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Anorexia nervosa as a systemic disease: multisystem complications and their clinical significance

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ABSTRACT

Anorexia nervosa (AN) is an eating disorder with many factors - biological, psychological, and social. Chronic energy deficiency leads to adaptive but also pathological changes in many body systems. AN is increasingly recognized as a systemic disease associated with metabolic, endocrine, cardiovascular, neurological, and immunological complications. This study aimed to review recent information on how anorexia nervosa affects different body systems. It focused on the causes of these changes and whether they can improve with proper nutrition. Chronic malnutrition in AN affects the endocrine, skeletal, cardiovascular, gastrointestinal, nervous, hematopoietic, and immune systems. Many of these problems can get better when patients regain weight and eat enough. But if the disease is severe or lasts a long time, some issues may stay, like weak bones or changes in the heart and brain. There is also a risk of sudden problems during refeeding. Treating anorexia nervosa needs careful diagnosis and care. Finding the disease early and starting nutrition on time is very important to prevent lasting damage and improve long-term health.

Keywords: anorexia nervosa; multisystem complications; endocrine; cardiovascular; bone metabolism

1. INTRODUCTION

Anorexia nervosa (AN) is a psychiatric disorder marked by intentional and long-term weight loss. The condition develops due to a strong fear of gaining weight and a distorted body image. According to the criteria of the International Classification of Diseases, 10th Revision (ICD-10), a key feature of the disorder is the active restriction of food intake, often accompanied by compensatory behaviors (WHO, 2016). The disease most commonly occurs in adolescent girls and young women, which is related to hormonal, social, and cultural factors. However, it can also develop in people of any age and in both men and women (van Eeden et al., 2021). It is worth mentioning that AN has one of the highest mortality rates in mental

illnesses, both somatic complications and the risk of suicide (Neale and Hudson, 2020).

The causes of AN are complex and include genetic, biological, psychological, and environmental factors. A higher rate of the disorder among relatives and twins supports the genetic role. Social pressure and the promotion of idealized, slim body images in social media contribute to the rising prevalence of AN. In recent years, the incidence of the disorder has increased (van Eeden, et al., 2021). Due to intentional caloric restriction in patients with AN, chronic energy deficiency plays the main role in the pathophysiology of the disorder. This leads to deficiencies in macro- and micronutrients and vitamins. It results in metabolic disturbances and multisystem clinical consequences (Misra and Klibanski, 2014). Among these are endocrine abnormalities such as hypoestrogenism, hypogonadism, decreased levels of leptin and insulin-like growth factor 1 (IGF-1), and dysfunction of the hypothalamic–pituitary–adrenal (HPA) axis. These changes lead to a range of disturbances. The most common are decreased bone mineral density, growth retardation in young patients, and menstrual irregularities (Gibson and Mehler, 2019). The multisystem clinical effects also involve the cardiovascular, gastrointestinal, nervous, hematopoietic, and immune systems (Misra and Klibanski, 2014). The aim of this paper is to discuss the various interrelationships between chronic energy deficiency in AN and multisystem disturbances in patients, with particular emphasis on their clinical implications.

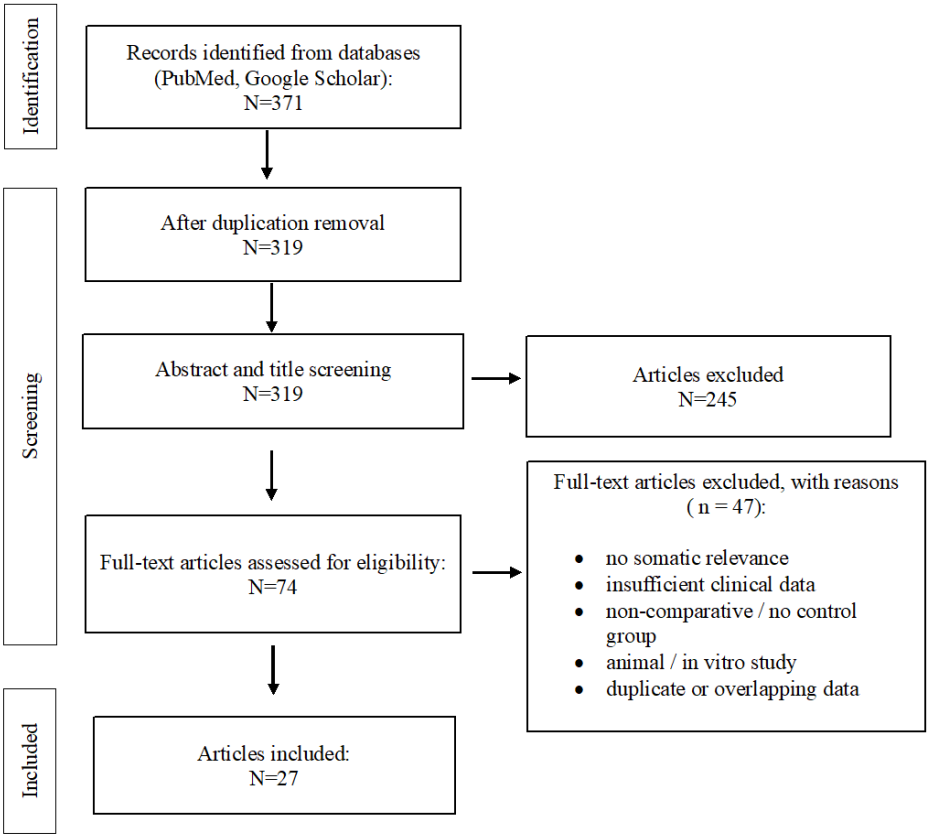

Figure 1. PRISMA flow diagram of the study selection process

2. REVIEW METHODS

The literature search was performed in PubMed and Google Scholar for papers published between January 2013 and August 2025. The search focused on medical and multiorgan complications of anorexia nervosa. The following keywords were used in different combinations: anorexia nervosa, medical complications, systemic effects, endocrine, cardiovascular, renal, gastrointestinal, hematologic, immunologic and brain MRI.

A total of 371 records were identified. We removed duplicates and then reviewed the titles and abstracts. We looked for studies on physical and organ-related effects of anorexia nervosa in adolescents and adults. We included clinical studies, reviews, guidelines, and case reports with valid medical data. We excluded studies focusing solely on psychological or behavioral aspects. The selection process is summarized in Figure 1 (PRISMA flow diagram).

3. RESULTS & DISCUSSION

Skeletal System in the Course of Anorexia Nervosa

One of the main complications of AN is reduced bone mineral density, what leads to an increased risk of osteoporosis, osteopenia, and low-energy fractures. In young patients who have not yet completed bone growth, peak bone mass may not be reached. This can lead to permanent effects in adulthood.

Several factors contribute to bone mineral loss in people with AN. It may seem that low intake of vitamin D and calcium due to dietary restriction is the leading cause. However, studies suggest that these nutrient levels are often higher in patients with AN than in the general population, as supplement use is more common in this group (Misra and Klibanski, 2014). Hormonal changes mainly cause lower bone mineral density in AN. Bone loss occurs because of problems in the hypothalamic–pituitary–gonadal axis, which lead to lower gonadotropin levels and reduced estrogen production. Low estrogen levels abolish its physiological protective effect on bone tissue, resulting in increased osteoclast activity and accelerated bone resorption (Misra and Klibanski, 2014). Decreased insulin-like growth factor 1 (IGF-1) levels also play a role in bone mineral loss in AN. Under normal physiological conditions, IGF-1 stimulates osteoblast proliferation and activity, promoting new bone formation. A lack of IGF-1 greatly slows this process (Fazeli and Klibanski, 2018). Reduced fat tissue in patients with AN also decreases leptin levels. It is a hormone produced by adipocytes. Leptin plays a vital role in the pathogenesis of bone mass loss in AN. Leptin is a satiety hormone that signals the hypothalamus to curb appetite. It also affects the hypothalamic–pituitary–gonadal axis. Under normal conditions, leptin stimulates the release of gonadotropins, so its deficiency worsens hypogonadism. In addition, leptin supports bone formation by activating osteoblasts and limiting bone resorption. Low leptin levels reduce this effect (Stefanakis et al., 2024).

Peptide YY (PYY) is another hormone essential for bone metabolism. It is produced in the intestine and acts together with leptin to signal satiety. In patients with AN, higher PYY levels further reduce appetite, helping to maintain the eating disorder. They also have an adverse effect on bone metabolism, as they are linked to lower bone mineral density. (Misra and Klibanski, 2014).

Cortisol is another key factor. Its levels are often high in AN. It is caused by overactivity of the hypothalamic–pituitary–adrenal axis. Cortisol usually helps the body respond to stress (Treviño et al., 2024). When it stays high for a long time, it harms bone tissue by slowing osteoblast activity. This can lead to osteocyte death and increased bone resorption. These changes get worse bone loss (Fazeli et al., 2018).

Together, these hormonal and metabolic changes in AN result in a marked drop in bone mineral density, leading to osteopenia and osteoporosis. Young patients can experience slow growth and incomplete bone development compared with healthy peers. As a result, the risk of fractures from minor injuries increases (Steinman and Shibli-Rahhal, 2019).

For these reasons, bone mineral density should be checked regularly in patients with long-term AN using dual-energy X-ray absorptiometry (DXA). Early prevention and treatment are strongly advised (Loisel et al., 2025). Table 1 shows the main hormonal and metabolic factors that contribute to bone loss in AN.

Table 1. Hormonal and metabolic mechanisms contributing to bone mineral density reduction in anorexia nervosa.

Factor	Level in AN	Physiological action	Consequence of disturbance in AN
Estrogen	Decreased	Inhibits bone resorption by osteoclasts; supports mineralization (Misra and Klibanski, 2014).	Increased bone resorption; decreased bone mass (Misra and Klibanski, 2014).
IGF-1	Decreased	Stimulates osteoblast proliferation and activity; promotes bone formation (Fazeli and Klibanski, 2018).	Reduced osteoblast activity; impaired mineralization and growth (Fazeli and Klibanski, 2018).
Leptin	Decreased	Satiety signal; modulates HPG axis; stimulates osteoblast activity (Stefanakis et al., 2024).	Exacerbated hypogonadism; reduced osteoblast activity; decreased bone

			mineralization (Stefanakis et al., 2024).
Cortisol	Increased	Stress response; regulates glucose metabolism (Treviño et al., 2024).	Inhibits osteoblasts; accelerates osteocyte apoptosis; increases osteoclastic resorption (Treviño et al., 2024).
Peptide YY	Increased	Suppresses appetite (Misra and Klibanski, 2014).	Decreased osteoblast activity; reduced bone mineral density (Misra and Klibanski, 2014).

Cardiovascular System in Anorexia Nervosa

Cardiovascular complications are some of the most serious problems in patients with AN (Springall et al., 2023). The most common sign is sinus bradycardia. It can happen because of increased vagal tone and reduced energy demands of the body (Buchhorn et al., 2021). Patients often also have low blood pressure and a longer QT interval. These changes raise the risk of dangerous heart rhythms and sudden cardiac death (Springall et al., 2023). Some studies have reported changes in the heart muscle of people with AN. These changes mostly include a decrease in heart muscle mass. Atrophy of the left ventricle is also common. Both effects result mainly from a long-term energy deficiency and increased catabolism (Spyra et al., 2025). These changes lower stroke volume and cardiac output. As a result, patients often experience worse exercise tolerance and low blood pressure. Another essential aspect in the pathophysiology of complications is electrolyte disturbances. These come from malnutrition as well as from the misuse of laxatives or self-induced vomiting. One of the most frequent electrolyte abnormalities in this patient group is hypokalemia. It can lead to life-threatening cardiac arrhythmias. Other disturbances that may occur during the disease or in refeeding syndrome include hypocalcemia, hypomagnesemia, and hypophosphatemia. They can also lead to dangerous ventricular arrhythmias (Puckett, 2023).

The clinical outcomes of these problems primarily include an increased risk of sudden cardiac death, more frequent arrhythmias, and heart failure, especially during intensive treatment and possible development of refeeding syndrome (Friars et al., 2023).

Gastrointestinal System in Anorexia Nervosa

AN can affect the entire gastrointestinal tract, what leads to frequently reported gastrointestinal symptoms in patients. Typical symptoms include postprandial fullness, early satiety, heartburn, nausea and vomiting, epigastric pain, bloating, and constipation (Jafar and Morgan, 2021).

One of the disorders that can cause these symptoms is delayed gastric emptying (gastroparesis). Under physiological conditions, proper gastrointestinal motility ensures normal digestion and transit of food. In patients with AN, low vagal tone and reduced stimulation from food causes slow stomach emptying. Motility disorders also involve structural changes in the smooth muscles of the gastrointestinal tract. These changes may further impair motility, although data specific to patients with AN are limited (Jafar and Morgan, 2021). Significant reductions in food intake combined with slowed intestinal transit are responsible for constipation in this patient group. The literature suggests that gastrointestinal motility may improve with treatment and normalization of body weight (Mehler and Brown, 2015).

Loss of fat surrounding the blood vessels can compress the duodenum. This can lead to a rare condition known as superior mesenteric artery (SMA) syndrome. This disorder leads to worsening digestive symptoms, such as bloating, nausea, vomiting, and upper abdominal pain. In severe cases there is a need of surgery (Mehler and Brown, 2015; Kurisu et al., 2021).

In addition to upper and lower gastrointestinal symptoms, many patients with AN have slightly increased aminotransferase levels. The exact cause is not clear. Possible mechanisms include liver cell autophagy caused by starvation, liver ischemia due to poor blood flow, and fatty changes in the liver (Mehler and Brown, 2015).

These gastrointestinal problems in AN significantly lower the quality of life and can make treatment more difficult. They may also increase the risk of medical complications. Because these symptoms are often nonspecific, they can resemble other digestive disorders and lead to misdiagnosis. Early recognition of AN and considering it in the differential diagnosis are important. Suitable dietary, pharmacological, or surgical treatment is necessary to provide complete care for patients (Jafar and Morgan, 2021).

Endocrine System in Anorexia Nervosa

Patients with AN show many hormonal disturbances. Some of these changes help the body adapt to chronic starvation, but they also cause serious complications. Low calorie intake disrupts the hypothalamic–pituitary–gonadal (HPG) axis. It leads to hypogonadotropic hypogonadism. In women, this causes secondary amenorrhea. This means the loss of menstruation after previously regular cycles. It happens mainly because of low estrogen levels.

In men, it leads to low testosterone. As a result, patients may experience reduced muscle mass and lower libido. In both sexes, these hormonal changes decrease fertility and weaken bones. As a result, patients are more likely to develop osteopenia and osteoporosis (Haines, 2023).

The hypothalamic–pituitary–thyroid (HPT) axis is also affected. Patients usually have low triiodothyronine (T3) levels. But thyroxine (T4) and thyroid-stimulating hormone (TSH) often remain normal. These changes serve as an adaptive response. They reduce the basal metabolic rate and help the body conserve energy during starvation (Jada et al., 2021).

Overactivity of the hypothalamic–pituitary–adrenal (HPA) axis is another important finding. It raises cortisol levels as part of the stress response to starvation. Higher cortisol helps release glucose and fatty acids for energy, but when it stays high for a long time, becomes harmful. It breaks down muscle proteins, reduces muscle mass, and weakens bones by increasing resorption and blocking bone formation.

Levels of other metabolic hormones also change. Leptin levels decrease because of low body fat. That worsens HPG axis dysfunction (Stefanakis et al., 2024). Ghrelin and peptide YY are higher in patients with AN. Ghrelin increases hunger, but peptide YY signals fullness, causing a mixed effect on appetite (Misra and Klibanski, 2014). Hormonal changes in AN effect the principal endocrine axes and several metabolic hormones. These changes help the body adapt to a long-term energy deficit. However, they also have harmful effects on reproduction, metabolism, and bone health. Doctors should regularly check and monitor these abnormalities to give an effective care for patients with AN.

Disorders in Other Systems in Anorexia Nervosa

Renal/Urinary System

Different kidney problems can occur in patients with AN. These range from mild decreases in estimated glomerular filtration rate (eGFR) to acute kidney injury (AKI) and chronic kidney disease (CKD). The exact reasons of these variations are not fully understood and may differ between patients. Some patients with AN use laxatives or diuretics excessively. These compensatory behaviors are common in specific subtypes of the disorder. They cause dehydration and electrolyte imbalances. As a result, kidney function may decrease (Stheneur et al., 2024).

Dehydration, low potassium levels (hypokalemia), and low blood volume (hypovolemia) are the leading causes of kidney damage. These factors reduce blood flow to the kidneys and can lead to tubular injury. Some research has shown that eGFR reduction in certain patients may be temporary. It is often caused by loss of muscle mass and dehydration. However, more studies are needed to find out how AN affects kidney function in the long term (Miyahara et al., 2024).

Nervous System

Patients with AN show several structural changes in the brain. MRI studies have shown a reduction in both gray and white matter volumes, enlarged ventricles and greater cerebrospinal fluid volumes. These findings indicate a general loss of brain volume in this group (Titova et al., 2013).

The brain changes in AN are widespread. However, some areas seem to be more affected. These include the hippocampus, anterior cingulate gyrus, and motor cortex (Seitz et al., 2018). Studies using voxel-based morphometry (VBM) have also shown smaller volumes in the hypothalamus, parietal lobe, and basal ganglia. These parts of the brain control appetite, emotions, and motivation.

Research shows that these changes are more common and more severe in younger patients. The developing brain may be more sensitive to long-term starvation, which could explain this difference (Seitz et al., 2018).

Nutritional rehabilitation can reverse many of these brain changes. Several studies have reported partial or even full recovery of brain volume after weight restoration. That shows the changes are at least partly reversible. Recovery seems faster and more complete in younger patients, likely because of higher neural plasticity. However, some research suggests that slight differences in cortical thickness or in the size of specific brain regions may persist even after body weight returns to normal (Kaufmann et al., 2020).

In summary, brain changes in AN are widespread but often affect some areas more than others. Weight restoration can normalize brain volume partly or entirely, but subtle and lasting differences may remain. Early nutritional intervention and treatment are therefore crucial to limit long-term effects on brain structure and function.

Hematopoietic and Immune Systems

Patients with anorexia nervosa usually have blood abnormalities such as anemia, low white blood cell count (leukopenia), and, less frequently, low platelet count (thrombocytopenia). These problems result from severe malnutrition and deficiencies in vitamins and trace elements. Bone marrow studies often show reduced cellularity and a change called gelatinous marrow transformation. This condition is typical of severe malnutrition and is linked to low blood cell counts (De Filippo et al., 2016; Barbin and Oliveira, 2017).

Patients may also have disturbances in the immune system- they may have higher levels of some inflammatory cytokines, such as IL-6 and TNF-α. The CD4/CD8 ratio may change, and patients may have lower neutrophil activity and reduced complement function. These findings suggest complex and sometimes paradoxical immune responses in AN (Gibson and Mehler, 2019).

Most of these blood and immune changes are functional and improve with good nutrition and weight recovery. However, in severe or long-lasting cases, regular monitoring is needed to make sure they fully return to normal. Disturbances in gut microbiota, oxidative stress, and hormonal imbalance may also play a role. It increases inflammation and slows regeneration of bone marrow and immune cells. In clinical care, it is crucial to check complete blood counts and immune markers in patients with severe malnutrition. Nutritional therapy should be the primary focus of treatment. A bone marrow examination is necessary only if results are unusual or fail to improve with proper nutrition (De Filippo et al., 2016; Gibson and Mehler, 2019).

4. CONCLUSION

Anorexia nervosa affects many body systems and can cause serious health problems. Hormonal, metabolic, and organ changes often get better with effective nutrition and weight restoration. However, long-lasting or severe disease may lead to persistent problems, like weak bones and changes in the heart or brain. Early diagnosis and proper treatment are very important to prevent irreversible damage.

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Data and materials availability

All data associated with this study will be available based on reasonable request to the Corresponding Author.

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