CASE REPORT

Bradycardia in anorexia nervosa

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SUMMARY

We present a case of a 24-year-old woman initially referred for a permanent pacemaker for symptomatic sinus bradycardia. Further consultations revealed significant weight loss and subsequent psychiatric review confirmed a diagnosis of anorexia nervosa.

BACKGROUND

Symptomatic bradycardia is a common symptom seen in a cardiology outpatient setting so the presentation of this woman was not unusual. However, the reason behind her bradycardia and the implications behind having a permanent pacemaker

Figure 1 Twelve-lead ECG prior to diagnosis and treatment.

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Figure 2 Twelve-lead ECG 1 year after diagnosis and treatment.

inserted would have potentially led to psychological as well as physical repercussions.

CASE PRESENTATION

A 24-year-old woman with abdominal pain and sinus bradycardia (heart rate 38 bpm on admission) was initially admitted under the surgical team and discharged following a reassuring CT scan of her abdomen and a diagnosis of constipation as symptoms resolved.

Over the following 12 months, the patient presented with a history of symptomatic bradycardia and 7.6 kg weight loss. On

examination, an incidental finding of Holmes-Aide pupil was found, and a normal brain MRI confirmed no sinister cause for this. There was no family history to note and the only medical history was a malignant melanoma excised and previous investigation for polycystic ovarian syndrome. However, transabdominal and transvaginal ultrasounds were reassuring and all hormone levels were within normal ranges.

INVESTIGATIONS

The patient underwent a number of blood tests, all of which were unremarkable, including thyroid function tests, serum ACE

Figure 3 Graph showing weight against time. BMI, body mass index.

for sarcoid, Lyme disease serology and porphyria. A 24 h tape showed rates between 31 and 101 bpm with a mean of 49 bpm, throughout which she had symptomatic episodes. Transthoracic echocardiography showed a structurally normal heart. Cardiac MRI revealed a small area of fibrosis into the left ventricular outflow tract from the basal septum; this was felt to be a normal variant or to represent a small area of discrete fibrosis around the atrioventricular node. During her tilt testing, the patient was hypotensive throughout, with a starting blood pressure of 64/49 mm Hg. After initial tilting, she felt dizzy and her blood pressure rose to 83/62 mm Hg with a heart rate 42 bpm (figure 1). There were several episodes of sudden drop in blood pressure, but no high degree of atrioventricular block.

TREATMENT

A discussion was held with the patient on the possibility of the insertion of a permanent pacemaker. However, the drop in her weight was marked and the possible diagnosis of anorexia nervosa was raised.

Psychiatric review confirmed no previous personality or eating disorders and a new diagnosis of anorexia nervosa characterised by extensive weight loss, the measures taken to maintain the weight loss as well as the central psychopathology of body dysmorphia. The patient now awaits treatment under the psychotherapy team, with no current plans for a permanent pacemaker.

OUTCOME AND FOLLOW-UP

Follow-up subsequent to psychiatric input has shown marked improvement in symptoms and a resting heart rate of 68 bpm (figures 2 and 3).

DISCUSSION

Anorexia nervosa is a condition characterised by sustained deliberate weight loss or failure to gain expected weight with varying degrees of undernutrition/malnutrition and secondary endocrine or metabolic change.¹ It is a life-threatening disorder, with a significant risk for sudden death (5–20%) due to severe cardiovascular complications.²

Up to 87% of patients suffer with cardiovascular compromise early in anorexia nervosa. Malnutrition causes cellular changes within cardiac muscle, leading to structural, functional or

electrocardiological complications. Commonly, cardiac rhythm disturbances occur; the majority develop sinus bradycardia as an adaptation to the hypometabolic state.³ Up to 85% may suffer with hypotension less than 90/60 mm Hg, usually secondary to chronic volume depletion and orthostatic changes resulting in frequent episodes of dizziness and occasional frank syncope. Structurally, the heart is atrophic in patients with eating disorders, this may relate to longstanding hypovolaemia. Patients have low cardiac output and increased peripheral vascular resistance despite the presence of hypotension.⁴ Starvation leads to low body weight, which may result in atrophic peripheral muscles, resulting in decreased venous return to the heart.^{5 6} PR interval prolongation is also a commonly recognised ECG change, often related to underlying structural or electrolyte abnormality.⁷

Commonly, sinus and nodal bradycardia are found in patients with anorexia nervosa with no evidence of other arrhythmias. There has been a clear positive correlation found between heart rate and body mass index, while a negative correlation exists between QT_c and body mass index.² Although many patients remain asymptomatic despite profound bradycardia, it can be a precursor of a potentially lethal arrhythmia.

Significant increases have been found in supine and standing heart rate, and in supine systolic blood pressure after weight gain.^{8 9} Unless there is evidence of severe symptomatic bradycardia or high-grade atrioventricular block is present, no temporary or permanent pacemaker implantation is indicated.¹⁰

Cardiovascular complications are common in patients with anorexia nervosa; sinus bradycardia, however, is the most common cardiovascular physical finding and the most common arrhythmia in

Learning points

- ▶ Continuity of care with the same consultant led to an early diagnosis of anorexia.
- ▶ Regular monitoring of the patient's weight as an outpatient was vital to the diagnosis in this patient.
- ▶ Thorough investigation and treatment of symptomatic bradycardia can extend to beyond cardiological intervention.
- ▶ Early referral and input from the psychiatry team was essential in successful treatment.

this group of patients. An increase in vagal tone and decreased metabolism of energy utilisation due to low calorie intake is thought to lead to physiological adaptation of bradycardia.^{11 12}

Contributors HS wrote the manuscript and involved in the literature review. JT gave consultant support and guidance.

Competing interests None declared.

Patient consent Obtained.

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