CASE REPORT

A Rare Presentation of Anorexia Nervosa as Cardiac Failure in an Adolescent Girl

Namitha Das[1], Sandhya Cherkil[2], Thoduka Rajan John[3]

Abstract

Aim: The aim of the study was to highlight the systemic life-threatening manifestations of anorexia nervosa (AN).

Background: AN is a psychiatric disorder commonly seen among young females with significant systemic manifestations and the highest mortality among psychiatric disorders.

Description: We present the case of an athletic adolescent girl (after obtaining written consent for publication from her father) presenting to the emergency department in shock with cardiac failure. She also had hypoalbuminemia and multiple electrolyte disturbances like hyponatremia, hypokalemia, hypocalcemia, and hypophosphatemia. She underwent extensive evaluation including coronary angiogram and other appropriate tests to rule out malabsorption syndrome. In the absence of organic causes, and as she gave a history of deliberate food restriction, she underwent a psychiatric evaluation. She was found to be a perfectionist with obsessive personality traits. She started to restrict her diet due to fear of gaining weight as she got bullied by her peers in the new school. There was a significant weight loss of almost 14 kg over a period of 6 months. Additionally, she had depressive symptoms like decreased mood, anhedonia, and lack of energy and initiative, concentration difficulties, and an apparent academic decline.

Conclusion: She met the criteria for the diagnosis of AN—restrictive type severe, comorbid with major depressive disorder, and was started on combined psychotherapy and pharmacotherapy with fluoxetine leading to a dramatic clinical improvement.

Clinical significance: The case highlights the life-threatening complications of AN and the role of timely psychiatric interventions in managing these patients.

Keywords: Adolescent girl, Anorexia nervosa, Cardiac failure, Depression.

Indian Journal of Private Psychiatry (2022): 10.5005/jp-journals-10067-0103

Introduction

Anorexia nervosa (AN) is characterized by an intense fear of weight gain and disturbed body image which motivate dietary restrictions and weight loss behaviors as purging or excess physical activity. Point prevalence of AN is estimated to be 0.3–0.5%[1] and lifetime prevalence in women is around 1%2.[2] It is significantly more common among females, with a female-to-male sex ratio of 8:13.[3] There is an obvious increase in AN among adolescents over the past 2–3 decades. Yet higher rates of recovery and lower mortality have been reported in adolescents than in adults (2 vs 5%).[4] AN is associated with a highest rate of mortality among all mental disorders[5] with an estimated crude mortality rate of 5.1 deaths per 1,000 person-years.[6] Most of the deaths are due to starvation-related medical complications, particularly cardiac complications and infections.[7]

Case Description

The patient is a 15-year-old, IX grade student with no relevant medical or psychiatric history and was referred to our Emergency Department with complaints of pedal edema and tiredness of a 2 week duration. Detailed history revealed she had progressive loss of weight and secondary amenorrhea for the last 3 months.

On examination, the child was poorly built and nourished. She had a height of 157 cm (15th centile) and a weight of 37.75 kg (5th centile), suggesting significant malnutrition. She had a body mass index (BMI) of 15.22. The skin was dry and scaly. She looked cachexic and emaciated with gross proximal muscle wasting. She was conscious, drowsy, and obeying simple oral commands. She had bradycardia (pulse rate—44/minute, regular in rhythm) and hypotension (blood pressure—70/46 mm Hg). An electrocardiogram (ECG) showed sinus bradycardia and no ST-T wave changes. Arterial blood gas analysis revealed high anion gap metabolic acidosis with serum lactate of 4.67 mmol/L. In view of hypotension and bradycardia, she was shifted to the pediatric intensive care unit and was started on inotropic support. Echocardiogram showed severe left ventricular dysfunction with an ejection fraction—35–40% and pericardial effusion. Temporary cardiac pacing was done for bradycardia and the rate was set at 80/mt. The troponin I level was elevated. A coronary angiogram done to rule out anomalies of the coronary arteries revealed normal coronaries.

Investigations revealed anemia [Hb—8.5 g/dL (normal—12–15 g/dL)], hypoalbuminemia [serum albumin—2.6 g/dL (normal—3.5–5.5 g/dL)], hypokalemia [serum potassium—2.9 mmol/L

[1,3]Department of Psychiatry, Aster Medcity, Kochi, Kerala, India

[2]Department of Neuropsychology, Aster Medcity, Kochi, Kerala, India

Corresponding Author: Namitha Das, Department of Psychiatry, Aster Medcity, Kochi, Kerala, India, Phone: +91 9995066596, e-mail: dr.namitha.m.das@gmail.com

How to cite this article: Das N, Cherkil S, John TR. A Rare Presentation of Anorexia Nervosa as Cardiac Failure in an Adolescent Girl. Ind J Priv Psychiatry 2022;16(1):42–43.

Source of support: Nil

Conflict of interest: None

(normal—3.5–5.2 mol/L)], hypocalcemia [serum calcium—6.1 mg/dL (normal—8.2–10 mg/dL)], hypomagnesemia [serum magnesium—1.3 mg/dL (normal—1.4–1.8 mg/dL)], and hypophosphatemia [serum phosphate—1.3 mg/dL (normal—3.5–5.5 mg/dL)]. Additional investigations like thyroid function tests and parathormone levels were normal. Virological markers for hepatitis B, hepatitis C, and human immunodeficiency virus (HIV) were negative. Vitamin D level was low [7.5 nmol/L (normal—75–185 nmol/L)]. Other investigations like Mantoux test and Antistreptolysin O titer were normal.

On further probing, there was a history of deliberate food restriction by the child for the past 6 months. Hence, a psychiatrist was called in subsequently. On our evaluation, she was found to be an apparently normal child with an average intellectual functioning, but with a few obsessive–compulsive personality traits as a need for perfectionism and apparent inflexibility. The child was an athlete and was into track events. She had a recent change of school 8 months prior to the onset of symptoms to an institution that focused on sports. The reported weight of the child then was around 52 kg.

In the new school, her peers bullied her for being overweight. Since then, she started restricting her diet as an initial restriction to snacks and energy drinks, which later progressed to avoiding routine meals. She was also noted to have associated depressive symptoms for the past 2–3 months as depressed mood, anhedonia reduced sleep, lack of energy and initiative, easy fatigue, and lack of concentration.

She met the criteria for AN—restrictive type, severe; comorbid with major depressive disorder, moderate, and single episode. The significant systemic manifestations she presented with were considered subsequent to the eating disorder. The cardiac manifestations resulted from nutritional deficiency and multiple electrolyte abnormalities. Amenorrhea was also thought to of nutritional origin. She had ongoing psychosocial stressors as alcohol use disorder in her father, marital discord between parents witnessing violence, lack of social support, and financial stressors. Primary diagnosis of depressive disorder was considered initially. But the child gave a clear-cut history of restricting her diet due to her body image concerns and peer bullying. Since the deliberate attempts to lose weight preceded the depressive symptoms, we confined to the primary diagnosis of AN comorbid with major depressive disorder.

We proceeded with symptomatic and supportive management for systemic complications incorporating a multidisciplinary team. Cardiac status improved with inotropic support and pacemaker, whereas the electrolyte abnormalities and vitamin deficiencies required appropriate supplementation. Nutritional counseling was given to the family and the child. Appropriate diet was introduced and small frequent supervised meal pattern was entertained and serially monitored with the help of diet charting and calorie calculation.

Psychological interventions were mainly focused upon family-based interventions, psychoeducation on AN, and reframing of family interactions. Fluoxetine titrated up to a dose of 20 mg/day was added considering the coexistent depressive episode. She was discharged from the hospital after a period of 2 weeks.

She continued to receive combined psychotherapy and pharmacotherapy as an outpatient. Two months after the discharge, her weight steadily improved to 41 kg, with the restoration of her menstrual periods. After 6 months, her weight increased to 48 kg, with a BMI of 19.5. Academic performance also improved significantly and she migrated with her mother to the Middle East.

Discussion

The case is unique in that the presenting symptom of the patient was a cardiac failure and not weight loss per se. No similar case of AN presenting as cardiac failure has been reported from our country. Despite presenting with life-threatening complications of eating disorder, timely diagnosis and appropriate management involving a multidisciplinary approach resulted in the desired outcome. Although high genetic heritability of almost 28–74%[8] has been reported in prior studies, the family history was negative in our case. Personality traits such as obsessions and perfectionism have been identified as a risk factor for AN9,[9] which was present in our patient. Comorbid mood disorders have been mentioned in three quarters of patients with AN10,[10] particularly depressive disorder which was quite similar to our findings too.

References

1. Smink FR, Van Hoeken D, Hoek HW. Epidemiology of eating disorders: incidence, prevalence and mortality rates. Curr Psychiatry Rep 2012;14(4):406–414. DOI: 10.1007/s11920-012-0282-y.
2. Hay P, Girosi F, Mond J. Prevalence and sociodemographic correlates of DSM-5 eating disorders in the Australian population. J Eat Disord 2015;3(1):19. DOI: 10.1186/s40337-015-0056-0.
3. Steinhausen HC, Jensen CM. Time trends in lifetime incidence rates of first-time diagnosed anorexia nervosa and bulimia nervosa across 16 years in a Danish nationwide psychiatric registry study. Int J Eat Disord 2015;48(7):845–850. DOI: 10.1002/eat.22402.
4. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. Am J Psychiatry 2002;159(8):1284–1293. DOI: 10.1176/appi.ajp.159.8.1284.
5. Harris C, Barraclough B. Excess mortality of mental disorder. Br J Psychiatry 1998;173(1):11–53. DOI: 10.1192/bjp.173.1.11.
6. Arcelus J, Mitchell AJ, Wales J, et al. Mortality rates in patients with anorexia nervosa and other eating disorders: a meta-analysis of 36 studies. Arch Gen Psychiatry 2011;68(7):724–731. DOI: 10.1001/archgenpsychiatry.2011.74.
7. Smink FR, Van Hoeken D, Hoek HW. Epidemiology of eating disorders: incidence, prevalence and mortality rates. Curr Psychiatry Rep 2012;14(4):406–414. DOI: 10.1007/s11920-012-0282-y.
8. Yilmaz Z, Hardaway JA, Bulik CM. Genetics and epigenetics of eating disorders. Adv Genomics Genet 2015;5:131. DOI: 10.2147/AGG.S55776.
9. Kaye WH, Wierenga CE, Bailer UF, et al. Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa. Trends Neurosci 2013;36(2):110–120. DOI: 10.1016/j.tins.2013.01.003.
10. Fernandez-Aranda F, Poyastro Pinheiro A, Tozzi F, et al. Symptom profile of major depressive disorder in women with eating disorders. Aust N Z J Psychiatry 2007;41(1):24–31. DOI: 10.1080/00048670601057718.