A case of anorexia nervosa with multiple medical complications

Sinem Akgül, Orhan Derman

Division of Adolescent Medicine, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey
E-mail: sinemhusnu@yahoo.com
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Anorexia nervosa (AN) is a potentially life-threatening eating disorder characterized by an intense fear of gaining weight and a distorted body image. Although AN is a psychiatric illness, it is also very important from a pediatric perspective, as it can cause major medical complications in every organ system in the growing and developing body. The medical complications of anorexia nervosa may endanger the patient in several ways, and the severity of medical complications may be underestimated. Pediatricians should be aware of the possibility of medical complications in adolescent patients who have an eating disorder and understand that, if not managed correctly, such complications may be fatal. This case report describes the vast number of medical complications that can be observed in an adolescent due to an eating disorder.

Key words: adolescent health, anorexia nervosa, blood, bone mineral density, cardiovascular, medical complication.

Anorexia nervosa (AN) is a potentially life-threatening eating disorder characterized by an intense fear of gaining weight and a distorted body image[1]. According to the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, the diagnostic criteria for AN include three essential elements: 1) persistent restriction of energy intake leading to significantly low body weight 2) intense fear of gaining weight, and 3) disturbance of body image. The diagnostic criteria also designate subtypes. The "restricting" subtype includes patients who restrict food intake, while those with the "binge eating/purging" subtype regularly engage in self-induced vomiting or misuse laxatives, diuretics, or enemas[2].

Eating disorders are the third most common chronic disease in adolescents, and AN has the highest mortality rate of all mental disorders[3]. The medical complications of AN may endanger the patient in several ways, and the severity of medical complications may be underestimated. Although AN is a psychiatric illness, what makes it important from a pediatric perspective is that it can cause major medical complications in every organ system in the growing and developing body[4].

Some of the metabolic changes that occur in these patients are due to the physiological adaptation of energy conserving, which leads to a slowing of the metabolic rate[5].

The two most common causes of mortality in AN patients are suicide and cardiovascular complications[6]. In addition, other medical complications that cause disability may contribute to the mortality of AN, such as hemodynamic, hematogical, gastrointestinal and bone metabolism changes[5].

It is for these reasons that physicians should be alerted to the possibility of medical complications in adolescent patients who have an eating disorder, as a majority are fully reversible with refeeding but may be fatal if not managed correctly. The aim of this paper is to describe an adolescent with anorexia nervosa who presented to the clinic with multiple medical complications.

Case Report

A female patient was referred to the Division of Adolescent Medicine at Hacettepe University Children's Hospital for severe emaciation associated with disturbed body perception, an immense fear of weight gain and syncope. She was the only child of two physicians; her history

revealed she had always been a perfectionist and a high academic achiever. The symptoms, which started at age 18, had been continuing for the past 6 months. The range of food she was eating had progressively decreased, and there was evidence of ritualistic behaviors associated with meals. She was noted to be unhappy, irritable and had withdrawn from social contacts. Throughout this time, there was no evidence of bulimia, self-induced vomiting or abuse of purgatives, although she had a history of excessive exercise. Her history revealed that a month before coming to our hospital, she had been hospitalized for 10 days due to severe abdominal pain and thrombocytopenia; she had an elevated amylase of 134 (U/L) (28-100) and was diagnosed with acute pancreatitis. She was managed conservatively and refused a nasogastric feeding tube; intravenous fluid infusion was initiated. Within 4 days there was improvement in her clinical status, and oral fluids were reintroduced. The patient was referred to the psychiatric ward but refused to comply with the program and was discharged. Against her will, she was brought to our clinic a month later.

When she came to our clinic, she had lost 14 kg in 5 months; her weight had dropped from 50 kg to 36.6 kg; and she had developed amenorrhea at 42 kg.

The patient was diagnosed with an eating disorder that fulfilled the diagnostic criteria for the restricting type of anorexia nervosa as defined in the DSM-IV[2]. Her height was 161 cm and her weight 38 kg, with a BMI of 14.4 kg/m^2. Vital signs revealed hypothermia (35°C), bradycardia (56/min) and hypotension (80/50 mmHg), but no orthostatic pulse or blood pressure changes. On examination she was severely cachectic and dehydrated, her skin was dry, lanugo-type hair was generally observed, she had bruises on her arms and legs, the breasts were atrophic, and her heart sounds were decreased; on admission, abdominal examination was normal.

Initial laboratory evaluation showed anemia and leucopoenia; blood chemistry levels showed elevation of BUN, creatinine, transaminases and amylase. Platelet levels were normal. The patient's values are given in Table I. Because of the decreased heart sounds, an echocardiogram was performed, showing mild pericardial effusion with normal cardiac function. An ECG confirmed the presence of sinus bradycardia (48/min) with a normal corrected QT interval of 0.40 (normal range: 0.35–0.45 s). Nocturnal cardiac monitoring revealed the lowest pulse rate to be 35 beats/min. A bone mineral density evaluation using dual-energy X-ray absorptiometry revealed low bone mineral density both at the proximal femur (Z-score: -2.1) and lumbar spine (Z-score: -1.7).

Due to the instability of vital signs, severe malnutrition, elevation of kidney function tests and refusal to feed, the patient was hospitalized in an internal medicine ward to ensure medical stabilization, proper nutritional restoration and weight gain. Her initial diet consisted predominantly of liquids with a calorie count of 1500 kcal. She refused solids, and enteral fluids were started. The department of psychiatry evaluated the patient, and 50 mg trazaodon was started. The patient was monitored closely with for refeeding syndrome. Nutritional rehabilitation was managed according to daily weight gain and increased 250 kcal if weight gain was less than 200 gr. BUN and creatinine levels improved by the fourth day. The patient was medically stable after two weeks of hospitalization, as hypothermia, hypotension and bradycardia improved, and was referred to the department of psychiatry for further therapy but again refused transfer and was discharged at week 3, at a weight of 40 kg. She is currently being followed as an outpatient.

Discussion

The case report above describes the vast number of medical complications that can be observed due to an eating disorder. What makes this case interesting is that a majority of the medical complications described in the literature were all seen in one patient. A study by Palla and Litt[7] reported a high prevalence of medical instability in AN adolescents, with over half of the patients requiring hospitalization. In that study, bradycardia (94%), hypotension (70%), hypothermia (100%), anemia (32%) and neutropenia (38%) were commonly seen medical problems.

In the initial evaluation of a patient with AN, vital signs play an important role. Physiological instabilities such as bradycardia, hypotension, hypothermia and orthostatic changes in

pulse and blood pressure are indications for hospitalization in an adolescent with an eating disorder[8]. Our patient was unstable for all of the vital signs.

A common, potentially life-threatening situation seen in connection with eating disorders is due to cardiac complications, which have been reported in up to 80% of patients[9]. Sinus bradycardia, seen in our patient, is the most common cardiovascular physical finding and the most common arrhythmia in patients with anorexia nervosa[10]. Other commonly seen cardiac complications are reduced left ventricular mass leading to decreased circulating blood volume, decreased voltage and prolonged QTc, and mitral valve prolapse (MVP)[4]. Echocardiographical changes in AN patients have also been reported in the literature[11]. In our clinic, echocardiograms are not routinely obtained unless clinically indicated. Due to a decrease in heart sounds on examination, an echocardiogram was obtained for our patient and revealed a pericardial effusion. A study by Frölich et al.[12] showed that 10 out of 65 adolescent AN patients evaluated by echocardiography had pericardial effusion with no clinical signs or symptoms of heart failure. The comparison of clinical, laboratory and cardiac parameters between patients with and without pericardial effusion revealed no differences; in eight patients pericardial effusion remitted partly or completely with weight gain. This was also observed in our patient.

Changes in the peripheral blood cell count in patients with AN is frequently observed. Changes seen in the complete blood count may mimic a hematological disease such as leukemia, aplastic anemia or idiopathic thrombocytopenia.[13] Pancytopenia is thought to be due to bone marrow atrophy, as studies show that examination of the bone marrow reveals signs of atrophy in approximately 50% of patients with AN; more rarely, AN patients can additionally suffer from gelatinous bone marrow transformation[14]. As studies show that both hematological and morphological alterations disappear completely and rapidly after sufficient refeeding[14], a bone marrow aspiration was not performed on our patient, and the white cell count slowly increased during refeeding.

Fluid restriction in patients with AN and fluid loss due to purging in patients with bulimia nervosa are frequent occurrences. Fluid restriction in our patient led to dehydration, which caused an increase in creatinine and BUN. Studies show that elevation of BUN occurs in 22% of adolescents with AN[7]. Changes in body fluid homeostasis may lead to life-threatening electrolyte abnormalities.[15]

A few cases of pancreatitis in the setting of anorexia nervosa have been described in the literature[16]. Severe malnutrition is known to be associated with pancreatic injury, through a number of pathogenetic mechanisms.[17] The patient had no history of vomiting, which may also have been a cause of the elevation in amylase level.

Osteopenia is an important long-term complication of AN, and reduced bone mineral density at one or more sites has been shown in approximately 50% of adolescents[18].

Table I. Laboratory Evaluation of the Patient.

	Normal value	Patient's value
Hemoglobin (g/dl)	11.7-15.5	10.6
Hematocrit (%)	34.5-46.3	29
MCV (fL)	80-100	85
RDW (%)	11.7-14.6	16.2
White blood cell count ($x10^3$ ml)	4.1-11.2	2.9
Platelet count ($x10^3$ ml)	159-388	203
ALT U/L	<33	64
AST U/L	<32	44
Pancreatic amylase (U/L)	13-53	66
BUN (mg/dl)	6-20	29
Creatinine (mg/dl)	0.5-0.9	1.2

Current recommendations related to bone densitometry in the pediatric population stem from the First Pediatric Consensus Development Conference on the use and interpretation of bone density studies in children, sponsored by the International Society for Clinical Densitometry[19]. Pediatric osteoporosis is defined as the presence of both a clinically significant fracture history and low bone mineral content (Z-score <2).[20] As the patient had no history of a fracture, she was diagnosed with low bone mineral density. Treatment recommendations for osteopenia associated with anorexia nervosa include restoration of weight with the resumption of menses, calcium (1300–1500 mg/day) and vitamin D supplementation and carefully monitored weight-bearing exercise[21]. The patient was started on calcium and vitamin D supplements.

In conclusion, as seen in this patient, a majority of the medical complications of AN relate to the duration of illness and the nutritional status of the individual. A patient with AN is especially at risk for the development of the described complications, and early intervention is extremely important. Pediatricians' awareness of the medical complications of AN plays a vital role in decreasing morbidity and mortality.

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