

## Management of sinus node dysfunction with junctional escape rhythm in a case of anorexia nervosa

Anoreksiya nervozalı bir olguda sinüs düğümü disfonksiyonu ve kavşak kaçış ritminin tedavisi

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We report on a 17-year-old female patient with anorexia nervosa (AN), who developed electrocardiographic abnormalities consisting of sinus arrest and junctional escape rhythm. She had complaints of general fatigue, lethargy, sweating, and nausea resulting from voluntary weight loss of more than 30 kg during the past six months. Her body weight was 40 kg, heart rate was 44 bpm, and blood pressure was 90/50 mmHg, and she had signs of dehydration. The electrocardiogram showed a junctional rhythm at 44 bpm, no P wave, QRS width of 60 msec, QT of 440 msec, QTc of 400 msec, and QU of 600 msec. Laboratory findings were normal except for hypokalemia. Management consisted of a multidisciplinary team approach with a re-feeding program together with psychiatric and dietary assistance. Due to the absence of atrioventricular node conduction disease and/or structural heart disease, pacemaker implantation was not considered. This management plan resulted in a successful outcome with return to sinus rhythm within 24 hours of admission.

**Key words:** Anorexia nervosa/complications; bradycardia/etiology; electrocardiography; sinoatrial node.

Anorexia nervosa (AN) is associated with the highest mortality rate among all psychiatric disorders. Much of this mortality is related to cardiovascular complications.<sup>[1]</sup> Sinus node dysfunction and bradycardic effects of severe weight loss are well-known in AN.<sup>[2,3]</sup> Cardiac structural and functional abnormalities induced by AN are often reversible in young adolescents through appropriate management.<sup>[4,5]</sup>

We report on a young female patient who developed electrocardiographic abnormalities consisting of sinus arrest and junctional escape rhythm.

Bu yazıda, sinüs durması ve kavşak kaçış ritmi gibi elektrokardiyografik anormallikler gelişen 17 yaşında, anoreksiya nervozalı bir kadın hasta sunuldu. Başvuru yakınmaları, son altı ayda 30 kilogramdan fazla istemli kilo kaybından kaynaklanan genel halsizlik, uyuşukluk, terleme ve kusma idi. Vücut ağırlığı 40 kg, kalp hızı 44 atım/dk, kan basıncı 90/50 mmHg ve su kaybı bulguları olan hastanın elektrokardiyogramında 44 atım/dk'da kavşak ritmi izlendi; P dalgası yoktu, QRS genişliği 60 msn, QT 440 msn, QTc 400 msn, ve QU 600 msn idi. Laboratuvar bulguları hipokalemi dışında normal sınırlardaydı. Hastanın tedavisine, multidisipliner ekip yaklaşımıyla psikiyatrik yardım ve diyet yardımını da içeren yeni bir beslenme programı oluşturularak başlandı. Atriyoventriküler düğüm ileti bozukluğu ve/veya yapısal kalp hastalığı olmadığından, geçici veya kalıcı kalp pili yerleştirilmesine gerek duyulmadı. Bu tedavi planı başarılı sonuç verdi ve yatıştan sonra 24 saat içinde hasta sinüs ritmine döndü.

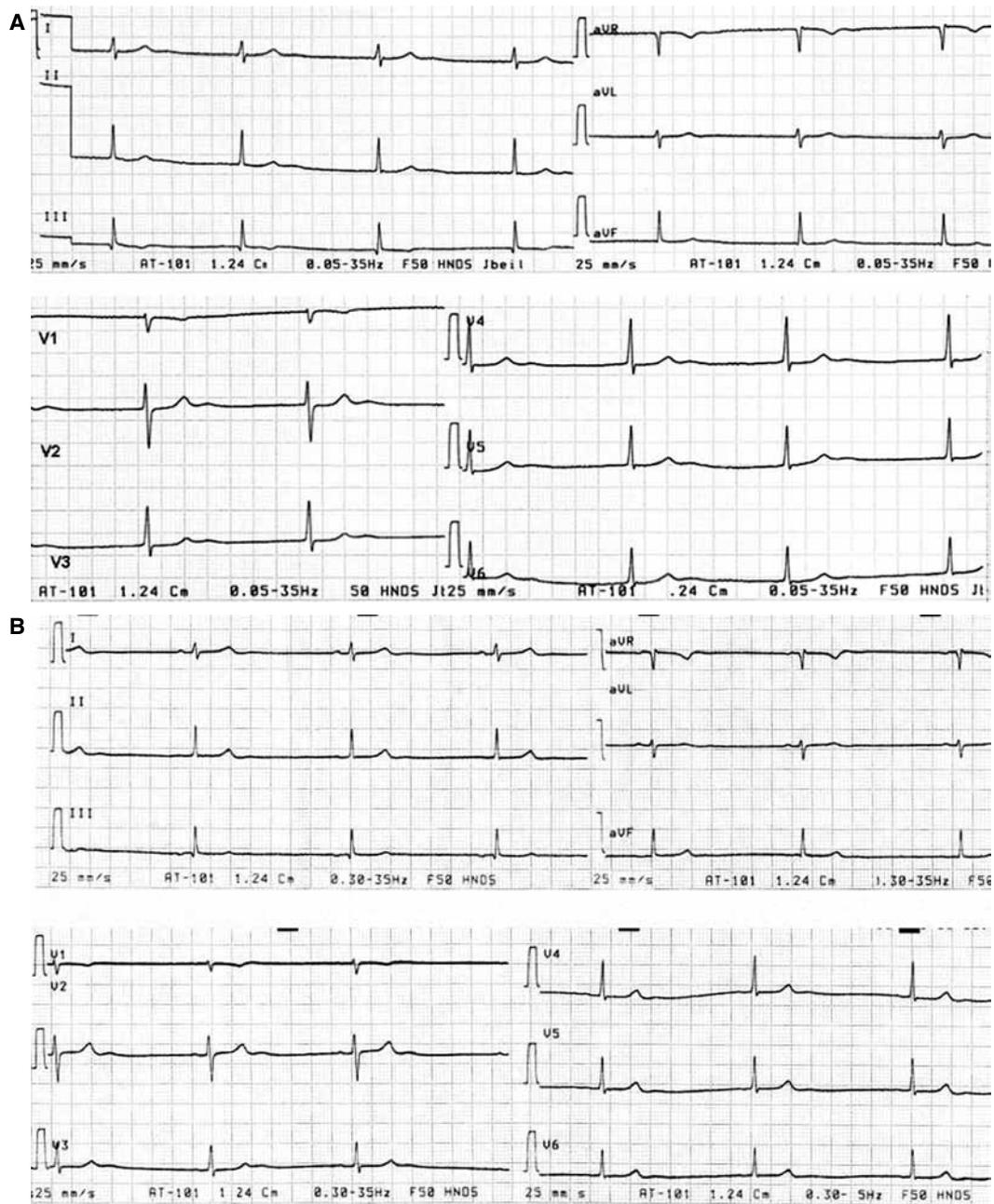
**Anahtar sözcükler:** Anoreksiya nervoza/komplikasyon; bradikardi/etiyoloji; elektrokardiyografi; sinoatriyal düğüm.

### CASE REPORT

A 17-year-old female patient presented to the emergency room with complaints of general fatigue, lethargy, sweating, and nausea. She had no past medical history. Initial history taking revealed voluntary weight loss of more than 30 kg during the past six months, loss of appetite, repetitive nausea, vomiting, and irregular menstrual cycles. Emergency psychiatric consultation confirmed the diagnosis of AN. On presentation, body weight was 40 kg, heart rate was 44 bpm, body temperature was 36 °C, body mass index (BMI) was 13.8

Received: October 25, 2009 Accepted: January 6, 2010

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**Figure 1.** Electrocardiograms recorded (A) at presentation and (B) 24 hours later.

kg/m<sup>2</sup>, and blood pressure was 90/50 mmHg, with signs of dehydration. The electrocardiogram showed a junctional rhythm at 44 bpm, no P wave, QRS width of 60 msec, QT of 440 msec, QTc of 400 msec, U waves, and QU of 600 msec (Fig. 1a).

The chest X-ray showed a small cardiac silhouette with normal lungs, without evidence for pathological findings such as infiltrate, mass, or pleural effusion. Bone density on the chest X-ray was noted to be within the normal range. Cardiac echogram showed normal pericardium, normal left ventricular ejection frac-

tion, and normal cardiac dimensions (when compared to BMI); mitral early diastolic peak (E wave) was 75 cm/sec and there was no late diastolic peak (A wave). Laboratory findings were within normal ranges except for hypokalemia (3.3 meq/l). Of note, serum sodium was 140 mmol/l, hemoglobin was 13.2 g/dl, folic acid was 13.3 ng/ml, TSH was 3.76 mIU/l, and FT4 was 12.3 pmol/l.

Beside prompt correction of hypokalemia and adequate rehydration, psychiatric evaluation was requested and a re-feeding program based on a

dietary consultation was initiated. Due to the absence of documented atrioventricular node conduction disease and/or structural heart disease, no temporary or permanent pacemaker implantation was undertaken. The patient reverted to sinus rhythm 24 hours after admission, the P waves were of low voltage probably related to a sinoatrial dysfunction occurring in this setting (Fig. 1b). Monitoring showed the persistence of bradycardia at 45-50 bpm. During 10 days of hospitalization, serial electrocardiograms remained identical to the one shown in Fig. 1b. Regular and close medical follow-up was scheduled prior to discharge and family support was advised for the patient's long-term plan of care.

## DISCUSSION

Anorexia nervosa is associated with the highest mortality rate among all psychiatric disorders. Much of this mortality is related to cardiovascular complications such as arrhythmias.<sup>[1]</sup> Structurally, the heart of AN patients is atrophic and this may be related to long-standing hypovolemia.<sup>[1]</sup>

Cardiac structural and functional abnormalities induced by AN are often reversible in young adolescents after a re-feeding program and weight gain.<sup>[4,5]</sup> Unless severe symptomatic bradycardia or high-grade atrioventricular block are present, no temporary or permanent pacemaker implantation is indicated.<sup>[2]</sup>

Sinus node dysfunction and bradycardic effects of severe weight loss are well-known in AN and they result from increased vagal tone associated with a low calorie-protein diet.<sup>[2,3]</sup> Excessive vagal activity is also partly explained by enhanced baroreflex sensitivity.<sup>[3]</sup> The classical bradycardia of AN is usually responsive

to vagolytic drugs, otherwise an intrinsic sinus node dysfunction must be suspected.<sup>[6]</sup>

In conclusion, patients with AN presenting with symptomatic sinus node dysfunction do not need permanent pacemaker implantation, unless they have severe intrinsic automatism or conduction disease. The management of dysrhythmia in this setting is best undertaken with an appropriate re-feeding and psychological rehabilitation program. The follow-up program must be defined before discharge and recovery is a long process that can take months to years.

**Acknowledgements.** The author would like to gratefully acknowledge the help of Dr P. Eddé for participating in editing this paper.

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