are at increased risk for acne.

Lagophthalmos

Individuals with AN may report vision changes due to a condition called lagophthalmos. This is presumed to develop from atrophy of the orbital fat pad, and subsequent inability of the eyelids to fully cover the cornea when closed, potentially causing corneal drying and irritation[133]. Treatment consists of weight restoration and lubricating eye drops.

Patulous eustachian tube

Individuals with AN may also describe abnormalities in hearing, often including aural fullness, subjective hearing loss, and autophony, a condition known as patulous eustachian tube. It is

believed that weight loss promotes atrophy of the adipose tissue surrounding the eustachian tube, causing the eustachian tube to remain abnormally open; however, rapid resolution of this condition with nutritional rehabilitation, but before any meaningful weight gain, suggests additional pathophysiology[134].

Immune system

Studies suggest multiple changes to the immune system in AN, including increased T cell proliferation and increased production of pro-inflammatory cytokines such as tumor necrosis factor-1 and interleukin-1[135]. It is also suggested that individuals with AN are not at greater risk for infectious diseases, although it is possible that a reduced fever response and reduced clinical signs of infection may delay recognition[136,137].

Conclusion

With the potential exception of low bone density and neurologic changes, the myriad medical complications associated with AN-R are reversible with weight restoration that can be achieved with multidisciplinary teams consisting of medical providers, dieticians, and behavioral health providers. Appropriate medical oversight during both times of starvation and refeeding allows the rest of the multidisciplinary team to focus on the psychological, nutritional, and relational healing that is necessary for recovery from AN.

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