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Case Reports

Anorexia Nervosa in a Young, Sportive Male with Severe Bradycardia

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Abstract Sinus bradycardia may be seen commonly in healthy individuals and athletes without any significance. It may also occur in cardiovascular and systemic diseases. Sinus bradycardia is a common clinical sign in patients with anorexia nervosa. However, it is not usual to experience a patient with severe bradycardia who was diagnosed to have anorexia nervosa after being treated with a permanent pacemaker. We presented a 16-year-old, sportive boy who was initially admitted to the Department of Paediatric Cardiology with severe bradycardia, treated with permanent cardiac pacemaker and then diagnosed with anorexia nervosa. As per this case, physicians should consider anorexia nervosa in the aetiology of severe bradycardia, especially in young patients on excessive exercise and dieting, and refer these patients to the mental health professionals.

Key words Adolescent; Anorexia nervosa; Bradycardia; Exercise; Sick sinus syndrome

Introduction

Sinus bradycardia may be seen commonly in healthy individuals and athletes without any significance. It may also occur in cardiovascular and systemic diseases such as myxoedema.[1] Clinicians usually encounter sinus bradycardia in patients with anorexia nervosa (AN) in their practice. However, it is not common to manage a patient with severe bradycardia who was diagnosed to have AN after being treated with a permanent pacemaker. AN is an uncommon psychiatric illness in males.[2] Previous research has established a relation between eating disorders and excessive exercising especially in males, as well.[3] In this case report, a 16-year-old, sportive boy who was admitted to the hospital with severe bradycardia and later diagnosed with AN is presented. Therefore, physicians should refer young patients with severe bradycardia on excessive exercise and dieting to mental health professionals for further evaluation.

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Case Report

A 16-year-old boy was evaluated by a physician for nausea and eating less, and then referred to our Department of Paediatric Cardiology with bradycardia. The patient did not have syncope, however he complained of exercise intolerance and mild tiredness. He described dizziness, shortness of breath and symptoms compatible with near-syncope with exercise. He did not have a history of drug use. His parents and siblings were healthy without any history of cardiac disease. His motor, cognitive, and

psychosocial developments were normal and he had otherwise been healthy.

On physical examination the only pathological finding was bradycardia (30 bpm). His weight, height and BMI were 62 kg, 180 cm, 19 kg/m^2, respectively. ECG revealed sinus bradycardia. Echocardiography pointed out a mild mitral insufficiency and a normal ejection fraction rate. The 24 hour Holter ECG recording was obtained using a three channel ambulatory electrocardiographic monitor. Holter recording showed findings that might be compatible with sick sinus syndrome; such as sinus bradycardia with supraventricular extrasystoles and sinus arrest, escape coronary sinus rhythm, and 22 sinus pauses greater than 2.5 seconds following spontaneous termination of the supraventricular extrasystoles (Figure 1). The longest sinus pause recorded was 3.7 seconds. The heart rate was 52 bpm in resting condition and increased to a maximum of 137 bpm (67% of the presumed heart rate) during exercise testing. The laboratory results of the patient on the first admission are given on Table 1. Complete blood count, erythrocyte sedimentation rate, CRP, biochemical markers of liver and kidney functions, TSH, free T4 and serum electrolyte levels were normal. Free T3 level was low. Checking thyroid function tests two weeks later was planned by the Department of Paediatric Endocrinology. Laboratory investigations demonstrated normal cardiac enzyme levels. Serological markers for autoimmune myocarditis were normal. Brain natriuretic peptide, ACTH and cortisol levels were within normal limits. Hospitalisation and further investigation of the patient was decided. The heart rate of the patient was recorded as 20-40 bpm in the hospitalisation period, however blood pressure never dropped to less than 85/50 mmHg. Since bradycardia continued throughout the day, Holter recording showed frequent pauses with the longest one being 3.7 seconds, and the patient experienced dizziness, shortness of breath and near-syncope symptoms with exercise, decision to implant a permanent cardiac pacemaker was made.

Because his mother mentioned that he had been eating less and exercising, and the observations in the inpatient service suggested selective eating, a psychiatric consultation was decided. Psychiatric examination revealed that the patient did not accept his concern about gaining weight, but picked out foods, exercised routinely and lost 20 kg throughout the last two years. He was resistant to talk about his eating pattern and related issues. Associated with being a male, hiding of the patient of his anxiety about gaining weight, lack of emaciation, the resistance and low insight of patient about his symptoms, gradual occurrence of weight loss over a long period of time and his history of regular exercise and sport might have hindered the precise diagnosis of anorexia nervosa. Psychiatric follow-up after hospital discharge was advised to clarify the diagnosis of anorexia nervosa. However, the patient refused psychiatric follow-up and did not show up at the Department of Child and Adolescent Psychiatry for his appointment. He was later admitted to the Department of Paediatric Endocrinology with complaints of muscle spasms in his left leg and losing excessive weight during the one month period following hospital discharge. His weight was 52 kg and BMI was 16 kg/m^2. There were deteriorations in his liver, kidney, thyroid functions and electrolyte levels (Table 1, second admission). Euthyroid sick syndrome was considered. The patient was again evaluated in the Department of Child and Adolescent Psychiatry. It was learned that he had complaint of being teased by his classmates for excessive eating and being obese at the end of elementary school. At high school, he had begun to play basketball and thus lost much of his weight. This together with positive and confirming comments about his appearance from his colleagues made him continue exercising and dieting. He gradually had lost 20 kg in the last two years. For a few months before his initial admission to the Department of Paediatric Cardiology, he had been eating less and exercising and lost at least two to three kilograms. Following discharge from the hospital, on losing 10 kg in one month due to excessive dieting and increased physical exercise, he was diagnosed with AN and re-hospitalised for four weeks for medical and psychiatric treatment of this disorder. The diagnostic criteria for anorexia nervosa in this 16 years old boy included significant weight loss (nearly 13 kg in a few months) leading to a body weight less than 85% of that expected (his body weight was 52 kg, expected body weight for his age and height was 77 kg), fear of gaining weight, dieting and exercising excessively even though he was underweight, and denial of the seriousness of his low body weight. Hypokalaemia (K: 2.89 mEq/l, normal range: 3.4-4.7), anaemia (haemoglobin 8.3 g/dl, normal range: 13.6-17.2) and peripheral oedema were experienced during treatment. ECG of patient showed no U wave and hypokalaemia was treated by repletion of patient with oral potassium. Serum iron, unsaturated iron binding capacity, total iron binding capacity and ferritin levels were 46 ug/dl (normal range: 59-158), 97 ug/dl (normal range: 110-370), 143 ug/ml (normal range: 228-448) and 845.2 ng/ml (normal range: 30-400), respectively. Bone marrow aspiration was normal and fecal occult blood test was negative. Anaemia was thought to be related to excessive water intake of patient

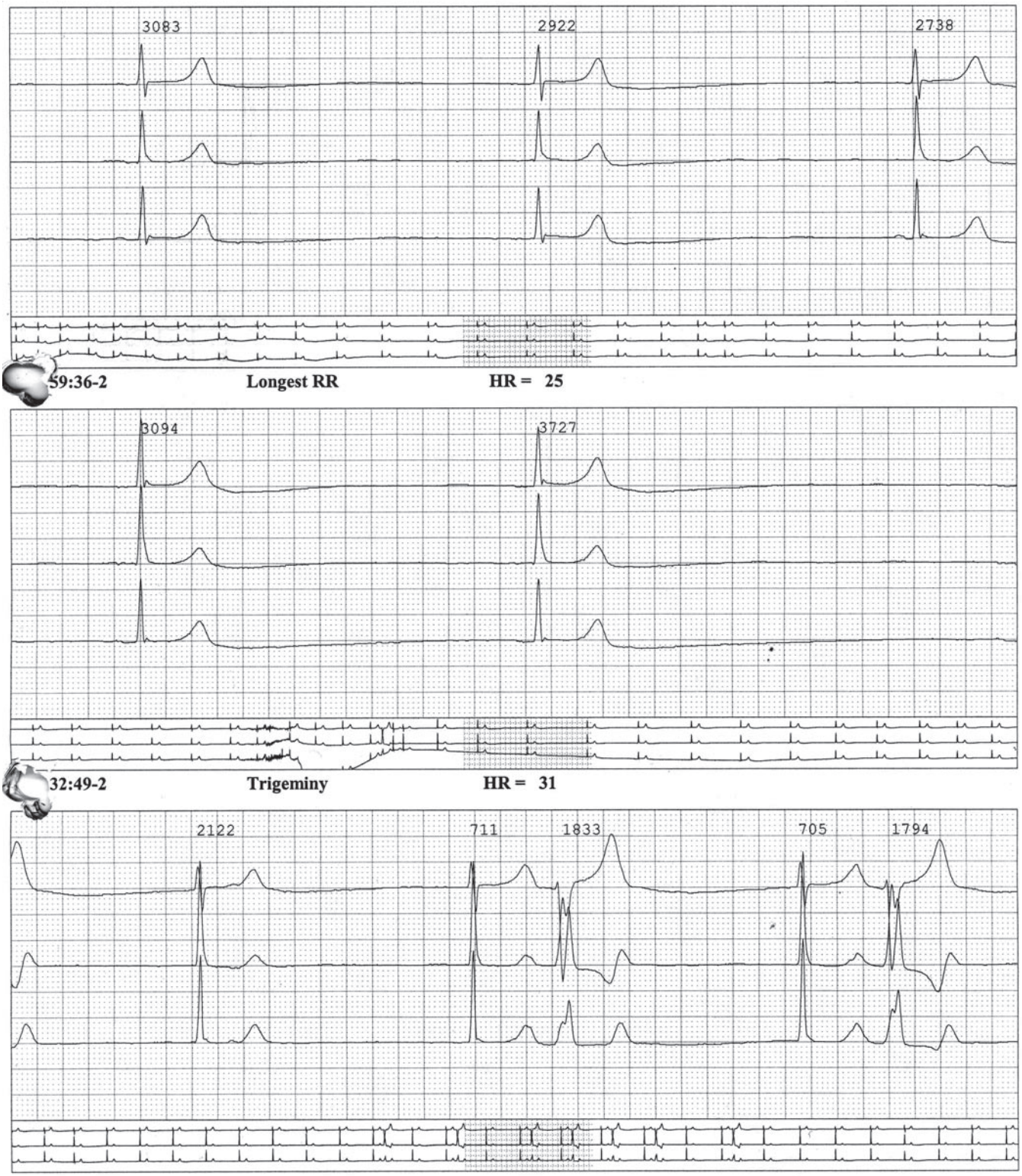

Figure 1 A sample from the Holter recording displaying the heart rate decreasing to 25 bpm with pauses.

Table 1 Laboratory results of the patient on the first and the second admissions

Laboratory tests	First admission	Second admission	Normal range of tests
Complete blood count			
Haemoglobin	14.6 g/dl	13.8 g/dl	13.6-17.2 g/dl
Red blood cell count (RBC)	4.95 $x10^6$ /µl	4.58 $x10^6$ /µl	4.38-5.77 $x10^6$ /µl
White blood cell count (WBC)	7.0 $x10^3$ /µl	6.0 $x10^3$ /µl	4.3-10.3 $x10^3$/µl
Thrombocyte count	232 $x10^3$ /µl	225 $x10^3$ /µl	156-373 $x10^3$/µl
Erythrocyte sedimentation rate	2 mm/h	2 mm/h	0-20 mm/h
CRP	<0.1 mg/dl	0.129 mg/dl	0-0.8 mg/dl
Liver function tests			
ALT	19 U/l	203 U/l	< 41 U/l
AST	24 U/l	248 U/l	< 37 U/l
GGT	15.5 U/l	26.3 U/l	8-61 U/l
Kidney function tests			
BUN	22 mg/dl	51.9 mg/dl	5-18 mg/dl
Creatinine	1.00 mg/dl	0.84 mg/dl	0.7-1.2 mg/dl
Albumin	4.87 g/dl	3.93 g/dl	3.2-4.5 g/dl
Thyroid function tests			
TSH	2.39 uIU/ml	2.7 uIU/ml	0.27-4.2 uIU/ml
Free T4	11.72 pmol/l	9.53 pmol/l	12-22 pmol/l
Free T3	1.7 pmol/l	0.985 pmol/l	3.1-6.8 pmol/l
Brain natriuretic peptide	12.6 pg/ml		0-100 pg/ml
ACTH	15.5 pg/ml		0-46 pg/ml
Cortisol (morning)	18.6 µg/dl		5-25 µg/dl
Cardiac enzyme levels			
CK/MB	4.85 ng/ml		0-4.94 ng/ml
Myoglobin	50.45 mg/ml		28-72 mg/ml
Troponin T	<0.01 mg/ml		0-0.1 mg/ml
Electrolyte levels			
Na	144 mEq/l	136 mEq/l	138-145 mEq/l
K	4.55 mEq/l	4.48 mEq/l	3.4-4.7 mEq/l
Ca	9.05 mg/dl	8.61 mg/dl	8.6-10.2 mg/dl
PO_4	3.71 mg/dl	1.31 mg/dl	2.7-4.5 mg/dl

(4 l/day) and starvation. Although peripheral oedema may be related to electrolyte imbalances and rapid re-feeding in these patients, it could be explained by hypoalbuminaemia (total protein 4.66 g/dl, normal range: 6-8; albumin 2.59 g/dl, normal range: 3.2-4.5) and excessive water intake of this patient. Anaemia and hypoalbuminaemia were handled by slow oral feeding of patient and restriction of fluid intake. Blood phosphorus level became normal by adding supplemental phosphorus to his treatment during hospitalisation. The patient was discharged from the hospital on stabilisation of his medical condition. Twice weekly visit to the Department of Child and Adolescent Psychiatry was scheduled. Psycho/pharmacotherapy of the patient has been continuing for twelve months. His weight and BMI are 78 kg and 24 kg/m^2. His liver, kidney, thyroid functions, and serum electrolyte levels are normal. He is also on follow up at the Department of Paediatric Cardiology, Adolescent Unit and other related outpatient clinics. Pacemaker of patient was removed four months ago and ECG during exercise testing and Holter monitoring are normal.

Discussion

A sinus heart rate less than 60 bpm is considered to be sinus bradycardia. It is a common finding in athletes and healthy young individuals especially during sleep.[1] However, the heart rate of 20-40 bpm and Holter monitoring findings of our patient should not be observed in healthy people, even if they exercise excessively. Sinus bradycardia may also be caused by sick sinus syndrome, which is a generalised abnormality of cardiac impulse formation that may be related to an intrinsic disease of the sinus node itself such as infiltrative, collagen vascular and infectious diseases or several extrinsic causes.[4] The syndrome includes signs and symptoms related to cerebral hypoperfusion in association with ECG findings such as sinus bradycardia, sinus arrest, sinoatrial block, or alternating episodes of bradycardia and tachycardia. Initial Holter examination of our patient was consistent with sick sinus syndrome. Extrinsic causes of sick sinus syndrome include toxic or pharmacologic exposure, electrolyte imbalance, hypothermia, hypoxia, hypothyroidism, increased intracranial pressure, and excessive vagal tone[4]. Intrinsic diseases of the sinus node and most of these extrinsic factors were not present in our patient. Mild decrease in free T3 associated with normal free T4 and TSH levels at the admission of our patient was due to euthyroid sick syndrome. Later, thyroid function was impaired as weight loss of the patient increased and became normal when the patient gained weight. Increased vagal tone and decreased thyroid hormones due to excessive weight loss and consequent alteration in sympathovagal balance might be the extrinsic causes of sick sinus syndrome in this patient.

AN is a kind of eating disorder in which people refuse to maintain a minimally normal weight for age and height, fear gaining weight and misinterpret their body weight and shape. The onset of the disorder is most commonly at puberty, with a decrease in younger generations. Although the frequency of eating disorders in males is increasing, they are still considered as female disorders. Incidence studies on males report that the incidence of AN among males is below 1.0 in 100,000 persons per year.[2] This disparity in the prevalence was reported to be related to differences in psychological maturation/identity formation, exposure to the pressure to be slim between the genders during these processes.[5] However, rapid sociocultural changes and slim body shape promoted by westernisation was accountable for the increasing incidence of eating disorders among male and female youngsters. Premorbid obesity which was experienced in our patient was presented as a risk factor for eating disorders in men.[6] Despite the differences in prevalence, no prominent gender differences in the symptoms of eating disorders, like the course and outcome of these illnesses, with the exception of greater weight concern among females and excessive exercising in men were reported.[3,7]

Cardiac problems such as prolongation of the QT interval and bradycardia are common in patients with AN and are usually benign and reversible after nutrition therapy.[8] Although severe bradycardia may be observed, first admission with this symptom is unusual in these patients. Hypophosphatemia which is usually a part of refeeding syndrome was found to be associated with cardiac failure, cardiac arrhythmia and cardiorespiratory arrest in patients with anorexia nervosa.[9] However, severe bradycardia was detected before falling of blood phosphorus level of our patient. Cardiac vagal hyperactivity and the consequent alteration in sympathovagal balance appear to be a mechanism of bradycardia, increased cardiovascular mortality and sudden death in patients with AN.[10]

Since AN is an uncommon psychiatric disorder in males and bradycardia is a common symptom in individuals regularly exercising, it may be reasonable to be unsure about the diagnosis of AN at first evaluation in young, sportive males who seem healthy and gradually lose weight over a long period of time. Additionally, patients with AN may sometimes have a strong resistance to treatment and may seem not to have an apparent anxiety about gaining weight, as per this case. Therefore, it is important to keep in mind AN in the differential diagnosis of severe bradycardia in young patients practicing excessive physical exercise and dieting. Physicians should refer these patients to mental health professionals for further evaluation. A longer duration of in-patient watching to take a more detailed history and to observe eating behaviors under close cardiac monitoring would have revealed features of an eating disorder in this patient. Prolonged in-patient observation might be warranted before a decision for permanent pacemaker insertion in these kinds of patients. Furthermore, patients with AN and symptomatic bradycardia necessitate in-patient treatment under close cardiac monitoring and complete bed rest during the re-feeding process. A permanent pacemaker may not be necessary in all patients.

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