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Ventricular couplets but not isolated extrasystoles induce a "female" sustained, supraventricular tachycardia. What is the mechanism?

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Abstract This is the case of a 28-year-old woman with sustained supraventricular tachycardia induced only by monomorphic ventricular couplets but not by isolated extrasystoles. The careful examination of electrocardiogram (ECG) was able to identify the mechanism of the tachycardia in the absence of electrophysiologic investigation. The tachycardia resulted sustained and symptomatic only during periods.

Keywords: Supraventricular tachycardia; Ventricular couplets

Case presentation

A 28-year-old woman was admitted to outpatient clinic because of palpitations particularly prolonged and not well tolerated only during menstrual cycle. Physical examination as well as resting 12-lead ECG and transthoracic echocardiogram were normal. During 12-lead 24-h Holter monitoring, frequent and isolated ventricular premature beats (VPBs) at fixed coupling interval of 400 ms were recorded with inferior axis deviation on frontal plane, q wave in L1-aVL, rS in V1 sometimes organized in couplets with similar morphology, coupling interval and QRS complex duration (R'R" interval = 320 ms; QRS duration = 160 ms). The isolated VPBs seemed to be retrogradely conducted with R-P interval of 280 ms (Fig. 1A). Interestingly, just and only following every couplets we observed brief episodes of self-terminating supraventricular tachycardia 150 bpm (four or five consecutive premature P waves negative in L2-L3-aVF-V3 → V6, positive in aVR-aVL, isodiphasic in V1-V2) with either 1:1 or 2:1 A-V conduction associated with narrow QRS complex or isolated beats showing right bundle branch morphology with left axis deviation (Fig. 1A, B). The tachycardia occurred with a retrograde jump of conduction (prolonged R-P interval of 400 ms) and a P-R interval of 220 ms (Fig. 1A, B); then the RP-PR intervals showed slight and variable shortening. The spontaneous termination of tachycardia occurred with either QRS complex or a blocked P wave (Fig. 1A, B).

A 3-lead 24 h ECG Holter monitoring (Fig. 2A, B, C) was then recorded during menstrual cycle and the same phenomenon was observed but self-terminating supraventricular tachycardia lasted sometimes longer (three minutes) showing 1:1 A-V conduction with a variable rate from 150 to 180 bpm associated with dizziness and thoracic pain. The first beats of this faster tachycardia showed aberrant QRS complex (Fig. 2A, B). Finally, as previously mentioned the P-R and R-P intervals slightly changed and were related to tachycardia rate. The patient refused the electrophysiologic investigation.

Commentary

What is the mechanism of tachycardia? The induction and termination of the tachycardia suggest an underlying reentry mechanism. An AV reentry tachycardia induced by ventricular beats and due to a concealed accessory pathway is ruled out because the ventricle is not required to sustain the tachycardia and the tachycardia goes on during AV block (1–2). The presumed mechanisms are: 1) A slow-slow AVNRT with either 1:1 or 2:1 antegrade conduction is strongly suggested by: induction by VPB s, initiation dependent on critical R-P interval longer than 60 ms, prolonged P-R interval, caudocephalic retrograde atrial activation (1–2). 2) Slow retrograde conduction of VPBs through the His bundle-AV node inducing an intra-atrial reentry tachycardia cannot be excluded. The different rate and a slightly different P-R interval related to the tachycardia rate are criteria but not crucial to infer the diagnosis of intra-atrial reentrant tachycardia [1]. The variations of P-R and R-P intervals are due to different antegrade and retrograde conduction.

Why was the tachycardia just induced only following every ventricular couplet and never following isolated ventricular

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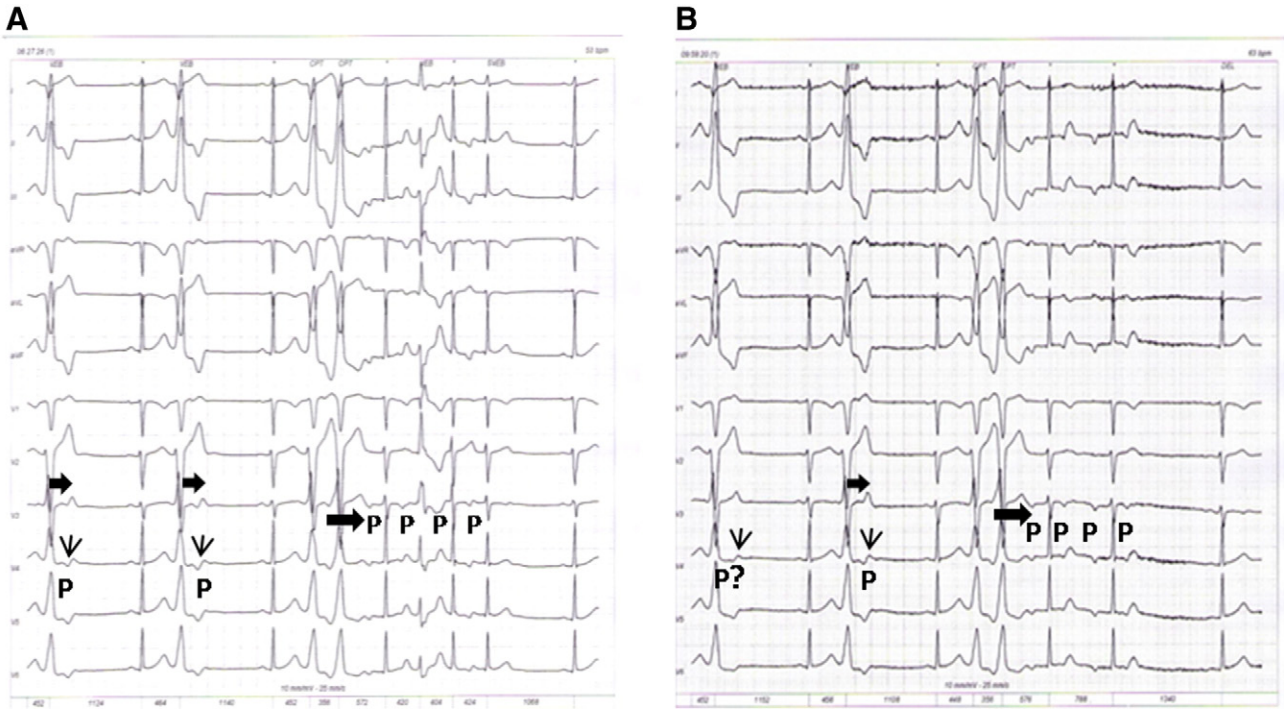

Fig. 1. Twelve-lead 24 h Holter monitoring out of periods. Thick horizontal arrows depict retrograde conduction, thin vertical arrows retrograde P waves. Following each isolated VPB there is apparently a retrograde P wave (R-P interval 280 ms) except the first VPB of panel B (retrograde concealed conduction not reaching the atrium?). Following the ventricular couplets a brief run of supraventricular tachycardia is induced by a jump of retrograde conduction (R-P interval 400 ms) with 1:1 A-V conduction and some beats showing right bundle branch block morphology and left axis deviation (A) or with 2:1 A-V conduction (B). Note that the interruption of tachycardia occurs either after a QRS complex (A) or a P wave (B).

extrasystoles? *What is the mechanism of QRS aberrancy of some beats and of the AV block*? *Why are the rate and the duration of tachycardia different out of and during periods*, *respectively*? The first ventricular beat of couplets is conducted retrogradely through an AV nodal pathway (pathway 1) sometimes reaching the atrium (Fig. 1A) or probably with retrograde concealed conduction not reaching the atrium (Fig. 1B), therefore the second ventricular beat finds the AV nodal pathway (pathway 1) refractory and is conducted to the atrium through a second slow AV nodal pathway (pathway 2) with a jump inducing an atypical slow–slow AVNRT tachycardia (Fig. 1A, B). Sometimes, the second beat of couplet is followed by a relatively long HH interval prolonging the refractory period (Ashman phenomenon) [2] of the HPS (see gray bars of Fig. 3 panel A and ladder diagrams for details). The AVNRT beat finds the HPS at the limit of its refractoriness resulting in a right bundle aberrancy and left anterior hemiblock (see* of Fig. 3A and ladder diagrams for details). The tachycardia finally terminates on the retrograde slow pathway. Then, the VPB couplet reinitiates the tachycardia and the induced HH interval is in this case even longer resulting in an increased refractoriness of the HPS, with an infra-hissian block of the atypical AVNRT beat (see** of Fig. 3B and ladder diagrams for details). Out of the menstrual cycle the sympathetic drive is reduced provoking a lower tachycardia rate and 2:1 AV block at level of the His bundle (See Fig. 3 and ladder diagrams), while during periods the sympathetic drive rises inducing faster tachycardia rate as well as 1:1 AVN-His bundle conduction (Fig. 2A, B). Alternatively, the slow retrograde conduction of the second ventricular beat is able to induce the tachycardia because it finds one of an atrial pathway refractory and the slow conduction through another one allows the induction of an intra-atrial reentry tachycardia [1–3]. Independently of the underlying mechanism, the morphology of P wave during tachycardia allows to identify the atrial breakthrough in the right septal atrial annulus near the coronary sinus ostium [4].This case confirms and stress that a careful examination of the electrocardiogram is able to infer the mechanism of an unusual supraventricular tachyarrhythmia in the absence of intracavitary electrophysiologic investigation. The pathophysiologic mechanisms by which the sex influences the occurrence and cycle length of tachyarrhythmias are far from being understood and some investigators hypothesized hormonal effects on the expression and function of the ion channels and gender differences in autonomic tone [5,6].

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