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Assessment of the clinical presentation and factors affecting the natural history of typical atrioventricular nodal reentrant tachycardia

Ocena przebiegu klinicznego i czynników wpływających na historię naturalną typowego częstoskurczu węzłowego nawrotnego

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Abstract

Introduction. Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular tachyarrhythmia; however, its clinical presentation has not been investigated in detail. The aim of the study was to assess the clinical presentation and identify the potential factors significantly affecting the natural history of AVNRT in patients undergoing RF ablation.

Material and methods. Ninety-two consecutive patients with invasive diagnosis of typical AVNRT who underwent RF ablation were included. All patients were actively screened for the presence of structural heart disease (SHD). The retrospective, arrhythmia focused interviews regarding its clinical history, affecting factors and treatment were conducted. The medical records and tachycardia ECG tracings data were included, if available.

Results. Seventy-five percent of patients had evidence of the arrhythmia exacerbation. No relationship between the coronary (66%) or brain (33%) hypoperfusion symptoms and clinical factors was found. Time to the tachycardia diagnosis was significantly longer in women (13 ± 11 vs 7 ± 8 years, p = 0.006). Pharmacological AVNRT management was not consistent with the ESC guidelines. The arrhythmia symptoms were occurring later if SHD was present (44 ± 13 vs 30 ± 5 years, p = 0.001). The longer AVNRT cycle length (CL), modified by the age when symptoms appeared and the presence of impaired myocardial contractility, the longer time to the arrhythmia diagnosis and exacerbation. Mitral valve prolapse (MVP) was more common in female patients compared with the general population and was found to be the factor delaying AVNRT diagnosis.

Conclusions. In the majority of patients AVNRT exacerbation takes place. The coronary and brain hypoperfusion symptoms that patients often experience are nonspecific. There is a significant delay in the arrhythmia diagnosis, especially in women. SHD, AVNRT CL and MVP were identified as factors that significantly modify the arrhythmia clinical presentation.

Key words: atrioventricular nodal reentrant tachycardia; structural heart disease, arrhythmia exacerbation

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Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular tachyarrhythmia, affecting 2–5/1000 adults [1, 2] and accounting for 50–60% of all regular tachyarrhytmias [3, 4]. Most researchers focus on the assessment of electrophysiological characteristics of AVNRT, whereas its clinical presentation has not been thoroughly investigated.

The aim of the study was to assess the clinical presentation and identify the potential factors significantly affecting the natural history of AVNRT in patients undergoing RF ablation.

Material and methods

Overall, 92 consecutive patients (59 women and 33 men aged 46.6 ± 17.6 years) with diagnosis of typical AVNRT confirmed with electrophysiology study (EPS) who underwent one-stage RF ablation in the years 2006–2009 were included in the study. The study received the approval of local Ethics Committee and informed written consent has been obtained from all participants prior to the study. All patients were actively screened for the presence of structural heart disease (SHD) based on medical history and physical examination, 12-lead electrocardiography (ECG) and transthoracic echocardiography (TTE), followed by additional cardiac diagnostic tests, if needed. Table 1 presents clinical entities that are categorised as SHD. The retrospective, arrhythmia focused interviews regarding its clinical history, affecting factors and treatment were conducted. The medical records were also included, if available. Pain or discomfort in the chest were considered as symptoms indicative of coronary hypoperfusion, whereas brain hypoperfusion was suspected if following symptoms were present: syncope, blurred vision, scotomas, mumbling speech, muscle weakness in extremities, and tingling/ /sensory disturbances in the face. Arrhythmia exacerbation, i.e. the moment from which arrhythmia episodes became more frequent and/or of longer duration, was established based on patient's subjective assessment. Mean frequency, duration and tolerance of arrhythmia episodes were also assessed by patients. The analysis included 12-lead ECG recordings with tachyarrhythmia performed prior to EPS (91% of patients). The assessment of 2003 ESC guidelines implementation [1] in terms of emergency and long-term therapies was limited to patients in whom the symptoms of arrhythmia occurred after these guidelines were issued (93% of patients).

Statistical analysis

Analyses were performed using *STATISTICA 8™* software (StatSoft Inc.). Data are presented as arithmetic means and standard deviation (± SD). Distribution normality was established with Shapiro-Wilk test. Between-group

Table 1. Prevalence of structural heart disease and most common concomitant diseases

No	Structural heart disease	Number of patients
1	Arterial hypertension complicated by myocardial hypertrophy*	11 (42%)
2	Coronary heart disease** without previous myocardial infarction	8 (31%)
3	Coronary heart disease** with previous myocardial infarction	6 (19%)
4	Significant aortic valve disease***	4 (16%)
5	Patent foramen ovale with high left-to-right shunt****	2 (8%)
6	Dilated cardiomyopathy	2 (8%)
7	Chronic heart failure NYHA class ≥ II	2 (8%)
8	Significant mitral valve disease***	2 (8%)
9	Sinus node dysfunction demanding pacemaker implantation	1 (4%)
10	Haemodynamically significant interatrial septal defect****	1 (4%)
	The most common concomitant diseases besides structural heart disease	
1	Mitral valve prolapse without valvular insufficiency	26 (45%)
2	Stage I hypertension according to WHO	15 (26%)
3	Thyroid diseases	8 (14%)
4	Atrial fibrillation and/or flutter	8 (14%)

*Based on left ventricular mass index; **confirmed by angiography; ***insufficiency or stenosis assessed by echocardiographist as moderate or severe; ****based on transoesophageal echocardiography; NYHA — New York Heart Association; WHO — World Health Organization

comparisons were performed using U Mann-Whitney, Wilcoxon, Pearson's chi-square and McNemara's chi-square tests. Correlation analysis was performed based on Pearson's linear correlation. P values < 0.05 were considered statistically significant. Statistical power was estimated for every test, assuming that acceptable value is ≥ 0.8.

Results

Onset of arrhythmia symptoms

First symptoms of arrhythmia occurred at the age of 34 ± 18 years (median ± interquartile range 51 ± 21 years); the variable distribution followed non-normal pattern ($p < 0.0001$), with marked peak in the second decade of life (30%). In 84% of patients arrhythmia occurred before the age of 40. It has been observed that arrhythmia symptoms occurred significantly later in patients with structural heart disease (44 ± 13 vs 30 ± 5 years, $p = 0.001$) and significant moderate negative correlation was observed with left ventricular ejection fraction (LVEF) ($p < 0.0001$).

Structural heart disease and other concomitant diseases

SHD was diagnosed in 28% of patients and in 30% of these patients 2 or more clinical entities were present. Sixty-three percent of patients have at least one of 11 concomitant diseases (Tab. 1).

Symptoms and provoking factors of AVNRT

Patients reported 23 different symptoms experienced during arrhythmia episode (Tab. 2). No relations were shown between possible symptoms of coronary (66% of patients) or brain (33% of patients) hypoperfusion and tachycardia cycle length (CL), presence of SHD and patients' age ($p > 0.05$). Overall, 89% of patients have identified 13 factors provoking arrhythmia (Tab. 2), but in 75% of them there were also idiopathic episodes of arrhythmia that occurred mostly during the day (100%), while performing usual daily activities (86%) or resting (34%), and more rarely during the night (36%). No patient noticed any differences between arrhythmia episodes in terms of heart rate.

Arrhythmia exacerbation

Arrhythmia exacerbation was observed in 75% of patients in whom arrhythmia episodes became significantly more frequent (0.3 ± 0.4/months vs 8 ± 19/months, $p < 0.0001$) and significantly longer (38 ± 53 minutes vs 254 ± 365 minutes, $p < 0.0001$), whereas in 25% of patients the frequency and duration of arrhythmia episodes remained unchanged (3 ± 3/year and 48 ± 55 minutes).

Table 2. Most common symptoms and provoking factors of atrioventricular nodal reentrant tachycardia (AVNRT)

No	Most common symptoms of AVNRT	Number of patients
1	Paroxysmal palpitations	79 (89%)
2	Asthenia	76 (83%)
3	Fatigability (reduced exercise tolerance)	70 (76%)
4	Dyspnoea	63 (68%)
5	Retrosternal pain and/or discomfort in the chest	61 (66%)
6	Dizziness	58 (63%)
7	Anxiety, irritability	55 (60%)
8	Urination during/after arrhythmia episode	27 (29%)
9	Blurred vision/scotomas	23 (25%)
10	Syncope	18 (20%)
11	Tingling/sensory disorders in the face	5 (5%)
12	Mumbling speech	4 (4%)
13	Considerable muscle weakness in extremities (paresis)	2 (2%)
	Most common provoking factors of AVNRT episodes	
1	Intense physical exercise	41 (45%)
2	Stress and/or emotion	37 (40%)
3	Sudden change of body position	34 (37%)
4	Sleeping (the patient woke up due to arrhythmia episode)	17 (18%)
5	Sudden change of ambient temperature (sauna, going outside in cold weather)	8 (9%)
6	Menstruation	8 (14% of women)
7	Pregnancy	1 (2% of women)

Time from first symptoms to exacerbation of arrhythmia was 14 ± 11 years. The duration of this period was significantly related to AVNRT CL — the longer arrhythmia CL, the longer time to exacerbation (Tab. 3). Symptom exacerbation was observed significantly more frequent in patients receiving oral drugs for arrhythmia prevention (84% vs 52%, p = 0.002), regardless the duration of treatment (p > 0.05). No relationship between arrhythmia exacerbation and patients' age was found (46 ± 16 vs 42 ± 12 years, p > 0.05).

Tachycardia tolerance

Good tolerance of tachycardia was found to be significantly more frequent in patients without tachycardia exacerbation (p = 0.045), dyspnoea (p = 0.004), blurred vision/scotomas (p = 0.02) or syncopes (p = 0.01) and in patients without provoked arrhythmia episodes during day- or night-time (p = 0.03). It was also more frequent during the rest (p = 0.02) and in patients who did not receive pharmacological SVT prevention (p = 0.02). There was no relationship between tachycardia tolerance and the presence of SHD, AVNRT CL and episode duration (p > 0.05). EPS did not reveal poor haemodynamic AVNRT tolerance in any patient.

Diagnosis of paroxysmal tachycardia

In 66% of cases SVT diagnosis was established at admission, in emergency room, or during hospitalization. In 20% of patients SVT was diagnosed by rescue crew, in 12% — by a GP practitioner, and in 2% — by a specialist in a cardiology clinic. The diagnosis was made based on 12-lead ECG (86%), 24-h Holter ECG (5%), transoesophageal stimulation (1%) or during EPS (8%). Eighty-five percent of patients reported that at least once the symptoms subsided before ECG recording. In 35% of patients 24-h ECG was performed and SVT was recorded in 28% of them (10% of all analysed patients). Time from first occurrence of arrhythmia symptoms to diagnosis of SVT was 11 ± 10 years. Only in 12% of patients the diagnosis was established during the first arrhythmia episode. Table 4 presents interpretation of symptoms and physician's recommendations prior to the diagnosis of paroxysmal SVT, based on medical records and patient interview. Time to diagnosis was significantly longer in women when: the arrhythmia episode was provoked by emotion (13 ± 9 vs 9 ± 11 years, p = 0.008); the symptom of an episode was the necessity of urination (13 ± 10 vs 10 ± 10 years, p = 0.03), and the patient did not experience

Table 3. Analysis of the relationships between clinical factors and atrioventricular nodal reentrant tachycardia (AVNRT) cycle length (CL)

Clinical factor	Yes	No	P value
Palpitations	345 ± 50 ms	400 ± 55 ms	0.001
Symptom onset at the age of ≥30	361 ± 50 ms	337 ± 56 ms	0.01
Significant ST depression during tachycardia	318 ± 42 ms	363 ± 52 ms	0.001
Impaired myocardial contractility	396 ± 47 ms	347 ± 52 ms	0.01
Correlation	**r**		**p**
Age at the onset of arrhythmia	0.3		0.006
Time from symptom onset to diagnosis of SVT	0.2		0.07
Time from symptom onset to arrhythmia exacerbation	0.3		0.03

SVT — supraventricular tachycardia

Table 4. Interpretation of reported symptoms and physician's recommendations

No	Potential cause of the symptoms	Number of patients
1	Coronary heart disease — cardiology consultation	29 (32%)
2	Mitral valve prolapse syndrome — follow-up	25 (27%)
4	Thyroid disease — endocrinology consultation	21 (23%)
5	Puberty — follow-up	21 (23%)
6	Perimenopausal period — gynaecology consultation	19 (32% of women)
7	Paroxysmal arrhythmia had resolved before the registration — 24-h ECG	17 (18%)
8	Smoking — smoking cessation	17 (18%)
9	Neurosis — sedatives	16 (17%)
10	Exacerbation of concomitant diseases — specialist consultation	11 (12%)
11	Electrolyte disturbances — K^+ and Mg^{2+} supplementation	2 (2%)

ECG — electrocardiogram

Table 5. Oral drugs used for SVT prevention

Medication	Number of patients
β-blocker	47 (70%)
Verapamil	19 (28%)
Propafenone	11 (16%)
Amiodarone	5 (7%)
Sotalol	5 (7%)
K^+/Mg^{2+} supplementation	6 (9%)
Mexiletine	1 (1%)
Quinidine	1 (1%)

SVT — supraventricular tachycardia

dizziness (14 ± 11 vs 9 ± 10 years, p = 0.03). Moreover it has been observed that the longer arrhythmia CL, the longer the time to diagnosis (Tab. 3).

Emergency and long-term medications

Pharmacological agents for prevention of SVT were used in 79% patients, often several drugs in monotherapy (Tab. 5). Only in 7% of these patients no SVT episodes were noted during subsequent follow-up. Mean treatment duration was 2.3 ± 4.2 years. Vagal manoeuvres, i.e. Valsalva manoeuvre and/or carotid sinus massage, were performed only by medical staff. Patients were not recommended to use them by oneself for stopping the arrhythmia. Methods of arrhythmia termination are presented in Table 6.

Analysis of the relationship between clinical factors and sex

Women were more prevalent in the study group (64%). The analysis of the relationship between clinical factors and sex is presented in Table 7.

Analysis of 12-lead ECG recordings with tachycardia

Ventricular CL during arrhythmia was 351 ± 53 ms (range 240–500 ms) and did not differ significantly from CL observed during EPS. Some factors significantly modified AVNRT CL thereby influencing the course of tachycardia (Tab. 3). In 25% of recordings, significant ST segment depression was observed during AVNRT (horizontal or downsloping > 1 mm), and in 6% of cases there was ST segment depression with T-wave inversion, absent in resting ECG. No relationship was found between the presence of ST-T abnormalities during tachycardia and clinical factors (p > 0.05), except for arrhythmia CL.

Discussion

It has been confirmed that AVNRT may occur at any age, most often before 40 [1, 4]. Previously unreported later occurrence of tachycardia in patients with SHD should be noted. It can be assumed that the presence of SHD resulted in modified electrophysiological properties of atrioventricular junction (AVJ) anisotropic area thereby temporarily

Table 6. Methods of emergency management of supraventricular tachycardia (SVT) and their effectiveness

Methods of SVT termination	Number of patients	Overall number of interventions	Effective	Ineffective
Vagal manoeuvre	**23 (25%)**	**23**	**6 (26%)**	**17 (74%)**
Electrical cardioversion	**4 (4%)**	**4**	**4 (100%)**	**0**
Intravenous drug	**85 (92%)**	**140**	**134 (96%)**	**6 (4%)**
Verapamil		50 (36%)	50 (100%)	0
Adenosine		34 (24%)	33 (97%)	1 (3%)
β-blocker		29 (21%)	26 (90%)	3 (10%)
Amiodarone		17 (12%)	15 (88%)	2 (12%)
Propafenone		10 (7%)	10 (100%)	0
Pill-in-the-pocket	**14 (15%)**	**18**	**18 (100%)**	**0**
Verapamil		7 (39%)	7 (100%)	0
β-blocker		6 (33%)	6 (100%)	0
Propafenone		3 (17%)	3 (100%)	0
Amiodarone		1 (6%)	1 (100%)	0
Sotalol		1 (6%)	1 (100%)	0

Table 7. Analysis of the relationship between clinical factors and sex

Clinical factor	W	M	P value
Structural heart diseases	12 (20%)	14 (42%)	0.02
Coronary heart disease with previous myocardial infarction	1 (2%)	5 (15%)	0.04
Concomitant diseases	43 (73%)	15 (45%)	0.009
Mitral valve prolapse (MVP)	23 (39%)	3 (9%)	0.005
Differential diagnosis: neurosis	14 (24%)	2 (6%)	0.06
Differential diagnosis: MVP	24 (41%)	4 (15%)	0.01
Differential diagnosis: coronary heart disease	8 (14%)	17 (52%)	0.0001
Symptoms of arrhythmia: anxiety, irritability	45 (76%)	10 (30%)	< 0.0001
Arrhythmia provocation by intense exercise	20 (34%)	21 (64%)	0.006
Arrhythmia provocation by emotion	29 (49%)	8 (24%)	0.002
Time from symptom onset to diagnosis of SVT (years)	13 ± 11	7 ± 8	0.006
Age at onset of symptoms (years)	32 ± 17	38 ± 19	0.09
LVEF (%)	62 ± 5	58 ± 8	< 0.0001
Frequency of episodes (n/year) before SVT exacerbation	2.6 ± 4.9	3.6 ± 3.8	0.02

SVT — supraventricular tachycardia; W — women; M — men

precluding the formation of persistent reentry circuit in 'electrophysiologically predisposed' subjects in whom arrhythmia hasn't manifested yet. On the other hand, the development of heart disease, usually at middle or older age, might have been a provoking factor for AVNRT, creating an appropriate background for it. Modification of AVJ area by SHD might have resulted from autonomic system changes in this area [4]. This may indirectly indicate the impact of impaired myocardial contractility on AVNRT CL.

In the study group, the percentage of patients with SHD was higher than that reported in the literature, where it was usually less than 15% [5, 6]; although, in older papers heart disease, mainly hypertensive-ischaemic type, was observed in 31–45% of patients [7]. The number of these patients depends of the age of the analysed population [7], although in our opinion the most important issue is unclear, and often subjective, definition of SHD.

Reported symptoms and provoking factors of tachycardia confirm that usually there are no specific clinical symptoms pathognomonic for AVNRT. Coronary and brain hypoperfusion symptoms are rarely described (< 3%), mainly in elderly patients, although less specific symptoms suggesting organ ischaemia, e.g. discomfort in the chest, are more frequent (40%) [1, 3, 5–9]. In our study, symptoms indicative of coronary or brain hypoperfusion were observed more frequently. Due to the fact that the presence of this symptoms was not associated with AVNRT CL, SHD and patients' age, the percentage of patients with tachycardia (4%) who required electric cardioversion was low, and there were no reports on poor haemodynamic tolerance during EPS, the diagnosis of actual ischaemia of central nervous system or myocardium during AVNRT seems to be unlikely. The nature of reported symptoms may be attributed to patients' individual sensitivity to tachycardia; therefore, differential diagnosis of syncope (20%) should include other potential reasons, besides AVNRT.

A new observation is that AVNRT exacerbation occurs in the majority of patients (75%) about 14 years after the symptom onset. The reason of this phenomenon remains unclear. Shorter AVNRT CL found to be a predictor for subsequent exacerbation of the arrhythmia. Marked association between arrhythmia exacerbation and pharmacological prevention of arrhythmia episodes is unequivocal. It may result from proarrhythmic effect of drugs taken. The most frequently used drugs, β-blockers and verapamil, affecting slow pathway of atrioventricular node, may prevent AVNRT recurrence, but in some patients they have opposite effect. Other researchers identified a phenomenon of spontaneous regression of arrhythmia symptoms, which was not observed in our study. In a study performed by D'Este et al. [6], 45% out of 38 untreated patients with AVNRT remained asymptomatic during recent 3 years of long-term follow-up. Prior to inclusion, these patients had significantly shorter history of arrhythmia; episodes occurred less frequently and were of shorter duration. This was not, however, a randomised trial, and the arrhythmia symptom regression had not been verified based on lack of AVNRT induction during EPS. It cannot be excluded that symptom recurrence might have been observed later, if the patients were followed-up longer. The authors speculate that the phenomenon of arrhythmia 'disappearance' may have been caused by the fact that electrophysiological properties of the element of reentry circuit change with time [6]. The frequency and duration of AVNRT episodes remained unchanged in long-

-term follow-up, until ablation. Maybe in those patients spontaneous symptom regression would also be observed, if the follow-up period was extended.

The tolerance of tachycardia is determined mostly by the presence of SHD, AVNRT CL and episode duration as well as patient's individual sensitivity to arrhythmia [1, 3, 10]. In this paper, it was found that the tolerance of AVNRT symptoms was related only to episode exacerbation and individual sensitivity to tachycardia expressed by the lack of some symptoms and/or provoking factors. One of the reasons for the discrepancy of published data may be different range of diseases categorised as SHD [5, 7, 8]. Additionally, the study population might have been too small, which translated to lower number of patients with advanced heart disease. Overrepresentation of patients with arrhythmia exacerbation is also possible.

Significant delay in establishing the diagnosis of arrhythmia has been observed, which seems to have multifactorial background. Paroxysmal, unpredictable nature of AVNRT caused considerable problems with its recording. Long-term monitoring techniques recommended by current guidelines [1] have not been used (event recorders, ECG telemetry) or were rarely used (Holter ECG). Moreover, non-specific symptoms and circumstances of AVNRT provocation were misinterpreted by a medical staff, particularly in women. For the first time, it has been observed that longer AVNRT CL (the parameter that depends on age at symptom onset and the presence of impaired myocardial contractility) is related to delayed SVT diagnosis. This relationship seems to be determined by lower frequency of palpitations (Tab. 3).

Lower than reported in the literature effectiveness of pharmacotherapy in the prevention of SVT episodes, estimated to be 30–50% [1, 11], results probably from the range of antiarrhythmic agents used and frequent use of drugs that were not recommended [1]. The methods used for AVNRT episode termination were inconsistent with current guidelines recommendations [1]. Despite relevant high-class recommendations, vagal nerve manoeuvres and drugs characterized with the highest effectiveness (adenosine, diltiazem, verapamil) [5] were rarely performed or advised and, even more, drugs that are not recommended were used (e.g. amiodarone) [1]. Ranges of used antiarrhythmic drags may have been determined by their availability. It is not without significance that basic ambulance crew composed of medical rescuers is allowed to administer only 2 antiarrhythmic drugs, i.e. amiodarone and lignocaine [12]. Also the drugs used as pill-in-the-pocket strategy were inconsistent with the guidelines [1], although all of the drugs used proved to be effective. It should be noted that, although current guidelines recommend the use of single oral dose or diltiazem or propranolol, such treatment has not been used in the study patients.

It has been confirmed that AVNRT is more prevalent in women (60–78%), in whom arrhythmia develops at 10 years younger age, and has different clinical history than in men [1, 3–5, 8, 9, 13]. In women, SHD (related to later manifestation of tachycardia), including coronary heart disease and reduced LVEF, occurs more rarely, which might be attributed to patient age distribution. Coronary heart disease becomes more prevalent in women than in men only after the age of 70 [14]. A new finding is more frequent occurrence of MVP in patients with AVNRT compared with general population (4–17%). MVP is more common in women between 20 and 40 years of age; it is often associated by impaired autonomic and neuroendocrine control leading to, among others, increased catecholamine levels. This mechanism may potentially affect electrophysiological properties of AVJ, enabling the formation of a critical reentry circuit for AVNRT, and numerous premature atrial complexes may lead to arrhythmic episode [15]. This may be the cause of more common occurrence of AVNRT in women. It has been confirmed that time to establishing diagnosis of paroxysmal SVT is considerably longer in female patients [16]. Main reason for the delay in diagnosis is misinterpretation of arrhythmia symptoms, particularly as related to neurosis, MVP and perimenopausal period or exacerbation of more common concomitant diseases. Furthermore, significant impact on increased time to diagnosis had also the presence of women-specific provoking factors (emotion), symptoms of AVNRT (anxiety, irritability) and lower frequency of episodes before arrhythmia exacerbation. Moreover, symptoms concomitant with MPV and provoking circumstances are very similar to those of paroxysmal SVT [15]. It seems that more frequent provocation of AVNRT by physical exercise and suspected coronary heart disease in men sped up establishing of proper diagnosis.

More frequent ST-T abnormalities during arrhythmia were observed, that were dependent on AVNRT CL and unrelated to coronary heart disease and symptoms suggesting myocardial ischaemia. It seems that these abnormalities are functional in nature and are not associated with myocardial ischaemia [17].

Limitations of the study

Patients included in the study were not consecutive patients with arrhythmia selected from general population, but subjects with AVNRT, who decided to underwent ablation. Those who refused to undergo invasive diagnostics for SVT and paediatric patients were excluded from the study. Probably, patients with more advanced disease, who are more prone to underwent invasive procedures, were overrepresented in the study group. Additionally, history data collected based on patient interview are subjective and usually cannot be verified. This may partly

explain the differences observed when compared with literature data.

Conclusions

In this study, it has been found that exacerbation of episodes of typical AVNRT in a group of patients who underwent ablation occurred in the majority of patients, which influence the tolerance of arrhythmia symptoms, and that frequently reported by patients symptoms of coronary and brain hypoperfusion were unspecific. Furthermore, considerable delay in diagnosis of arrhythmia was observed, particularly in women, which was at least in part dependent on medical staff (misinterpretation of symptoms, ECG recording methods used). Moreover, emergency and long-term pharmacotherapy of AVNRT was inconsistent with therapeutic guideline recommendations. Also, potential factors significantly influencing natural history of AVNRT has been identified. It has been found that SHD is related to later development of tachycardia and AVNRT occurrence at younger age in women. AVNRT CL (a parameter that is dependent on the age at arrhythmia occurrence and the presence of impaired myocardial contractility) influences many factors such as time to establishing the diagnosis of SVT and to exacerbation of arrhythmia, the presence of palpitations or ST-T abnormalities during tachycardia. More frequent presence of MVP in women with AVNRT compared with general population is one of factors causing delay in diagnosis SVT due to misinterpretation of the symptoms of arrhythmia that are attributed to MVP. This may also be the reason why AVNRT is more common in women.

Conflict of interest

None declared.

Streszczenie

Wstęp. Nawrotny częstoskurcz węzłowy (AVNRT) jest najczęstszą regularną tachyarytmią, ale obraz kliniczny częstoskurczu nie został dotychczas szczegółowo zbadany. Celem pracy była ocena przebiegu klinicznego i identyfikacja potencjalnych czynników mających istotny wpływ na historię naturalną typowej postaci AVNRT u chorych poddawanych ablacji.

Materiał i metody. Do badania włączono 92 kolejne osoby z rozpoznanym podczas badania elektrofizjologicznego typowym AVNRT, poddane ablacji podłoża arytmii. U wszystkich pacjentów aktywnie poszukiwano obecności choroby organicznej serca (SHD). Zebrano celowany, retrospektywny wywiad dotyczący przebiegu arytmii, wpływających na niego czynników oraz stosowanego leczenia, z uwzględnieniem dostępnej dokumentacji medycznej i zapisu elektrokardiograficznego z obecnością AVNRT.

Wyniki. U 75% pacjentów wystąpiło nasilenie arytmii. Nie wykazano zależności zgłaszanych objawów hipoperfuzji wieńcowej (66%) i mózgowej (33%) od czynników klinicznych. Czas do rozpoznania częstoskurczu był dłuższy u kobiet (13 ± 11 *v.* 7 ± 8 lat; p = 0,006). Leczenie farmakologiczne AVNRT odbiegało od zalecanych wytycznych postępowania. Objawy arytmii pojawiały się później w obecności SHD (44 ± 13 *v.* 30 ± 5 lat; p = 0,001). Dłuższa długość cyklu (CL) AVNRT, na którą wpływał wiek pojawienia się objawów i obecność zaburzeń kurczliwości mięśnia sercowego, wydłużała czas do rozpoznania i nasilenia arytmii. Wypadanie płatka zastawki mitralnej (MVP) występowało u pacjentek częściej w porównaniu do populacji ogólnej i było czynnikiem opóźniającym rozpoznanie AVNRT.

Wnioski. Nasilenie napadów AVNRT następuje u większości pacjentów. Często zgłaszane objawy hipoperfuzji wieńcowej i mózgowej mają niespecyficzny charakter. Ma miejsce znaczne opóźnienie rozpoznania arytmii, zwłaszcza u kobiet. Strukturalna choroba serca, AVNRT CL i MVP stanowią czynniki istotnie modyfikujące przebieg częstoskurczu.

Słowa kluczowe: częstoskurcz węzłowy nawrotny, choroba organiczna serca, nasilenie arytmii

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