Cardiovascular and Metabolic Science www.ancam.org.mx

Chapter 16

Vol. 33 Supplement 5
October-December2022

doi: 10.35366/108057

Frequent cardiac arrhythmias in women

Arritmias cardiacas más frecuentes en la mujer

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INTRODUCTION

Gender is a determining factor in arrhythmias' incidence, etiology, and clinical presentation.[1] These cover a broad spectrum of clinical manifestations, ranging from benign extrasystoles on the electrocardiogram (ECG) to arrhythmias that can represent a significant clinical threat. The predominant factors that determine the differences in women are sex hormones.[2]

Epidemiology of the most frequent arrhythmias in women

The general population's prevalence of supraventricular tachycardia (SVT) is 2.25/1,000 persons, and the incidence is 35/100,000 persons/year. Women have twice the risk of SVT than men. Atrioventricular node reentrant tachycardia (AVNRT) is the most treated type of SVT after atrial fibrillation (AF), followed by atrial flutter and atrioventricular reentrant tachycardia (AVRT). Women are more likely to suffer from AVNRT than men (70:30). It has been suggested that there is a relationship between the menstrual cycle and that the episodes are more frequent during pregnancy, especially in women with pre-existing SVT.[3]

Inappropriate sinus tachycardia (IST)

It generally affects women between the ages of 15 and 45, with a prevalence four times higher than men. The demographics of patients affected by IST can be confused by its association with psychological disorders; therefore, this syndrome is not well recognized. Some evidence links IST to hormonal changes associated with age, pregnancy, and menstruation in women.[4]

Acquired long QT syndrome (LQTS)

The female sex has been associated with a higher risk of torsade de pointes (TdP).

Acquired LQTS is clinically more common than congenital LQTS, associated with female gender, other anomalies, and the use of drugs that prolong the QT interval (QTI). Therefore, class IA and III antiarrhythmic drugs have a higher risk of TdP in women than in men.[1]

Ventricular arrhythmias of the right ventricular outflow tract (VA RVOT)

RVOT occurs more frequently in women. Men have a higher incidence of ventricular arrhythmias of the LV outflow tract, of the tricuspid and mitral rings, and of the ventricular septum compared to women.[5]

Sex-specific triggers were described in a short report of 47 RVOT patients. In 20 of 34 (59%) female patients reported initiation of RVOT with recognized states of hormonal flux (premenstrual, gestational, perimenopausal, and coincident with the administration of contraceptive pills).[1]

Conduction system diseases

Sex differences have been described in various bradyarrhythmia. Women have a higher incidence of sinus node dysfunction, and men

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How to cite: Encarnación-Roa C, Gonzáles-Luna AC, Núñez-Ayala E. Frequent cardiac arrhythmias in women. Cardiovasc Metab Sci. 2022; 33 (s5): s474-s476. https://dx.doi.org/10.35366/108057

have a higher incidence of atrioventricular node dysfunction. Women, on average, are older at the time of permanent pacemaker implantation, receive more single-chamber pacemakers, and have higher complication rates (higher incidence of pocket hematoma and pneumothorax), regardless of age and type of device implanted.[5]

Mechanisms and pathophysiology

Two mechanisms explaining the differences between the sexes in the incidence and mechanisms of different types of cardiac arrhythmias have been proposed: 1) differences in autonomic tone and 2) hormonal effects on the expression or function of ion channels or a combination of both.

Mechanisms

Autonomous regulation: it plays a relevant role in arrhythmogenesis. Premenopausal adult women have faster heart rates (HR) than men. Spectral analyses of HR variability in women have reported an increase in high-frequency components associated with vagal modulation of the sinoatrial node. The ratio between the low-frequency and high-frequency components, which expresses the sympathovagal balance, is consequently lower in women.

Effects of sex hormones: differences in the expression of ion channel subunits and the modulation of their function between the sexes have been described with longer duration of the action potential of female myocytes and differences in ventricular repolarization. Progesterone and testosterone shorten the ventricular action potential, while estrogens lengthen the action potential and have a proarrhythmic effect.[2] More dispersion of Ca^{+2} currents and, therefore, greater susceptibility to early post-depolarizations have been reported. Activity triggered by the increased risk of drug induced TdP and sudden death in patients with congenital LQTS has also been reported.[1]

This combined effect translates into a more pronounced parasympathetic activity in women, with gonadal steroids determining the differences, due to their different effects on the cell membrane's ion channels.[6]

These differences may be why AVNRT and acquired LQTS are more frequent in women and why women with ischemia experience less ventricular tachyarrhythmia than men.

TREATMENT

Vagal maneuvers can be performed safely in women with SVT. Adenosine is recommended as a first-line drug when vagal activation fails to stop SVT. Synchronized cardioversion is recommended in hemodynamically unstable arrhythmias or when drug therapy is ineffective.[6]

A diagnostic electrophysiological study (EPS) may be offered in women with symptoms suggestive of SVT even before the arrhythmia is documented. In documented SVT, the same access to catheter ablation should be provided. In women with a previous electrophysiology study without inducible arrhythmias, a second electrophysiology study scheduled in the first few days of the menstrual cycle may be recommended to render the arrhythmia inducible. Catheter ablation should be offered equally to women and men with symptomatic ventricular arrhythmias.[1]

Beta-blocker therapy is recommended as class I in all women with LQTS. Women treated with class IA or class III antiarrhythmic drugs (AADs) should be aware of the risk and symptoms associated with TdP. ECG monitoring should be considered at the onset of AAF to monitor HR and QTI. It should be contraindicated in women with prolonged QTI (> 500 ms) or significant SA or AV node disease without a permanent pacemaker. Amiodarone should be considered in the setting of life-threatening arrhythmias or when other therapies with better safety profiles have failed.[5]

Women who meet guideline indications for pacemaker, defibrillator, or cardiac resynchronization therapy (CRT) therapy should receive it; however, they may have a lower all-cause mortality benefit from defibrillation as primary prevention, sex-specific differences in therapy benefits should not be considered for risk stratification. Women have a high probability of responding to CRT.[1]

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