Case Reports in Women's Health 24 (2019) e00153

Contents lists available at ScienceDirect

Case Reports in Women's Health

journal homepage: www.elsevier.com/locate/crwh

Invited Editorial

Supraventricular tachycardia and the menstrual cycle

Keywords:
Arrhythmia
Sex hormones
Menstrual cycle
Diagnostics

Over the menstrual period, various conditions, such as migraine, asthma and epilepsy, can be exacerbated by the variations in hormone levels [1]. Female sex hormones have a multitude of effects on the cardiovascular system through several mechanisms, ranging from estradiol-induced vasodilation associated with fluctuations in blood pressure to increased elasticity of aortic smooth muscle cells after exposure to higher levels of estradiol and progesterone [2,3]. Although it is known that both estrogen and progesterone have electrophysiological properties, only a few studies have assessed the effect of the menstrual cycle on arrhythmia. In general, women less often develop ventricular arrhythmia than men; however, they have an increased risk of long-QT-induced torsades de pointes and long-QT-associated drug-induced ventricular arrhythmia [4]. Clinically significant variations in QT interval duration have been linked to the menstrual cycle and fluctuations in sex hormone levels, so an association between phases of the menstrual cycle and the occurrence of ventricular tachycardia would be quite possible [5]. However, to our knowledge, no studies have been published on this topic.

This editorial gives a brief overview of the current knowledge of the effect of changing ovarian hormone levels across the menstrual cycle in women with supraventricular tachycardia (SVT) and its possible diagnostic and therapeutic consequences.

The menstrual cycle can be divided in several phases. The first phase, starting with the first day of menstruation, is known as the follicular phase, when estradiol and progesterone levels are low. Mid-cycle, at ovulation, estradiol shows a sharp surge but progesterone is still low. Finally, in the premenstrual or luteal phase, estradiol and progesterone levels are initially high but then decrease (so levels are low again at the next menstruation).

Both estradiol and progesterone influence heart rate in women, via several pathways. It is reported that estradiol has a direct negative chronotropic effect by suppressing T-type calcium channels and by its influence on the cardiac autonomic nervous system [6,7]. Progesterone, on the other hand, activates the renin-angiotensin system, leading to fluid retention and consequently an increase in circulating blood volume and increased heart rate [8].

A study of 49 healthy premenopausal women with a regular menstrual cycle found a significantly lower average heart rate (−2.33 bpm), but an increased heart rate variability, during the follicular (menstrual) phase compared with the luteal (premenstrual) phase. This implies that estradiol and progesterone fluctuations affect cardiac autonomic regulation [9].

Based on these results, one might expect most episodes of tachycardia to occur during the premenstrual luteal phase. Indeed, in a study of 26 premenopausal women with a regular menstrual cycle and paroxysmal SVT, who underwent weekly 48 -h ambulatory electrocardiographic monitoring during one menstrual cycle, significantly more episodes of paroxysmal SVT were recorded in the premenstrual days than in the menstrual days. Moreover, an inverse correlation between frequency and duration of episodes of paroxysmal SVT and estradiol level was found [10]. In a study of 42 premenopausal women with symptomatic paroxysmal SVT, 40 % of the patients reported a clustering of SVT episodes in the premenstrual period [11]. Moreover, this study showed that the cyclic increased sensitivity for episodes of SVT has clinical consequences for diagnostic testing. All women underwent diagnostic electrophysiological testing, including provocation with isoproterenol, which was performed to induce episodes of SVT. First, electrophysiological tests were performed mid-cycle. Six patients in whom episodes of SVT were not inducible during the initial test underwent a second electrophysiological test in the premenstrual period. All six women who initially had a negative electrophysiological mid-cycle test had induced episodes of paroxysmal SVT during the repeat test in the premenstrual phase. Therefore, performing electrophysiological procedures in women with SVT in their premenstrual period may lead to more accurate diagnosis [11].

The influence of the menstrual cycle on the occurrence of arrhythmia has been investigated in only a few studies, with small samples. As indicated above, taking the menstrual cycle into account when scheduling diagnostic tests such as ambulatory electrocardiographic (ECG) monitoring and electrophysiological tests might lead to more accurate diagnoses. It is recommended that ECG monitoring be performed during the premenstrual phase, as the occurrence of arrhythmias is highest when estradiol levels are low and progesterone levels are high. If premenstrual-related arrhythmia is diagnosed, it would be interesting to investigate whether treatment can be tailored around the menstrual phase. Research is needed to evaluate whether adjusting the dosage of antiarrhythmic drugs in different phases of the menstrual cycle will lead to

https://doi.org/10.1016/j.crwh.2019.e00153

less symptomatic arrhythmia in premenopausal women. Another option would be to learn more about the effect of continuous estradiol treatment by testing if the oral contraceptive pill (without the often-used week of interruption) decreases the number of episodes of SVT. However, the most exciting direction will be to reveal the precise pathways by which sex hormones affect SVT and to modulate these in a more sophisticated fashion, which might lead to new antiarrhythmic treatment options, not only for premenopausal women but also for postmenopausal women and men.

Contributors

M.M. Schreuder contributed to the literature search and wrote the first draft of the manuscript.

M. Sunamura contributed to the literature search and revision of the manuscript.

J.E. Roeters van Lennep contributed to the literature search and revision of the manuscript.

Conflict of interest

The author has no conflict of interest regarding the publication of this editorial.

Funding

No funding was sought or secured in relation to this editorial.

Provenance and peer review

This editorial was commissioned and not externally peer reviewed.

References

[1] A.M. Case, R.L. Reid, Effects of the menstrual cycle on medical disorders, Arch. Intern. Med. 158 (1998) 1405–1412.
[2] E.J. Adkisson, D.P. Casey, D.T. Beck, A.N. Gurovich, J.S. Martin, R.W. Braith, Central, peripheral and resistance arterial reactivity: fluctuates during the phases of the menstrual cycle, Exp. Biol. Med. (Maywood) 235 (2010) 111–118.
[3] R.W. Chow, D.J. Handelsman, Ng M.K. Minireview, Rapid actions of sex steroids in the endothelium, Endocrinology 151 (2010) 2411–2422.
[4] B.M. Gowd, P.D. Thompson, Effect of female sex on cardiac arrhythmias, Cardiol. Rev. 20 (2012) 297–303.
[5] J.E. Salem, J. Alexandre, A. Bachelot, C. Funck-Brentano, Influence of steroid hormones on ventricular repolarization, Pharmacol. Ther. 167 (2016) 38–47.
[6] T.M. Saleh, B.J. Connell, Role of oestrogen in the central regulation of autonomic function, Clin. Exp. Pharmacol. Physiol. 34 (2007) 827–832.
[7] F. Marni, Y. Wang, M. Morishima, T. Shimaoka, T. Uchino, M. Zheng, T. Kaku, K. Ono, 17 beta-estradiol modulates expression of low-voltage-activated Ca(V)3.2 T-type calcium channel via extracellularly regulated kinase pathway in cardiomyocytes, Endocrinology 150 (2009) 879–888.
[8] N.S. Stachenfeld, H.S. Taylor, Progesterone increases plasma volume independent of estradiol, J. Appl. Physiol. 2005 (98) (1985) 1991–1997.
[9] P.S. McKinley, A.R. King, P.A. Shapiro, I. Slavov, Y. Fang, I.S. Chen, L.D. Jamner, R.P. Sloan, The impact of menstrual cycle phase on cardiac autonomic regulation, Psychophysiology 46 (2009) 904–911.
[10] G.M. Rosano, F. Leonardo, P.M. Sarrel, C.M. Beale, F. De Luca, P. Collins, Cyclical variation in paroxysmal supraventricular tachycardia in women, Lancet 347 (1996) 786–788.
[11] R.J. Myerburg, M.M. Cox, A. Interian Jr., R. Mitrani, I. Girgis, J. Dylewski, A. Castellanos, Cycling of inducibility of paroxysmal supraventricular tachycardia in women and its implications for timing of electrophysiologic procedures, Am. J. Cardiol. 83 (1999) 1049–1054.

M.M. Schreuder
Department of Internal medicine, Vascular Medicine, Erasmus Medical Centre, Rotterdam, the Netherlands

M. Sunamura
Capri Cardiac Rehabilitation Center, Department of Cardiology Franciscus, Rotterdam, the Netherlands

J.E. Roeters van Lennep*
Department of Internal medicine, Vascular Medicine, Erasmus Medical Centre, Rotterdam, the Netherlands

* Corresponding author at: Dr. Molenwaterplein 40, 3015 GD, Rotterdam, Postbus 2040, 3000 CA, Rotterdam, Room: Rg-531, the Netherlands.
E-mail address: j.roetersvanlennep@erasmusmc.nl (J.E. Roeters van Lennep)

30 September 2019