

Anorexia Nervosa: a Disease With Potentially Lethal Repercussions on the Heart

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Anorexia nervosa is a disease which mainly affects adolescents, and is more common in girls, with a ratio of 10:1.1 Eating disorders (anorexia and bulimia) affect about 5 million persons each year in the United States and their prevalence is increasing. Around 3% of young women in western countries have an eating disorder.^{1,2} Anorexia nervosa is a potentially fatal disease with a mortality rate of 0.56% per year, which is more than 12 times as high as that of young women in the general population. One third of all deaths in patients with anorexia nervosa are estimated to be due to cardiac causes, mainly sudden death,³⁻⁵ although few data are available. Furthermore, the fact that many of these patients are not under medical control leads to the risk of potentially fatal complications sometimes being underestimated.

The study by Vázquez et al,⁶ which analyzes cardiac disorders in a series of 30 consecutive patients, reveals that up to 40% of patients have a prolonged corrected QT interval. This prolongation, together with the presence of bradycardia, is a risk factor for ventricular arrhythmia due to torsade de pointes polymorphic ventricular tachycardia, and thus constitutes a risk factor for sudden death. The authors also found that heart dimensions and heart mass were lower in patients than in healthy women. Similar findings have been reported by others.^{5,7-9}

Cardiac disorders in young women with anorexia nervosa can generally be divided into structural abnormalities or rhythm disorders, although they are probably related. The rhythm disorders seen in the patients in this study, though, were those associated with a risk of sudden death, whereas structural abnormalities rarely lead to clinical repercussions of heart failure.

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Rhythm disorders in anorexia nervosa

Bradycardia, as described by Vázquez et al, is one of the most common findings in patients with anorexia nervosa,^{1,10} and it is in fact one of the factors which contribute to the risk of polymorphic ventricular tachycardia. Bradycardia is also associated with increased vagal tone, which can be shown by an increased heart rate variability, and which is reversible on recovery of weight.¹⁰ Like Vázquez et al, several authors have also noted prolongation of the QT interval. Cooke et al⁹ analyzed the QT interval in 41 adult patients and showed a prolonged QT interval in 15% of the patients, with sudden death occurring in two. Others, on the other hand, have reported a normal corrected QT interval when electrolytes were kept within normal ranges. As Vázquez et al do not report the state of the ions, the prolonged QT interval may be secondary to hypopotassemia or hypomagnesemia. Indeed, in a series of 31 consecutive adolescents with anorexia nervosa studied in our center,¹⁰ no patient was found to have a prolonged corrected QT interval; however, electrolyte imbalances had been corrected prior to obtaining the electrocardiogram and Holter. It is therefore probable that the prolonged corrected QT interval seen by some authors is in fact secondary to ion disorders. The risk of severe ventricular arrhythmia could thus be prevented by strict control of the hypopotassemia, hypomagnesemia or hypocalcemia.

From the practical point of view, prevention of severe ventricular arrhythmias and sudden death in patients with anorexia nervosa should be based on strict control of factors leading to ventricular arrhythmia. Admission criteria for a patient with anorexia nervosa should not depend solely on degree of malnutrition and treatment response, but should also take into account the presence of any alarm signal, such as syncope, extreme bradycardia, prolonged QT interval or electrolyte imbalance. The routine protocol for patients with anorexia nervosa at our institution includes an electrocardiogram, a full ionogram with measurements of sodium, potassium, calcium and magnesium, as well as a complete biochemical workup and exhaustive

hormone study to rule out endocrine disorders. If severe bradycardia is detected (heart rate lower than 45 bpm), the patient is admitted for overnight electrocardiographic monitoring. The patient is also admitted if ion disorders are detected. Patients with purgative anorexia (with self-induced vomiting or abuse of laxatives) undergo more frequent ionograms.

Echocardiographic parameters

In their series Vázquez et al report reduced heart dimensions and ventricular mass compared to the control group. Other authors have described similar changes, with reduced wall thickness or cardiac output. To what extent structural alterations, with their associated reduced cardiac output, are responsible for symptoms such as asthenia is difficult to establish because these patients generally tend to deny any symptoms of their disease. Several authors, though, have demonstrated reversibility of these changes after recovering weight. In our study of 31 adolescents who were re-evaluated after weight recovery,¹⁰ there was a significant increase in cardiac dimensions, ventricular mass and cardiac output. This demonstrates that during initial phases of the disease the structural changes noted are partly reversible, at least in adolescent patients. However, whether values return to what the patients would have had if they had not suffered the disease is unknown. There may be permanent subclinical effects, as seen in bone mass for example, if the duration of the disease is prolonged.

In summary, the study by Vázquez et al reminds us that anorexia nervosa is a disease which can lead to

potentially fatal cardiac complications. Patients therefore require not only adequate psychotherapeutic treatment but also careful follow-up to prevent these potentially severe complications.

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