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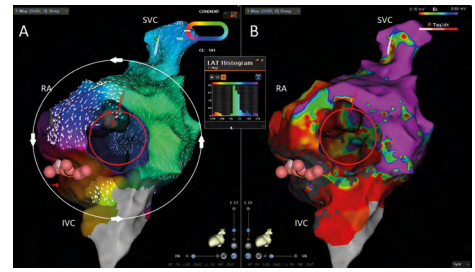

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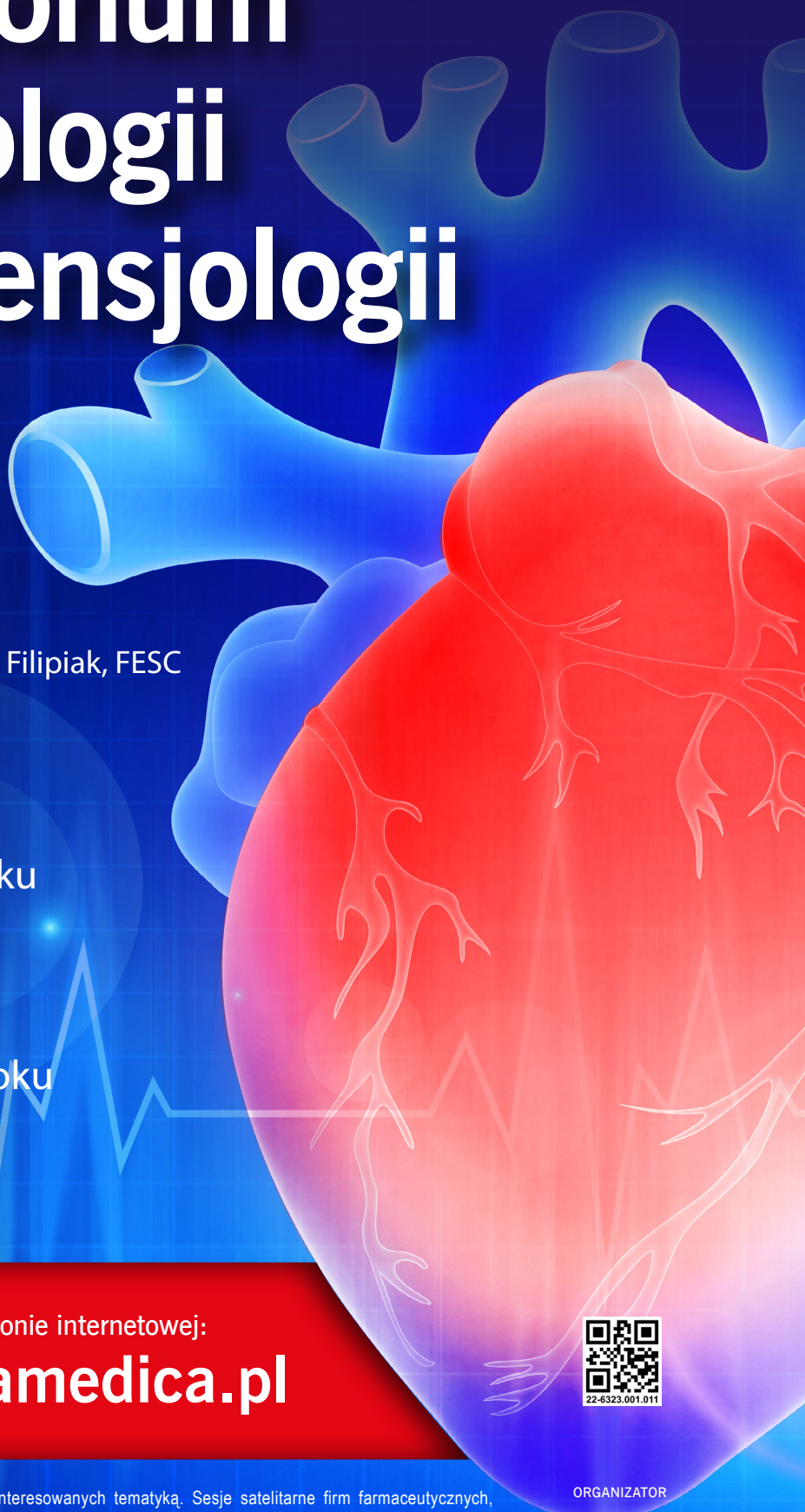

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2023, vol. 18, no. 2

Table of Contents/Spis treści

ORIGINAL ARTICLE/PRACA ORYGINALNA

REVIEW ARTICLES/PRACE POGLĄDOWE

CASE REPORTS/PRACE KAZUISTYCZNE

SPRAWOZDANIE Z KONFERENCJI

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Od Redaktora

Drodzy Czytelnicy,

zbliża się długi weekend majowy, a my na deszczowe (ale i słoneczne) dni przygotowaliśmy dla Państwa drugi w tym roku numer naszego czasopisma, a w nim stałe działy i relacja z corocznej konferencji pisma, która odbyła się w Warszawie 21–22 kwietnia 2023 roku.

Zdajemy sobie sprawę, jak ważne jest stosowanie się do zaleceń lekarskich, jak trudno wprowadzić w życie i kontrolować *compliance* i adherencję do zaleconej farmakoterapii, też dobrze wiemy. Praca „Electronic measurement of medication adherence in patients with heart failure" Macieja Nadela i wsp. z II Kliniki Kardiologii Uniwersytetu Medycznego w Łodzi poświęcona jest ocenie regularności przyjmowania leków i jej związku ze zmiennymi demograficznymi i klinicznymi u pacjentów z niewydolnością serca. Przyjmowanie leków oceniano za pomocą elektronicznego systemu monitorowania przyjmowania leków (MEMS) — elektronicznej nakrętki zakładanej na pojemnik z wybranym lekiem. W 30-dniowej obserwacji około 27% pacjentów przyjmowało leki nieregularnie, byli to chorzy młodsi, o dziwo — z niższą frakcją wyrzutową, częściej hospitalizowane z powodu ostrej niewydolności serca.

W numerze znajdziecie Państwo dwie ciekawe prace poglądowe z dziedziny diagnostyki i elektroterapii. W pracy „CA-125: a promising biomarker in heart failure. Summary of the current knowledge" Marii Sawościan i Małgorzaty Lelonek z Zakładu Kardiologii Nieinwazyjnej Katedry Chorób Wewnętrznych i Kardiologii Uniwersytetu Medycznego w Łodzi omówiono znaczenie nowego biomarkera, antygenu węglowodanowego 125 (CA-125), powszechnie używanego do tej pory w onkologii, ale coraz bardziej obiecującego jako marker przydatny w ocenie zastoju oraz procesów zapalnych w niewydolności serca. Artykuł „Catheter ablation of the cavo-tricuspid isthmus as a bridging therapy for symptomatic atrial fibrillation?" Tomasza Wcisły i Michała Plewki z Kliniki Kardiologii Interwencyjnej i Zaburzeń Rytmu Serca Uniwersyteckiego Szpitala Klinicznego im. WAM z Łodzi przedstawia w sposób skondensowany strategie postępowania u chorych z migotaniem i trzepotaniem przedsionków. Obie arytmie mają inne podłoże, inną podatność na stosowane leczenie farmakologiczne oraz wymagają odmiennego podejścia do leczenia niefarmakologicznego.

Dział przypadków klinicznych obfituje w opisy czterech niezmiernie ciekawych i ważnych problemów klinicznych z dziedziny elektrofizjologii (z ośrodków kieleckiego i warszawskiego), hemodynamiki (z ośrodka białostockiego) i pediatrii (z ośrodków katowickich).

Zapraszam wobec tego do lektury.

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Electronic measurement of medication adherence in patients with heart failure

Elektroniczny pomiar regularności przyjmowania leków u pacjentów z niewydolnością serca

Maciej Nadel, Robert Morawiec, Oliwia Matuszewska-Brycht, Jarosław Drożdż

2nd Department of Cardiology, Central University Hospital, Medical University of Lodz, Łódź, Poland

Abstract

Introduction. Heart failure (HF) is a significant clinical and socioeconomic problem both in Poland and around the world. However, objective data on the level of adherence in the era of improved medical therapy is lacking. Therefore, the aim of the study was to investigate the level of medication adherence and its association with demographical and clinical variables in patients with HF.

Material and methods. We have conducted a prospective cohort study of 25 patients with diagnosed HF. Medication adherence was measured for 30 consecutive days using the Medication Event Monitoring System (MEMS) — an electronic cap attached to the medication container, allowing to record the exact moment of taking the measured medicine. Based on the acquired data, patients were classified as adherent or non-adherent using an evidence-based cut-point. In addition to adherence measurement, patients' demographic and clinical information was collected.

Results. Twenty-two patients provided full results from the MEMS devices. The median age of the patients was 70 years (interquartile range =14), and the mean left ventricular ejection fraction was 33% ± 12. The mean percentage of correct doses was 89% ± 17. Twenty-seven percent of patients (n = 6) were classified as non-adherent. Patients classified as non-adherent were significantly younger (54 vs. 71 years; p = 0.015), had a lower left ventricular ejection fraction (24 vs. 36%; p = 0.04), and were more frequently enrolled after HF hospitalization (83 vs. 19%; p = 0.011).

Conclusions. In the short-term observation, a significant proportion of patients with HF were found to be non-adherent. In our study, we identified a population with an increased risk of non-adherence. Those patients require the implementation of more intensive and targeted healthcare system-based interventions in order to improve their prognosis.

Key words: heart failure, medication adherence, telemedicine

Folia Cardiologica 2023; 18, 2: 59–64

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Introduction

Heart failure (HF) is a significant clinical and socio-economic problem both in Poland and around the world. It is estimated that the prevalence of HF in the general population of adult patients is about 1–2% [1]. In Poland, there are around 1,240,000 patients with heart failure, and according to the statistics of the National Health Fund and the Central Statistical Office from 2021, it was the primary cause of over 120,000 deaths per year [2, 3]. Patients diagnosed with HF are hospitalized on average once a year, and the financial burden associated with this diagnosis is very high — it is estimated that expenditure related to HF accounts for approximately 0.6% of Poland's gross domestic product [1, 2]. Recently, new drugs have been registered in the treatment of HF, like angiotensin receptor-neprilysin inhibitor (ARNI) and sodium-glucose cotransporter-2 inhibitors (SGLT-2i), which were proven to reduce mortality and the number of hospitalizations for cardiovascular reasons [4, 5]. However, as the patient's pill burden is gradually increasing, so are concerns about non-adherence to guideline-directed medical therapy, which may lead to deterioration of treatment results [6, 7]. It has been previously shown that non-adherence can be the cause of 20–64% of HF readmissions [8]. However, objective data on the level of adherence in the era of improved medical therapy is lacking. Therefore,the aim of the study was to investigate the level of medication adherence and its association with demographical and clinical variables in patients with HF.

Materials and methods

Study design and population

We have conducted a prospective cohort study of patients with diagnosed HF. Twenty-five participants were enrolled in the period from April 2021 to March 2022, both directly after HF-related hospitalization in the Cardiology Department of the Central Clinical Hospital in Lodz, as well as in the cardiology outpatient clinic. Eligible patients were aged 18 years or older, with the diagnosis of HF according to the European Society of Cardiology Guidelines [1], with symptoms in I–III New York Heart Association (NYHA) class and N-terminal pro-B-type natriuretic peptide concentration greater than or equal to 125 pg/mL in patients with sinus rhythm or 365 pg/mL in patients with atrial fibrillation. Exclusion criteria were defined as life expectancy < 1 year, acute coronary syndrome or stroke within the last 3 months, and presence of cognitive impairment that, in the investigator's opinion, prevents the proper use of the monitoring device in accordance with the instructions. The study complied with the Declaration of Helsinki and was approved by the local medical ethics committee. Written informed consent was provided by all patients prior to their participation in the study.

Figure 1. The medication Adherence Monitoring System (MEMS), consisted of an electronic cap, medication container, and a reader, enabling the programing of the device as well as transferring the adherence data

Measurements of adherence

Medication adherence was continuously measured using the Medication Event Monitoring System (MEMS, Aardex Group, Belgium). MEMS is an electronic cap attached to the medication container, allowing one to record the exact moment of taking the measured medicine. The recorded data can then be transferred with a dedicated electronic reader, as shown in Figure 1. Devices of this type have been vastly used in studies evaluating adherence of patients treated for chronic conditions like ischemic heart disease, hypertension, and HF [9–11]. The information stored on the device was transferred to the Study Site during the follow-up visit, which took place at least 30 days after the enrollment. If such a visit was delayed, only the data from 30 days after the beginning of monitoring was analyzed. The choice of the monitored drug was made at the beginning of the study after consultation with the patient. Preferentially it was a HF drug (for example, angiotensin-converting-enzyme inhibitor, angiotensin II receptor blocker, ARNI, beta-blocker, mineralocorticoid receptor antagonist or SGLT-2i) with priority for the substance with twice-daily dosing. Using diuretics as a monitored drug was avoided. It was possible to change the monitored drug under special circumstances after consultation with the Site, provided that the new drug was dosed in the same way.

A missed dose was defined as the device not being opened for 24 hours for once-daily drugs and 12 hours from midnight to noon and noon to midnight for twice-daily drugs. Satisfactory dose intake (dosing adherence) was defined as at least 88% of doses taken out of all planned

doses, and patients with this percentage were classified as adherent. Conversely, patients with lower dosing adherence were classified as non-adherent. The above cut-off point was selected based on an earlier study in patients with HF, where it was shown that the percentage of doses taken equal to or greater than 88% is associated with a significant reduction in cardiovascular events [12]. This cut-off point was also subsequently used in other studies of patients with HF [11, 13].

Subjects' demographic and clinical data

Before handing over the measuring device, patient characteristics were collected, such as age, gender, years of education, marital status, presence of cohabitants, place of residence (village, city), number of years since the diagnosis of HF, the overall number of drugs taken, number of hospitalizations due to HF in the previous 12 months. Clinical information such as left ventricular ejection fraction (LVEF, %), NYHA functional class, and level of N-terminal pro-B-type natriuretic peptide was also acquired from the medical record.

Data analysis

All the data from the study were analyzed using STATISTICA 13.3 software (TIBCO, Palo Alto, CA, USA). Descriptive statistics were used to characterize the study population and assess medication adherence. The normality of data was assessed with the Shapiro–Wilk test. Categorical variables are presented as percentages, while continuous variables are presented using means with standard deviation or median with interquartile range. Differences between adherent and non-adherent patients were tested using Fisher's exact test for dichotomous variables and Student's t-test or Mann–Whitney test for normally and non-normally distributed continuous variables respectively. The p-value < 0.05 was considered statistically significant.

Results

Twenty-five patients were enrolled in the study, of whom 22 provided full results from the MEMS devices. One patient lost the device during another HF hospitalization and 2 patients refused to use the device during the follow-up. The characteristics of the patients with full data acquired are presented in Table 1.

The mean percentage of correctly taken doses was 89% and 73% of patients (n = 16) were classified as adherent. Examples of readings from devices used by adherent and non-adherent patients are presented in Figure 2. Patients classified as non-adherent (27%; n = 6) were significantly younger (54 vs. 71; p = 0.015), had a lower LVEF (24 vs. 36%; p = 0.04), and were more frequently enrolled

Table 1. Characteristics of the study population with differences between the adherent and non-adherent patients

	All patients (n = 22)	Adherent patients (n = 16)	Non-adherent patients (n = 6)	p-value
Age (years) median, (IQR)	70 (14)	71 (5)	54 (11)	**0.015**
Female % (n)	27% (6)	31% (5)	17% (1)	0.634
Not living alone % (n)	86% (19)	81% (13)	100% (6)	0.532
Married % (n)	59% (13)	50% (8)	83% (5)	0.333
Living in a city with population > 20,000	86% (19)	88% (14)	83% (5)	> 0.999
Higher education % (n)	23% (5)	19% (3)	33% (2)	0.585
> 1 year since diagnosis of HF % (n)	50% (11)	50% (8)	50% (3)	> 0.999
Inclusion in the study directly after HF hospitalization % (n)	36% (8)	19% (3)	83% (5)	**0.011**
NYHA class % (n)				> 0.999
• I	5% (1)	6% (1)	0% (0)	
• II	82% (18)	81% (13)	83% (5)	
• III	14% (3)	13% (2)	17% (1)	
NT-proBNP pg/mL, median (IQR)	1571 (1856)	1519 (1682)	1711 (4492)	0.971
LVEF (%), mean ± SD	33 (12)	36 (13)	24 (7)	**0.04**
Total number of medications, mean ± SD	10 (3)	10 (3)	10 (4)	0.884
Dosage twice a day % (n)	23% (5)	25% (4)	17% (1)	> 0.999
Dosing adherence %, mean ± SD	89 (17)	98 (3)	67 (20)	

HF — heart failure; NT-proBNP — N-terminal pro-B-type natriuretic peptide; NYHA — New York Heart Association

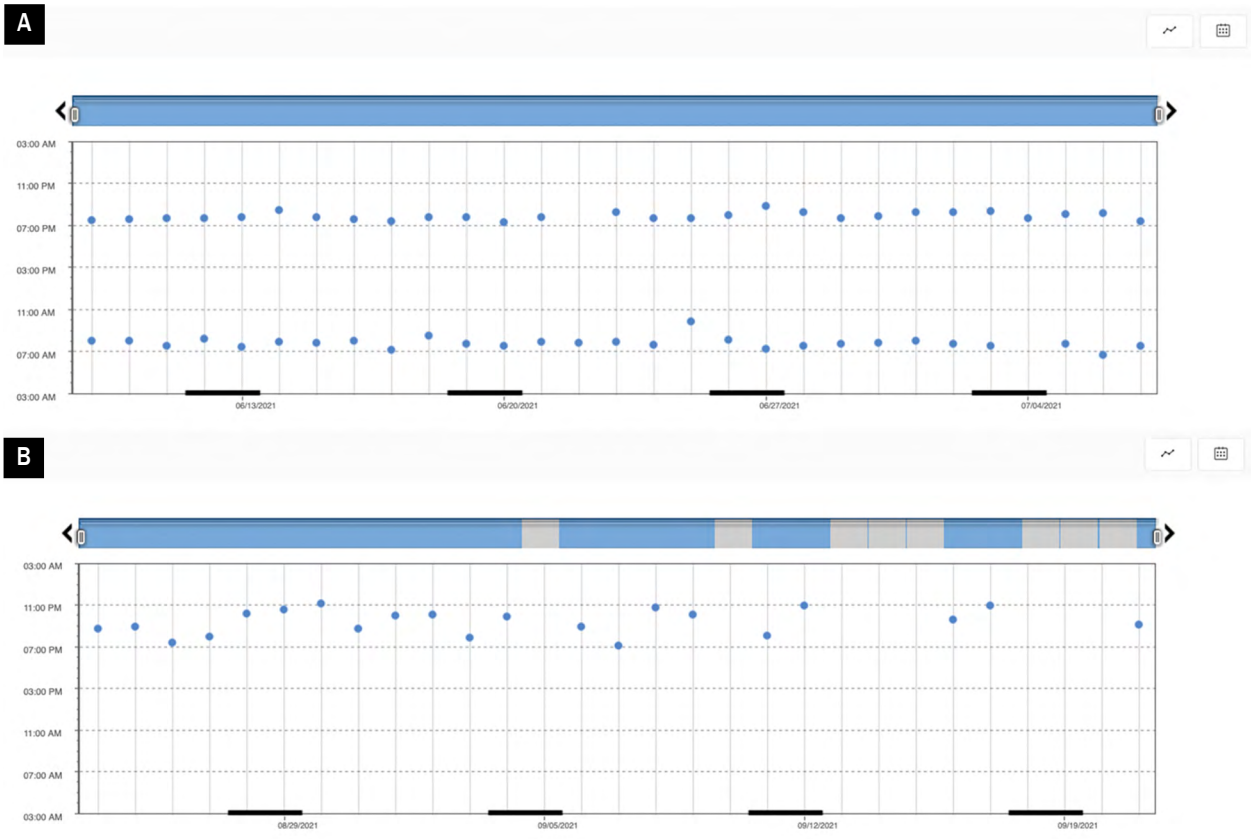

Figure 2. Data from a MEMS device was acquired from two patients. Blue dots indicate the precise moment of opening the device: **A.** Patient with twice-daily dosing of a monitored drug. The patient omitted 2 of 60 doses of the drug, hence his dosing adherence is 97%. This patient is considered adherent (dosing adherence ≥ 88%); **B.** Patient with once-daily dosing of a monitored drug. The patient omitted 8 of 30 doses of the drug (dosing adherence 73%). This patient is considered non-adherent

after HF hospitalization (83 vs. 19%; p = 0.011). There were no statistically significant differences in other parameters.

Discussion

The presented study provides insight into the objectively measured level of adherence in a prospective cohort of contemporary patients with HF. Although the mean dosing adherence of the studied population is 89%, the percentage of patients meeting the evidence-based criteria for satisfactory adherence is only 73%. Previous research conducted on patients with HF showed different levels of medication non-adherence ranging from 11% to 61% [14–16]. Those significant discrepancies may be caused by varying adherence assessment methods (questionnaires, electronic monitoring devices, prescription databases), as well as diverse populations and different healthcare systems in which participants were recruited. However, one study based on a similar methodology estimated the percentage of adherent ambulatory HF patients to be 76% [13].

In our study, the non-adherent patients were significantly younger. The results of previous studies evaluating the effect of age on adherence in patients with cardiovascular disease are ambiguous [17–20]. One of the studies on hypertensive patients showed that there is a U-shaped relationship — both older and younger patients are less adherent, with most adherent patients at the age of 60 to 69 years [21]. However, a systematic literature review of several studies measuring adherence specifically in patients with HF showed that older age alone is not related to poorer medication adherence and in fact, there might be a positive correlation of age and adherence, which is consistent with the results of our study [22].

Furthermore, the non-adherent patients had significantly lower ejection fraction. Similar results have been reported previously in a study using a questionnaire method of assessing medication adherence [23]. Moreover, this relationship may be also related to the fact that the non-adherent patients were more frequently enrolled after HF hospitalization as opposed to the adherent patients. We did not find any previous research on such a relationship in the literature, however, it may reflect the established fact that non-adherence is a major cause of failure exacerbation, resulting in a greater number of non-adherent patients

in the hospital population [8]. For that reason, future interventions aimed at increasing adherence should consider focusing on patients after recent HF hospitalization.

Interestingly, we have not detected any association between adherence and the complexity of the monitored drug regimen (once daily vs. twice daily). Such association has been previously reported in several studies using MEMS [9, 13]. This difference may stem from the fact that the overall number of patients with twice-daily medication regimens was relatively low. Also, the mean total number of medications did not differ between adherent and non-adherent patients, supporting the results of an earlier study in hypertensive patients in which the complexity of the medication regimen, not the number of medications alone, was a predictor of non-adherence [24].

Limitations

There are several limitations to our study. First, the study sample was relatively small, hence we have not performed multivariate analysis. In addition, adherence for only one medication was measured and only the act of opening the device was assessed, therefore, there remains a level of uncertainty about whether patients actually ingested the medicine. Additionally, we can assume that patients who agreed to participate in the study and received the monitoring device are more motivated and may have higher adherence. Nonetheless, apart from repeated measurements of serum drug concentration, electronic measurement of medication adherence is considered to be a gold standard in studies assessing adherence [25].

Conclusions

In the short-term observation, a significant proportion of patients with HF were found to be non-adherent. In our study, we identified a population with an increased risk of non-adherence. Those patients require the implementation of more intensive and targeted healthcare system-based interventions in order to improve their prognosis. Further research conducted on a larger population is needed to confirm the aforementioned observations and to develop effective interventions to increase the level of medication adherence.

Conflict of interest

None declared

Funding

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Streszczenie

Wstęp. Niewydolność serca (HF) jest istotnym problemem klinicznym i socjoekonomicznym zarówno w Polsce, jak i na świecie. Pomimo rozwoju nowoczesnej i skutecznej farmakoterapii, brakuje obiektywnych danych na temat stosowania się do zaleceń lekarskich dotyczących przyjmowania leków przez pacjentów. Celem poniższej pracy była ocena regularności przyjmowania leków i jej związku ze zmiennymi demograficznymi i klinicznymi u pacjentów z HF.

Materiał i metody. Przeprowadzono prospektywne badanie kohortowe na populacji 25 pacjentów z rozpoznaną HF. Przyjmowanie leków oceniano przez 30 kolejnych dni za pomocą elektronicznego systemu monitorowania przyjmowania leków (MEMS) — elektronicznej nakrętki zakładanej na pojemnik z wybranym lekiem, pozwalającej na cyfrowy pomiar dokładnego momentu przyjęcia leku stosowanego w HF. Na podstawie uzyskanych danych pacjentów sklasyfikowano jako przestrzegających lub nieprzestrzegających zaleceń oraz oceniono wpływ zmiennych klinicznych i demograficznych na regularność przyjmowania leków.

Wyniki. Uzyskano pełne wyniki z urządzeń MEMS od 22 pacjentów. Mediana wieku pacjentów wynosiła 70 lat (IQR = 14), średnia frakcja wyrzutowa lewej komory wynosiła 33% ± 12. Średni odsetek prawidłowo przyjętych dawek wynosił 89% ± 17. Jako nieregularnie przyjmujących leki zostało sklasyfikowanych 27% pacjentów (n = 6). Byli oni istotnie młodsi (54 *vs.* 71 lat; p = 0,015), mieli niższą frakcję wyrzutową lewej komory (24 *vs.* 36%; p = 0,04) i byli częściej hospitalizowani z powodu ostrej HF (83 *vs.* 19%; p = 0,011).

Wnioski. W krótkoterminowej obserwacji znaczący odsetek pacjentów z HF nie przyjmował leków zgodnie z zaleceniami. Zidentyfikowana w niniejszym badaniu populacja pacjentów częściej nieprzestrzegających zaleceń lekarskich wymaga intensywniejszej i ukierunkowanej interwencji ze strony systemu opieki zdrowotnej. Opracowanie skutecznej interwencji i zogniskowanie jej na powyższej podgrupie ma szansę poprawić rokowanie chorych.

Słowa kluczowe: niewydolność serca, przestrzeganie zaleceń terapeutycznych, telemedycyna

Folia Cardiologica 2023; 18, 2: 59–64

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CA-125: a promising biomarker in heart failure. Summary of the current knowledge

CA-125 — obiecujący biomarker w niewydolności serca. Podsumowanie bieżącego stanu wiedzy

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Abstract

The search for suitable biomarkers for monitoring and treatment of patients with heart failure (HF) is still ongoing. The novel biomarker — carbohydrate antigen 125 (CA-125), used commonly so far in oncology, was proven to be useful in assessing congestion and inflammation in heart failure. Its elevated values are also strongly correlated with impaired right ventricle function. What is more, the increased level of CA-125 correlates positively with HF readmissions and a higher risk of death. Hence, that makes a need to tailor a potential treatment strategy, which was performed in the CHANCE-HF study. Despite the advantages of this biomarker, such as convenient access or significantly low cost of assessments, conducting multicentre and international studies is necessary to implement suitable guidelines.

Key words: heart failure, acute heart failure, echocardiography, biomarkers, clinical practice

Folia Cardiologica 2023; 18, 2: 65–70

Introduction

The search for new biomarkers in heart failure (HF) that would be useful in patients' monitoring and treatment is still ongoing. Given the fact that HF should be defined as a clinical syndrome, not a single diagnosis [1] and considering its diverse pathophysiology, it is essential to select an appropriate set of biomarkers that can be helpful in the differential diagnosis of HF, risk stratification and modification of treatment [2].

Whereas congestion is one of the biggest challenges in the management of patients with acute heart failure (AHF) [3], the novel biomarker — carbohydrate antigen 125 (CA-125) appears to have great clinical potential that would provide accurate identification of HF patients and monitor their treatment [4]. CA-125 is a commonly used biomarker for monitoring ovarian cancer [5] which is also normally present on the cell surface in different tissues, such as pericardium, endometrium, endocervix, peritoneum, salpinges, lung, pleura or prostate [6]. Since the elevation of CA-125 in non-malignant conditions such as liver cirrhosis or ascites was confirmed [6], the findings which indicate its role in pathophysiological processes in HF appeared as well [7, 8].

This review aims to condense the current state of knowledge about this promising biomarker in prognosis and risk stratification of HF.

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The role of CA-125 in congestion and inflammation

Congestion is often a reason for a decompensated HF that may require an urgent need for hospitalization [9]. Núñez et al. [10] emphasized the relationship between peripheral oedema, serosal effusion and elevated level of CA-125. The mechanical stress caused by volume overload was found as an association between fluid congestion and increased secretion of CA-125 [6]. Both, mechanical stress and inflammatory stimuli may initiate c-Jun N-terminal kinase pathways which leads to stimulation of CA-125 [11]. On the other hand, Zeillemaker et al. [12] found an association between the secretion of CA-125 and inflammatory cytokines such as interleukin-1 (IL-1), tumour necrosis factor-α (TNF-α) and lipopolysaccharide. Furthermore, Bulska-Będkowska et al. [13] reported additionally that the level of CA-125 correlates positively with the high-sensitivity C-reactive protein and the IL-6. It is also worth noting that congestion and inflammation are interrelated [14] and it also seems that peripheral venous fluid overload leads to increased secretion of inflammatory markers — IL-6 [14]. Although the unknown biological role of CA-125, it was proved that mechanical stress, inflammation or fluid congestion stimulates mesothelial cells to increase the secretion of CA-125 [15]. The mutual interaction between the two components of HF pathophysiology is shown in Figure 1.

CA-125 and echocardiographic parameters

The right ventricle and its function play a significant role in a patient population with left-sided HF, regardless of the ejection fraction of the left ventricle [16]. Hence, CA-125 is firmly correlated with right-sided HF parameters such as tricuspid regurgitation (TR) [16] which is linked with the worse prognosis [17]. Consistently, Kouris et al. [18] reported the correlation of high CA-125 levels with right ventricular systolic pressure as well as a link between CA-125 concentration and severity of AHF and fluid overload. On the contrary, D'Aloia et al. [19] reported the correlation between the CA-125 levels and the echocardiographic parameters regarding right and left heart filling pressures and diastolic anomalies. What is more, significantly higher CA-125 values were found in patients with New York Heart Association class III/IV than in I/II [19]. Subsequently, some findings proved the association between CA-125 serum concentrations and worse prognosis in pulmonary hypertension [20].

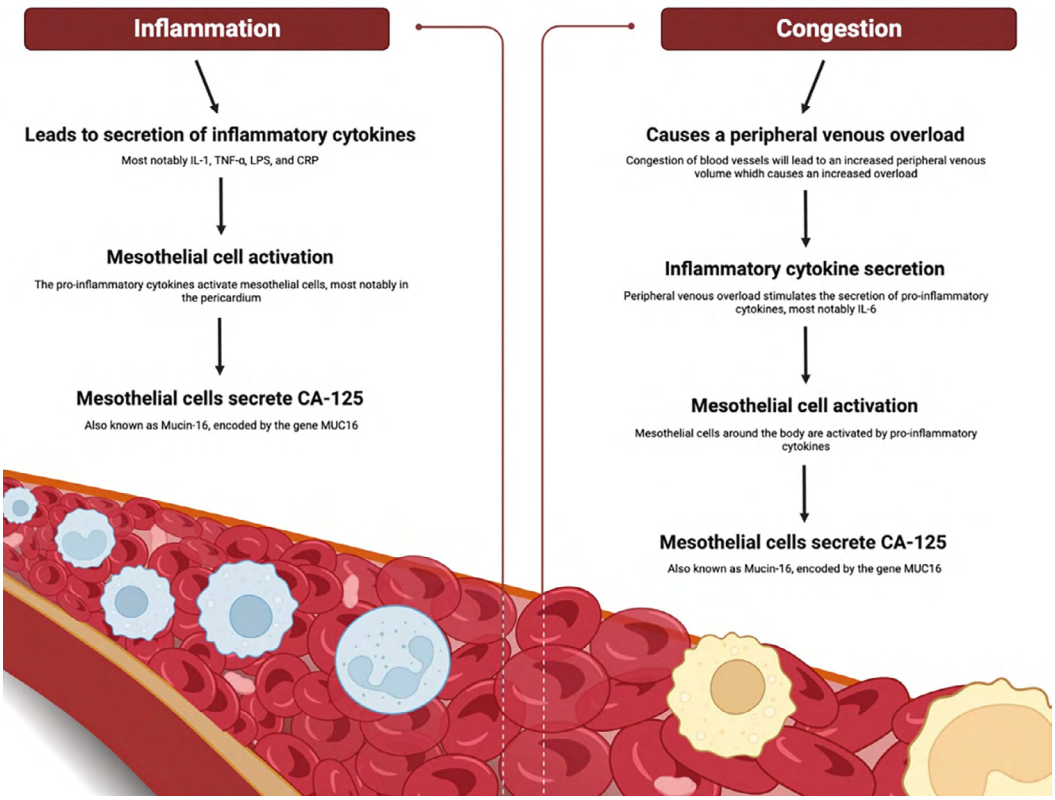

Figure 1. The mutual interaction between inflammation and congestion in heart failure

Table 1. Comparison of selected heart failure biomarkers

Biomarker	Clinical significance	References
CA-125	• congestion assessment • risk stratification	[21], [16], [29]
NT-proBNP	• myocardial injury and remodelling • diagnostic, risk stratification	[22], [23], [30]
hs-CRP	• inflammation • risk stratification, prognosis	[24], [31]
hs-TnT	• myocardial injury • risk stratification	[25], [22], [30]
RDW	• marker of hypoxia • prediction of long-term prognosis	[26]
GDF-15	• inflammation • correlation with CVD events	[27], [22], [30]
Gal-3	• myocardial remodelling • correlation with CVD and all-cause mortality	[28], [22], [30]
IGFBP-7	• metabolic dysfunction • biomarker of prognosis, especially for HFpEF	[27]

CA-125 — carbohydrate antigen 125; Gal-3 — galectin 3; GDF-15 — growth differentiation factor 15; hs-CRP — high sensitivity C-reactive protein; hs-TnT — high sensitivity cardiac troponin; IGFBP-7 — insulin-like growth factor-binding protein; NT-proBNP — N-terminal pro-B-type natriuretic peptide; RDW — red cell distribution width

CA-125 and other heart failure biomarkers

A well-understood role and clinical utility of a particular biomarker allow the selection of the appropriate set of assessments that are necessary for the management of HF patients. Therefore, a comparison of selected biomarkers is presented in Table 1. The purpose of the comparison of those molecules is to indicate their application to clinical practice as well as show their biological role.

CA-125 as a prognostic factor in risk stratification

In the last twenty years, plenty of studies indicate the prognostic value of an elevated CA-125 level in HF. Apart from CHANCE-HF, a randomized clinical trial by Núñez et al. [32], most of the research was an observational study. However, the BIOSTAT-CHF, a multicentre, international, prospective and observational subanalysis regarding worsening HF (n = 2516) is worth mentioning [33]. In multivariable survival analysis, increased CA-125 level was related to a higher risk of mortality and the composite of death/HF rehospitalization ($p < 0.001$ for both results) [33]. What is more, the prognostic role was proved independently of the severity of systemic congestion. The strategy involved managing diuretic treatment based on CA-125 levels (n = 187) vs. standard of care (n = 193) was suggested by the researchers in CHANCE-HF [32]. The CA-125 strategy showed a significant decrease of the primary endpoint, which was assessed as time to first event (66 vs. 84 events; $p = 0.017$) as well as the lessening in the rate of recurrent events in 1-year-follow-up (85 vs. 165 events, IRR: 0.49; 95% CI: 0.28–0.82; $p = 0.008$) [32]. The presentation of selected studies is shown in Table 2. Due to the novel HF treatment more and more patients are treated with sodium-glucose co-transporter 2 inhibitors, which influences the cardiovascular and renal effects [34]. In the retrospective cohort [35] of patients with the diagnosis of HF and diabetes mellitus type 2 that were receiving empagliflozin the decrease of CA-125 level without modifying the N-terminal pro-B-type natriuretic peptide (NT-proBNP) trajectory was observed. Therefore, it confirms a hypothesis that empagliflozin promotes extra-vascular decongestion [35].

CA-125 and its utility in clinical practice

Since CA-125 is not a cardiac-specific biomarker and its elevated values may also appear in other benign and malignant clinical conditions, the results should be carefully interpreted without the HF diagnosis [21]. In order to monitor the patients properly and tailor therapy for them, it is necessary to take into account the long half-life of CA-125 [21]. Since CA-125 provides information about the fluid congestion within the last week, in patients with HF exacerbation it may maintain a stable or even increased level, during the following days after a decongestion [21]. Figure 2 shows different cut-off groups designed by the few last studies that may help define the HF phenotypes according to CA-125 serum level.

Moreover, CA-125 has many advantages that promote it to be commonly performed in clinical practice. First, CA-125 is a well-known biomarker, with years of diagnostic and prognostic use in ovarian cancer. Secondly, the results of CA-125, unlike NT-proBNP are not influenced by age, weight or renal parameters [6]. Finally, the cost of its assessment is significantly lower than the assessment of NT-proBNP [21].

Conclusions

Certainly, CA-125 has proved to be a promising biomarker for the assessment of congestion and inflammation in both acute HF and chronic HF. Common access to CA-125 may lead to widespread implementation of its assessment in clinical practice. However, there still is a need to establish reference ranges, specific to HF. Therefore, it is necessary to conduct multicentre and international studies to provide

Table 2. Summary of the latest studies involving CA-125 risk stratification

Author	Year	Type of study	N	Study population	Proposed CA 125 cut-off	Results
D'Aloia et al. [19]	2003	Observational, prospective	286	Congestive HFrEF	35 U/mL	Patients with CA 125 > 35 U/mL died or were hospitalized due to HF more often at 6-month follow-up
Zhuang et al. [36]	2014	Meta-analysis	4159 (23 studies)	Acute and chronic HF	Not specified	Higher CA-125 levels occurred in patients with a higher risk of short- and long-term mortality
Núñez et al. [32]	2016	Randomized, prospective, multicentre	380	Patients discharged for HF	35 U/mL	CA 125 strategy was found beneficial compared to the SOC in terms of decreasing the risk of 1-year death or AHF readmission
Li et al. [37]	2018	Meta-analysis	8401 (16 studies)	AHF	Not specified	Increased CA-125 values were linked with a higher incidence of HF readmission and risk of death
Soler et al. [16]	2020	Observational, prospective	2961	AHF with TR	Continuous	Higher CA-125 levels were found in patients who had a higher risk of long-term all-cause mortality, especially those with severe TR
Núñez et al. [33]	2020	Observational, prospective, multicentre	2516	Worsening HF	Quartiles, continuous	Patients with a higher risk of 1-year all-cause mortality had increased CA-125 levels, which were positively correlated with clinical indicators of congestion
de la Espriella et al. [35]	2021	Observational, prospective	60	Chronic HF and T2D	35 U/mL	Empagliflozin administration was linked with a decrease in CA-125 values with no modification in NT-proBNP trajectory
Lorenzo et al. [38]	2021	Observational, retrospective	1387	Patients discharged for AHF	35 U/mL	Higher levels of CA-125 in patients with AHF may identify the need for a prolonged hospitalization
Lourenço et al. [29]	2022	Observational, prospective	363	Patients admitted for AHF	35 U/mL, 60 U/mL	Early CA-125 assessment (> 10 days) after an AHF hospitalization may be potentially useful in patient management

AHF — acute heart failure; CA-125 — carbohydrate antigen 125; HF — heart failure; HFrEF — heart failure with reduced ejection fraction; NT-proBNP — N-terminal pro-B-type natriuretic peptide; SOC — standard of care; T2D — type 2 diabetes; TR — tricuspid regurgitation

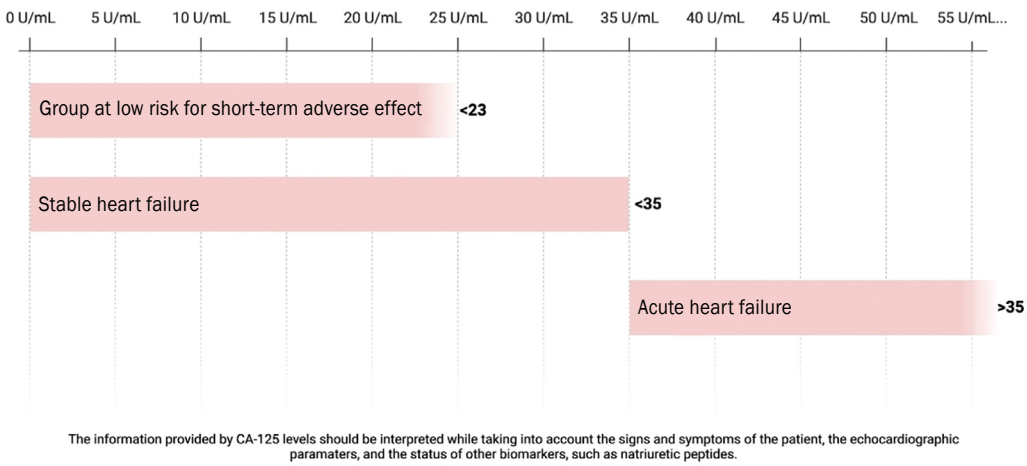

Figure 2. Heart failure cut-off groups according to CA-125 levels

an appropriate strategy regarding diagnosis and treatment monitoring.

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Conflict of interest

None declared.

Streszczenie

Wciąż trwają poszukiwania odpowiednich biomarkerów do monitorowania i leczenia pacjentów z niewydolnością serca (HF). Nowy biomarker — antygen węglowodanowy 125 (CA-125), powszechnie używany do tej pory w onkologii, okazał się przydatny w ocenie zastoju oraz procesów zapalnych w niewydolności serca. Jego podwyższone wartości są ściśle związane z upośledzoną funkcją prawej komory. Co więcej, podwyższone stężenie CA-125 jest związane z ponownymi hospitalizacjami z powodu HF oraz wyższym ryzykiem zgonu. W związku z tym, pojawiła się potrzeba stworzenia potencjalnej strategii leczenia, co zostało wykonane w badaniu CHANCE-HF. Pomimo zalet tego biomarkera, takich jak powszechny dostęp lub znacząco niski koszt oznaczenia, konieczne jest przeprowadzenie wieloośrodkowych i międzynarodowych badań, aby wprowadzić odpowiednie wytyczne.

Słowa kluczowe: niewydolność serca, ostra niewydolność serca, echokardiografia, biomarkery, praktyka kliniczna

Folia Cardiologica 2023; 18, 2: 65–70

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REVIEW ARTICLE

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Catheter ablation of the cavo-tricuspid isthmus as a bridging therapy for symptomatic atrial fibrillation?

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Abstract

Atrial fibrillation and atrial flutter are the most common supraventricular arrhythmias. These arrhythmias often coexist, approximately one third of patients with atrial fibrillation presenting episodes of atrial flutter. Many physicians have difficulty making a correct diagnosis based on the electrocardiogram. However, it is fundamental for the choice of management strategy. Each arrhythmia has a different background, different sensitivity to pharmacological treatment and different approach to non-pharmacological treatment.

Key words: atrial fibrillation, atrial flutter, arrhythmia

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Introduction

Atrial fibrillation (AF) is the most common supraventricular arrhythmia and occurs in as much as 2–4% of the general population [1]. In the coming years, its incidence is expected to increase by as much as 2–3 times due to increasing life expectancy in the general population and more frequent search for previously undiagnosed cases of AF. The second most common supraventricular arrhythmia is atrial flutter (AFl). Many multi-centre trials emphasise the comorbidity of these two arrhythmias. According to the 2020 European Society of Cardiology (ESC) Guidelines for the management of these arrhythmias, pharmacological treatment and surgical treatment (ablation) are used. Decision concerning surgical treatment depends on the centre and the experience of the operating physician who performs the surgery. The long waiting time for ablation leads to establishment of the arrhythmia and worse long-term prognosis [2]. In addition, the form of AF (paroxysmal, persistent or longstanding persistent) affects both early and long-term effects of pulmonary vein isolation [2, 3].

Pathogenesis and indications for treatment of atrial fibrillation

Pathogenic mechanisms of AF are related to focal beats from pulmonary veins or multiple microreentries in the left atrium (Figure 1). The basis of treatment is pharmacotherapy – antiarrhythmic drugs (**class I** — for patients with no comorbidities, with no organic heart diseases [propafenone, flecainide] or **class III** — for patients with an organic heart disease [amiodarone]). Pharmacotherapy is effective in stopping and preventing the recurrence of AF, but in some patients it leads to conversion to another supraventricular arrhythmia — AFl. An invasive method for treatment of AF is catheter ablation, the purpose of which is to achieve pulmonary vein isolation (PVI). It is recommended for patients in whom either a class I or class III antiarrhythmic drug was ineffective or for patients who do not tolerate therapy using these drugs (**class of recommendation I, level of evidence A**) [1]. Radiofrequency ablation — also known as classic ablation — is difficult to perform, takes a lot of time, involves prolonged fluoroscopy and is characterised

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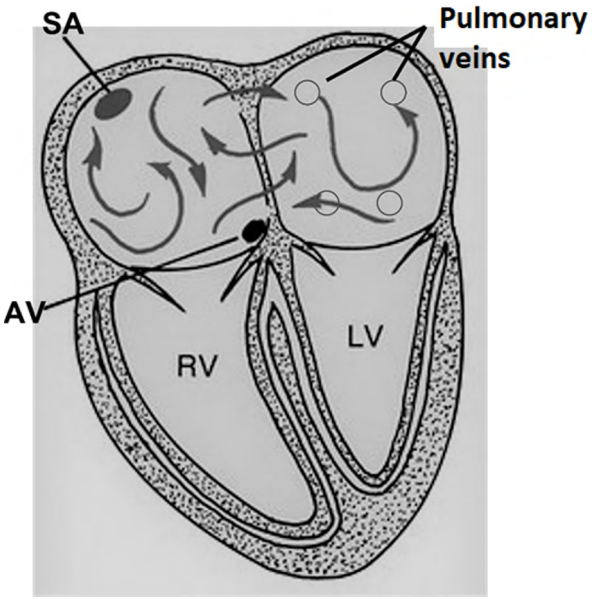

Figure 1. Atrial fibrillation — microreentry; AV — atrioventricular node; LV — left ventricular; RV — right ventricular; SA — sinoatrial node

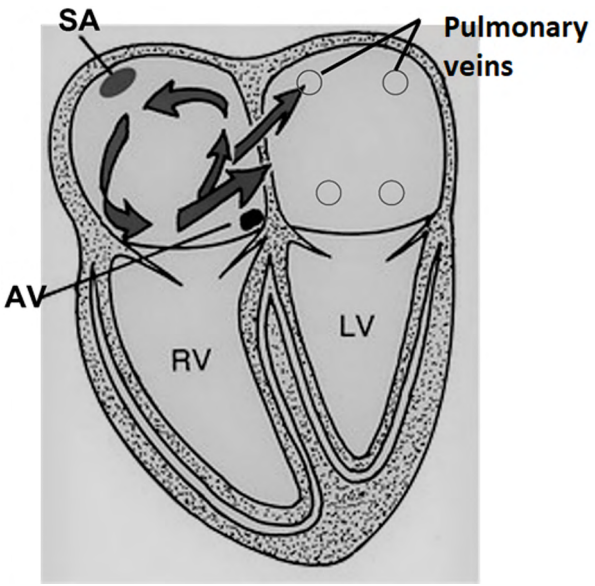

Figure 2. Atrial flutter — macroreentry; AV — atrioventricular node; LV — left ventricular; RV — right ventricular; SA — sinoatrial node

by a high rate of complications (pulmonary vein stenosis, post-ablation atrial tachycardia, atrioesophageal fistula). A newer method is balloon cryoablation of pulmonary vein ostia, which involves inducing left atrium tissue necrosis through a freezing/unfreezing process [2]. It is safer and associated with lower risk of complications. Both surgeries are characterised by comparable effectiveness in terms of maintaining sinus rhythm in long-term follow-up, i.e. 75% for paroxysmal and approx. 40% for persistent AF [2–4].

Pathogenesis and indications for treatment of atrial flutter

This arrhythmia is caused by macroreentry (Figure 2) in the right atrium, around the tricuspid ring. Antiarrhythmic treatment is ineffective for stopping and preventing the recurrence of this arrhythmia. The treatment of choice for AFl is radiofrequency ablation or cavotricuspid isthmus (CTI) cryoablation. It is indicated for poorly-tolerated recurring AFl flutter as well as AFl following pharmacological treatment of AF (**class of recommendation I, level of evidence B**) [1]. Ablation should also be considered for the first episode of AFl, and for well-tolerated and atypical AFl (**class of recommendation II, level of evidence A**) [1] The effectiveness of invasive treatment of AFl is nearly 100%.

Methods for preventing the recurrence of supraventricular arrhythmias

In the trial with the acronym of **STAR-AF II** (**Substrate and Trigger Ablation for Reduction of Atrial Fibrillation Trial-Part II**), Verma et al. [5] tried to answer the question of whether electrical isolation of pulmonary vein ostia is sufficient to maintain the sinus rhythm in long-term follow-up. The trial included 589 patients with persistent AF, who were assigned to the following groups: I — PVI ablation (67 patients), II — PVI ablation plus ablation of areas of fragmented atrial potentials (263 patients) and III — PVI ablation plus linear ablation of the left atrial roof and the mitral isthmus (259 patients). The follow-up duration was 18 months. The primary outcome was no documented recurrence of AF lasting longer than 30 seconds after ablation. In their results, the authors stress that the duration of the procedure was much shorter for group I compared to groups II and III ($p < 0.001$). During follow-up, no recurrence of AF was observed for 59% of patients from group I, 49% of patients from group II and 46% of patients from group III ($p = 0.15$). In their conclusions, the authors emphasise that for patients with persistent AF, no reduction in the frequency of recurrence of AF was observed when ablative lines were applied in the left atrium in addition to pulmonary vein isolation. The relatively low effectiveness of ablation may stem from mechanisms which lead to unfavourable remodelling and fibrosis of the left atrium, such as: atrial myocardium stretching, AF episodes, other supraventricular arrhythmias or activation of the renin-angiotensin-aldosterone system.

Another frequently encountered situation is the primary occurrence of typical AFl and periodic AF episodes. What procedure should be performed in such a case? This question was tackled by authors of a pilot study (**Cavotricuspid isthmus ablation among patients with persistent**

atrial fibrillation as a bridging therapy to maintain sinus rhythm — a pilot study) [6], in which a hypothesis was assumed that sinus rhythm may be achieved through cavo-tricuspid isthmus ablation combined with pharmacological treatment using an aldosterone receptor antagonist. Follow-up, with a mean duration of 13.9 months, included 64 patients, assigned to 3 groups: I – AFI (n = 34); II – AFI with a history of AF episodes (n = 13); III – persistent AF despite antiarrhythmic treatment (n = 17). All patients underwent CTI ablation. For group II, antiarrhythmic treatment was commenced for all patients after the surgery, while for group III, the previously used treatment was continued. In addition, an aldosterone receptor antagonist was used for some patients from group I and all patients from other groups. During long-term follow-up, AFI recurrence was noted in 3 patients from group I (8.8%) and 1 patient from group II (7.7%) over 5 months; 1 AF episode was also observed. In group III, there were 7 AF episodes; for these patients, either another pulmonary vein isolation procedure was performed or AF was considered permanent. The rate of effectiveness of procedures in individual groups was 91% vs. 85% vs. 59% respectively ($p < 0.05$), and was statistically significant. In their conclusions, the authors emphasise that CTI ablation combined with antiarrhythmic treatment and aldosterone receptor antagonists may be an effective bridge therapy for patients with persistent AF before the final pulmonary vein isolation. It can be used to maintain sinus rhythm. Further multi-centre trials are needed to confirm this hypothesis.

In the work by Bianco et al. [7], the authors studied the frequency of occurrence of AF following CTI-dependent AFI ablation. The study included 84 patients with no prior history of AF who underwent CTI-dependent AFI ablation. During follow-up, with a mean duration of 26 ± 18 months, AFI recurrence was observed in 10 patients (11.5%), while 45 patients (53.6%) experienced their first AF episode. Throughout the entire study, no predictor variables for occurrence of a new AF episode were identified. This means that elimination of CTI-dependent AFI does not prevent the occurrence of AF episodes. This effect has been observed in numerous multi-centre clinical trials which studied the development of atrial arrhythmias, including AF, following CTI ablation due to AFI.

In relation to that, the latest data suggests that preventive ablation of pulmonary vein ostia may be an effective strategy for preventing new AF episodes in patients who undergo CTI-dependent AFI ablation. This is evidenced by results of the **PREVENT AF I** study [8]. Follow-up included 50 patients with CTI-dependent AFI with no prior history of AF and randomised into two groups, at a 1:1 ratio: I — CTI — only CTI ablation and II — CTI + PVI — CTI ablation combined with PVI cryoablation. It was emphasised in results that new AF occurred with statistically significantly lower frequency in the CTI + PVI group vs. the CTI group (12% vs. 52% respectively; $p = 0.003$). In addition, Romanov et al. [8] presented in their work the results of a prolonged 3-year follow-up for the **PREVENT AF I** study, noting the significantly lower rate of occurrence of any atrial arrhythmia in the CTI + PVI group compared to the CTI group (48% vs. 20%; $p = 0.01$).

Conclusions

On the basis of the latest ESC/Polish Cardiac Society Guidelines for management of AF, CTI ablation may be considered during AF ablation for patients with history of typical AFI, or if typical AFI was induced during ablation of pulmonary vein ostia (**class of recommendation IIb, level of evidence B**) [1]. However, what procedure should be used when the situation is reversed? Can/should simultaneous ablation of pulmonary vein ostia be considered in case of CTI ablation due to typical AFI and AF occurrence? On one hand, this would constitute a single, if prolonged, procedure, which could treat the patient, or at least minimise the frequency of occurrence of episodes of supraventricular arrhythmias: AFI and AF. However, given the lack of data from clinical trials, such strategy is not, at the moment, justified. For this reason, currently neither application of additional ablative lines in the left atrium nor PVI ablation are performed during CTI ablation. To conclude this article, it should also be emphasised that there is evidence that CTI-dependent AFI ablation is a safe and effective procedure, although it only solves the clinical problem of patients with isolated AFI. In long-term follow-up, the occurrence of AF following CTI ablation is frequently observed. On the basis of the presented results of trials and studies, the authors remain of the opinion that there is still no sufficient evidence to recommend the combination of CTI and PVI ablation in treatment of isolated AFI to prevent the occurrence of AF. More prospective multi-centre trials, with more patients and longer follow-up, are needed to assess the benefits of application of this treatment method.

Conflict of interest

None declared.

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Ablacja cieśni trójdzielno-żylnej jako terapia pomostowa w leczeniu objawowego migotania przedsionków?

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Streszczenie

Migotanie przedsionków (AF, *atrial fibrillation*) oraz trzepotanie przedsionków (AFl, *atrial flutter*) są najczęstszymi arytmiami nadkomorowymi. Arytmie te nierzadko ze sobą współistnieją — u mniej więcej co trzeciego pacjenta z migotaniem przedsionków rejestrowane są epizody trzepotania przedsionków. Wielu lekarzy nie zwraca uwagi na to, z jaką postacią arytmii nadkomorowej mamy do czynienia. Jest to jednak kluczowe z punktu widzenia wyboru strategii postępowania. Obie arytmie mają inne podłoże, inną wrażliwość na stosowane leczenie farmakologiczne oraz wymagają odmiennego podejścia do leczenia niefarmakologicznego.

Słowa kluczowe: migotanie przedsionków, trzepotanie przedsionków, arytmia

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Wstęp

Migotanie przedsionków (AF, *atrial fibrillation*) jest najczęstszą arytmią pochodzenia nadkomorowego, występującą nawet u 2–4% ogólnej populacji [1]. W kolejnych latach spodziewany jest nawet 2–3 krotny wzrost częstości jej występowania z powodu wydłużającego się czasu życia w populacji ogólnej oraz częstszego poszukiwania nierozpoznanych dotychczas przypadków AF. Drugą co do częstości występowania tachyarytmii nadkomorowych jest trzepotanie przedsionków przedsionków (AFl, *atrial flutter*). W wielu badaniach wieloośrodkowych podkreśla się współwystępowanie obydwu tych arytmii. Według wytycznych Europejskiego Towarzystwa Kardiologicznego (ESC, *European Society of Cardiology*) z 2020 roku dotyczących leczenia wyżej wymienionych arytmii stosuje się leczenie farmakologiczne oraz zabiegowe (ablacja). Decyzja o podjętym leczeniu zabiegowym zależy od ośrodka jak i doświadczenia operatora wykonującego zabieg. Długi czas oczekiwania na wykonanie ablacji skutkuje utrwaleniem się arytmii i gorszym rokowaniem odległym [2]. Dodatkowo, postać migotania przedsionków (napadowe, przetrwałe i długotrwające przetrwałe) wpływa na wczesny jak i długotrwały efekt izolacji żył płucnych [2, 3].

Mechanizm powstawania, wskazania do leczenia migotania przedsionków

Mechanizmem powstawania AF są pobudzenia ogniskowe pochodzące z żył płucnych bądź liczne fale pobudzenia nawrotnego w lewym przedsionku (*microreentry*, ryc. 1). Podstawą leczenia jest farmakoterapia — leki antyarytmiczne (**klasa I** — u chorych bez obciążeń, bez organicznej choroby serca [propafenon, flekainid] oraz **klasa III** — u chorych z organiczną chorobą serca [amiodaron]). Leczenie to jest skuteczne w przerywaniu i zapobieganiu nawrotom AF, jednak u części chorych powoduje konwersję do innej arytmii nadkomorowej — AFl. Inwazyjną metodą leczenia AF jest ablacja przezcewnikowa, której celem jest uzyskanie izolacji elektrycznej żył płucnych (PVI, *pulmonary vein isolation*). Zalecana jest u osób, u których jeden z leków antyarytmicznych klasy I lub III był nieskuteczny, bądź chorzy

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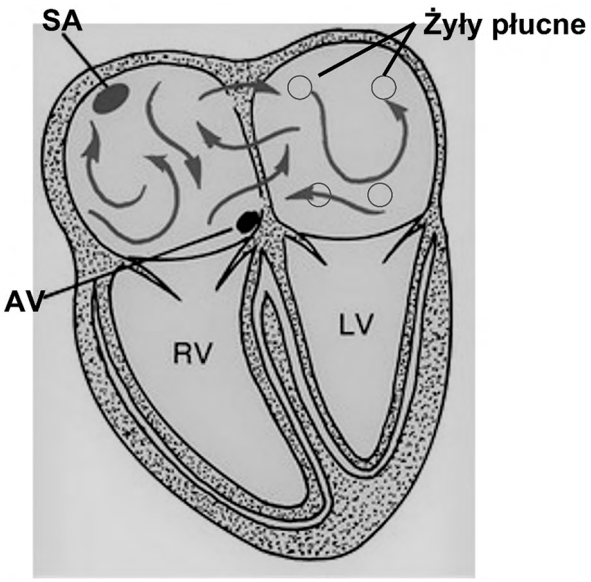
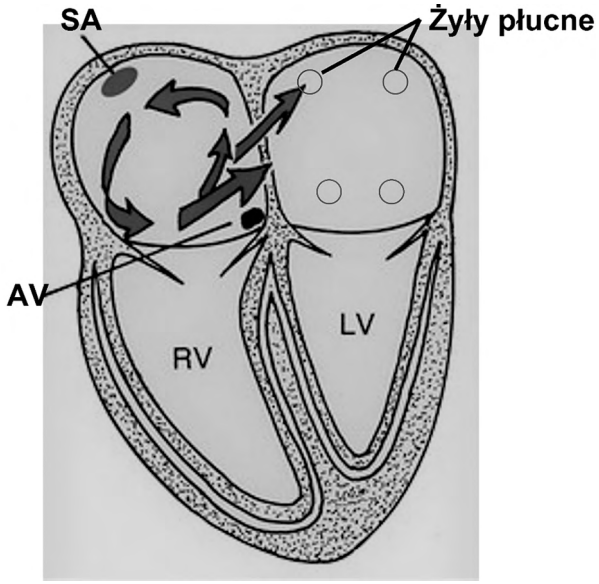


Rycina 1. Migotanie przedsionków — fala pobudzenia nawrotnego (*macroreentry*); AV (*atrioventricular*) — węzeł przedsionkowo-komorowy; LV (*left ventricular*) — lewa komora; RV (*right ventricular*) — prawa komora; SA (*sinoatrial*) — węzeł zatokowo-przedsionkowy



Rycina 2. Trzepotanie przedsionków — fala pobudzenia nawrotnego (*microreentry*); AV (*atrioventricular*) — węzeł przedsionkowo-komorowy; LV (*left ventricular*) — lewa komora; RV (*right ventricular*) — prawa komora; SA (*sinoatrial*) — węzeł zatokowo-przedsionkowy

ci nie tolerują takiego leczenia (**klasa zaleceń I, poziom wiarygodności A**) [1]. Ablacja prądem o częstotliwości radiowej (RF, radio frequency) — tzw. ablacja klasyczna — jest trudna, czasochłonna, związana z długim czasem fluoroskopii i obarczona dużym odsetkiem powikłań (stenoza żył płucnych, poablacyjne częstoskurcze przedsionkowe, przetoka przełykowo-przedsionkowa). Nowszą metodą jest krioablacja balonowa ujść żył płucnych, polegająca na wytworzeniu martwicy tkanki lewego przedsionka na skutek procesu zamrażania/rozmrażania [2]. Jest to metoda bezpieczniejsza, związana z mniejszym ryzykiem powikłań. Obydwa rodzaje zabiegów mają porównywalną skuteczność w utrzymaniu rytmu zatokowego w obserwacji odległej — 75% w napadowym i około 40% w przetrwałym AF [2–4].

Mechanizm powstawania, wskazania do leczenia trzepotania przedsionków

Arytmia ta powstaje na skutek występowania dużej pętli pobudzenia nawrotnego (*macroreentry*, ryc. 2) krążącej w prawym przedsionku, wokół pierścienia trójdzielnego. Leczenie antyarytmiczne jest mało skuteczne do przerywania i zapobiegania nawrotom tej arytmii. Leczeniem z wyboru AFl jest ablacja RF bądź krioablacja cieśni trójdzielno-żylnej (CTI, *cavotricuspid isthmus*). Wskazaniem do niej jest źle tolerowane nawracające trzepotanie przedsionków oraz trzepotanie przedsionków po leczeniu farmakologicznym migotania przedsionków (**klasa zaleceń I, poziom wiarygodności B**) [1]. Ablację należy również rozważyć w przypadku pierwszego epizodu AFl, dobrze tolerowanego oraz atypowego AFl (**klasa zaleceń II, poziom wiarygodności A**) [1]. Skuteczność leczenia inwazyjnego trzepotania przedsionków wynosi blisko 100%.

Metody zapobiegania nawrotom arytmii nadkomorowych

Verma i wsp. [5] w badaniu o akronimie **STAR-AF II** (***Substrate and Trigger Ablation for Reduction of Atrial Fibrillation Trial-Part II***) starali się odpowiedzieć na pytanie czy izolacja elektryczna ujść żył płucnych jest wystarczająca do utrzymania rytmu zatokowego w obserwacji długoterminowej. Do badania włączono 589 pacjentów z przetrwałym migotaniem przedsionków i przydzielono do grup: I — ablacji PVI (67 pacjentów), II — ablacji PVI plus ablację stref pofragmentowanych potencjałów przedsionkowych (263 pacjentów) i III — ablacji PVI plus ablacji linijnej szczytu lewego przedsionka i cieśni mitralnej (259 pacjentów). Czas obserwacji wynosił 18 miesięcy. Pierwszorzędowym punktem końcowym był brak jakichkolwiek udokumentowanych nawrotów migotania przedsionków trwających dłużej niż 30 sekund po ablacji. W wynikach autorzy podkreślają, że czas trwania procedury był znacznie krótszy w grupie I niż w grupie II i III ($p < 0{,}001$). W trakcie obserwacji bez nawrotu migotania przedsionków było: 59% pacjentów z grupy I; 49% pacjentów z grupy II i 46% pacjentów z grupy III ($p = 0{,}15$). We wnioskach autorzy podkreślają, że wśród pacjentów z przetrwałym migotaniem przedsionków nie stwierdzono

zmniejszenia częstości nawrotów migotania przedsionków, gdy oprócz izolacji żył płucnych przeprowadzano dodatkowo linie ablacyjne w lewym przedsionku. Względnie niska skuteczność ablacji może wynikać z mechanizmów, wpływających na niekorzystny remodeling i włóknienie lewego przedsionka, takich jak: rozciąganie miokardium przedsionka, epizody AF, inne nadkomorowe arytmie bądź aktywacja układu renina–angiotensyna–aldosteron.

Inną, często spotykaną sytuacją jest występowanie pierwotnie typowego trzepotania przedsionków oraz okresowo napadów migotania przedsionków. Jaki zabieg należałoby wtedy wykonać? Na to pytanie próbowali odpowiedzieć autorzy badania pilotażowego (***Cavotricuspid isthmus ablation among patients with persistent atrial fibrillation as a bridging therapy to maintain sinus rhythm — a pilot study***) [6], w którym przyjęto hipotezę, że uzyskanie rytmu zatokowego można osiągnąć poprzez ablacje cieśni trójdzielno-żylnej połączoną z leczeniem farmakologicznym antagonistą receptora aldosteronowego. Obserwacją, trwającą średnio 13,9 miesiąca, objęto 64 pacjentów, których przydzielono do 3 grup: I — AFl (n = 34); II — AFl z napadami AF w wywiadzie (n = 13); III — przetrwałe AF mimo leczenia antyarytmicznego (n = 17). U wszystkich pacjentów wykonano ablację CTI. W grupie II po zabiegu u wszystkich chorych rozpoczęto leczenie antyarytmiczne, a w grupie III kontynuowano dotychczasowe leczenie. Dodatkowo u części chorych z grupy I oraz u wszystkich z pozostałych grup stosowano antagonistę receptora aldosteronowego. Podczas obserwacji odległej nawrót AFl stwierdzono: u 3 pacjentów z grupy I (8,8%), u 1 pacjenta z grupy II (7,7%) w ciągu 5 miesięcy oraz 1 epizod AF. W grupie III wystąpiło 7 epizodów AF — u chorych tych wykonano kolejny zabieg izolacji żył płucnych lub uznano migotanie przedsionków za utrwalone. Odsetek skuteczności zabiegów w poszczególnych grupach wyniósł odpowiednio: 91% vs. 85% vs. 59%; ($p < 0,05$) i był istotny statystycznie. We wnioskach autorzy podkreślają, że ablacja CTI w połączeniu z leczeniem antyarytmicznym i antagonistami receptora aldosteronowego może być skuteczną terapią pomostową u chorych w przetrwałym AF przed ostateczną izolacją żył płucnych. Postępowanie takie może być stosowane celem utrzymania rytmu zatokowego. Do potwierdzenia tej hipotezy potrzebne są dalsze badania wieloośrodkowe.

W pracy Bianco i wsp. [7] autorzy badali częstość występowania AF po ablacji AFl zależnego od CTI. Do badania włączono 84 pacjentów bez wcześniejszej historii AF, poddanych ablacji AFl zależnego od CTI. W trakcie obserwacji trwającej średnio 26 ± 18 miesięcy, 10 pacjentów (11,5%) miało nawrót AFl, a u 45 (53,6%) stwierdzono pierwszy epizod AF. Podczas całego badania nie zidentyfikowano zmiennych będących predyktorami wystąpienia nowego epizodu AF. Zatem eliminacja AFl zależnego od CTI, nie zapobiega występowaniu epizodów AF. Efekt ten obserwowano w licznych wieloośrodkowych badaniach klinicznych badających powstawanie arytmii przedsionkowych, w tym AF, po ablacji CTI z powodu AFl.

W związku z tym, najnowsze dane sugerują, że profilaktyczna ablacja ujść żył płucnych może być skuteczną strategią zapobiegania nowym epizodom AF u pacjentów poddawanych ablacji AFl zależnego od CTI. Dowodem na to są wyniki badania **PREVENT AF I** [8]. Obserwacji poddano 50 pacjentów z AFl zależnym od CTI bez wcześniejszej historii AF i zrandomizowano do dwóch grup w stosunku 1:1: I — CTI — ablacji wyłącznie CTI oraz II — CTI + PVI — ablacji CTI połączonej z krioablacją PVI. W wynikach podkreślano, że nowe migotanie przedsionków wystąpiło istotnie statystycznie rzadziej w grupie pacjentów poddanych CTI + PVI vs. CTI i wynosiło odpowiednio (12% *vs.* 52%, $p = 0,003$). Dodatkowo Romanov i wsp. [8] w swojej pracy przedstawił wyniki przedłużonej obserwacji 3-letniej badania **PREVENT AF I**, stwierdzając znacząco mniejszy odsetek występowania jakiejkolwiek arytmii przedsionkowej w grupie CTI + PVI w porównaniu z grupą CTI (48% vs. 20%, $p = 0,01$).

Wnioski

Na podstawie najnowszych wytycznych ESC/PTK leczenia migotania przedsionków, podczas ablacji AF, można rozważyć ablację CTI u chorego z wywiadem typowego AFl, bądź jeśli podczas ablacji ujść żył płucnych indukowano typowe AFl (**klasa zaleceń IIb, poziom wiarygodności B**) [1]. Ale jakie może być postępowanie, gdy mamy do czynienia z sytuacją odwrotną? Czy w razie ablacji CTI z powodu typowego AFl oraz wystąpienia AF można/należy jednoczasowo rozważyć ablację ujść żył płucnych? Z jednej strony byłaby to jedna, aczkolwiek wydłużona procedura, która mogłaby pacjenta wyleczyć, a przynajmniej zminimalizować częstość występowania napadów arytmii nadkomorowych: AFl oraz AF. Jednakże, przy braku danych z badań klinicznych, taka strategia postępowania nie ma aktualnie uzasadnienia. Dlatego też na chwilę obecną podczas ablacji CTI nie wykonuje się dodatkowych linii ablacyjnych w lewym przedsionku ani ablacji PVI. Podsumowując powyższy artykuł należy podkreślić także, że istnieją dowody na to, że ablacja AFl zależnego od CTI jest bezpieczną i skuteczną procedurą, aczkolwiek rozwiązuje jedynie problem kliniczny chorego z izolowanym AFl. Często w obserwacji długoterminowej obserwuje się występowanie AF po ablacji CTI. Na podstawie przedstawionych wyników badań autorzy uważają, że wciąż nie ma wystarczających dowodów, aby zalecać połączenie ablacji CTI oraz PVI w leczeniu samego AFl w celu zapobiegania wystąpienia AF. Potrzebne są kolejne prospektywne badania wieloośrodkowe z większą liczbą pacjentów i dłuższą obserwacją do oceny korzyści płynących z zastosowania takiej metody leczenia.

Konflikt interesów

Nie zgłoszono.

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Atrial tachycardia or ”atypical” atrial flutter? Clinician’s dilemmas resolved by electrophysiologist

Częstoskurcz przedsionkowy czy „atypowe” trzepotanie przedsionków? Dylematy klinicysty rozstrzygnięte przez elektrofizjologa

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Abstract

We present a case report of a patient who underwent ablation of “atypical” atrial flutter. In the surface electrocardiogram, atrial tachycardia was initially diagnosed. As a result of electrophysiological mapping using the CARTOPRIME module, the diagnosis was verified and “atypical” atrial flutter was diagnosed, whose propagation wave in the right atrium met the criteria for the diagnosis of typical atrial flutter.

Key words: arrhythmia, atrial flutter, ablation, electrophysiological examination

Folia Cardiologica 2023; 18, 2: 79–83

Case report

A 60-year-old patient with post-myocardial infarction heart failure, after multiple myocardial infarctions, following coronary artery bypass surgery in 2005, after implantation of a cardioverter-defibrillator for the primary prevention of sudden cardiac death in 2017, and with paroxysmal atrial fibrillation, was admitted to the Department of Cardiology for worsening symptoms of circulatory failure in the course of persistent supraventricular arrhythmia, which was initially classified as atrial tachycardia. A 12-lead surface electrocardiogram (ECG) recorded atrial arrhythmia with a cycle length of 580 ms with negative P-waves in leads II, III and aVF, positive ones in aVR and aVL, and a flat P-wave in lead I, which indicated a right atrial origin of the arrhythmia.

The ECG recordings showed an isoelectric line between the P waves. The ECG also recorded Q-waves in leads II, III, aVF indicative of a history of inferior wall myocardial infarction (MI) (Figure 1) . During outpatient treatment, the patient’s dose of beta-blockers (metoprolol) was escalated, achieving the slowing of ventricular rhythm without affecting the atrial arrhythmia cycle length. Echocardiography revealed enlargement of the right atrium (right atrial volume index — 72 mL/m^2), left atrium (left atrial volume index — 62 mL/m^2) and right ventricle, left ventricular segmental wall motion abnormalities in the inferior, inferolateral and interventricular septum walls, hypokinesis of the remaining left ventricular walls with an ejection fraction of 26%. At the Department of Cardiology, the patient was scheduled for an electrophysiology study and an attempt to ablate

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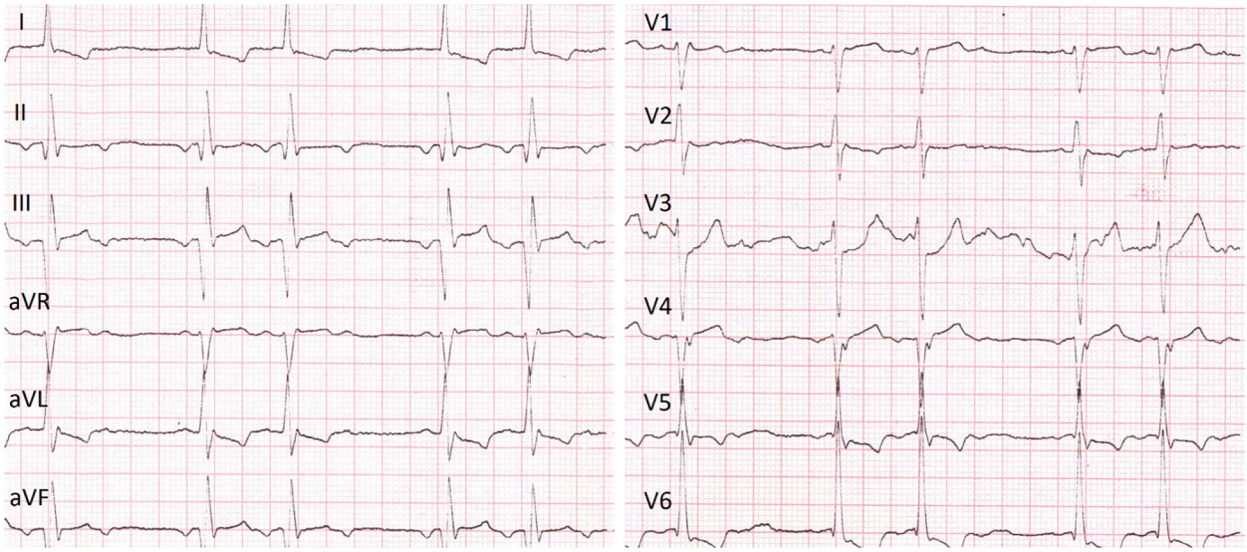

Figure 1. The 12-lead electrocardiographic recording before atrial tachycardia/atrial flutter

the arrhythmia. A transesophageal echocardiogram was performed before the ablation procedure, ruling out the presence of thrombi in the cardiac chambers. An electrophysiological study was performed using a decapolar electrode, which was inserted into the coronary sinus, and a PentaRay electrode (Biosense Webster, Inc., Diamond Bar, CA, USA). Moreover, an electroanatomical map of the right atrium was performed using the CARTO system (Biosense Webster, Inc., Diamond Bar, CA, USA). Due to the risk of arrhythmia termination due to irritation of the right atrial wall with a mapping electrode, the right atrial roof was not mapped after a full cycle length of arrhythmia was achieved. Then, using the Coherent CARTOPRIME™ module of the CARTO system, the extensive low-voltage zone was localized on the lateral wall of the right atrium and the arrhythmia cycle length was mapped (Figure 2A and 2B). Figure 2 shows the propagation waves of re-entry arrhythmia propagating counterclockwise around the tricuspid annulus. Very slow conduction of the arrhythmia wave was recorded on the lateral wall in the low-voltage zone. After the analysis of arrhythmia wave propagation around the tricuspid valve annulus, the patient was diagnosed with atrial flutter (AFl) and arrhythmia wave propagation that is specific to typical atrial flutter. Subsequently, a Smarttouch SF electrode (Biosense Webster, Inc., Diamond Bar, CA, USA) was inserted and an application was made at the site of slowed conduction on the lateral wall of the right atrium, obtaining termination of the arrhythmia and the return of sinus rhythm (Figures 3 and 4). Then, several consolidation applications were made to connect the free conduction zone to the tricuspid valve annulus. Currently, the patient is under the constant care of the Cardiac Rhythm Disorder Centre and no recurrence of arrhythmia has been recorded.

Discussion

According to current guidelines, the ECG-based diagnosis of unifocal atrial tachycardia (AT) is made when there are P waves of identical morphology that is different from the morphology of the P waves of sinus rhythm, there is an isoelectric line between P waves in the limb leads, the frequency of P waves is in the range from 100/min. (600 ms cycle length) to 250/min (240 ms cycle length), usually above 140/min. The criteria accept P-wave irregularity. The PQ interval exceeds 100 ms, the frequency of the QRS complexes corresponds to the frequency of the atrial rhythm or is lower due to atrioventricular conduction disorders. The morphology of the QRS complexes remains unchanged except for intraventricular conduction aberrations or intraventricular conduction disorders. AT should be differentiated from typical or atypical AFl. The diagnosis of AT is supported by both the presence of an isoelectric line between P waves and P-wave irregularity [1]. AT should be considered if AFl is suspected, especially if the P-wave or F-wave frequency is in the range of 200–250/min.

In the patient in question, the criteria for the diagnosis of AT were undoubtedly met in the recorded ECG before the ablation procedure: P waves had identical morphology, were positive in aVR and aVL leads, negative in II, III and aVF leads, flat in I lead, and their frequency was approximately

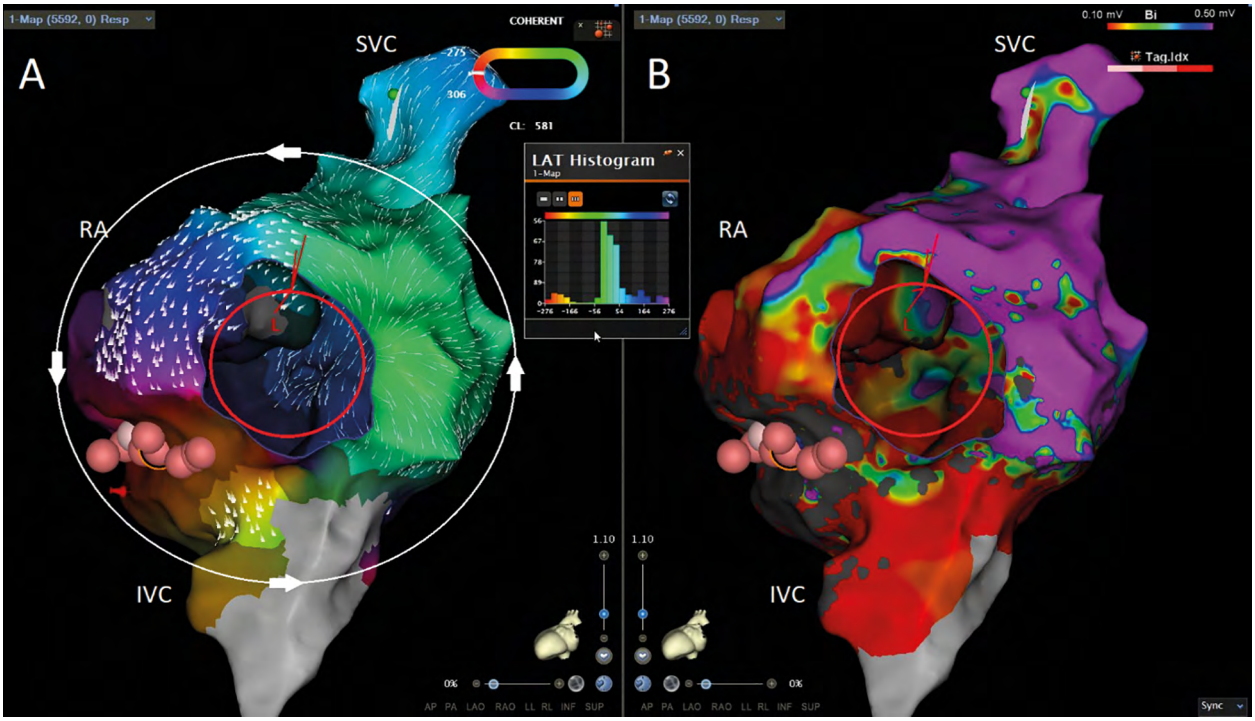

Figure 2. The electroanatomic map of the right atrium in the left oblique projection. In the activation map (**A**), the electrical excitation propagation waveform (white circle) revolves around the tricuspid valve annulus (red circle). On the potential map (**B**), red and gray mark the zones of slowed conduction of electrical excitation; IVC — inferior vena cava; RA — right atrium; SVC — superior vena cava

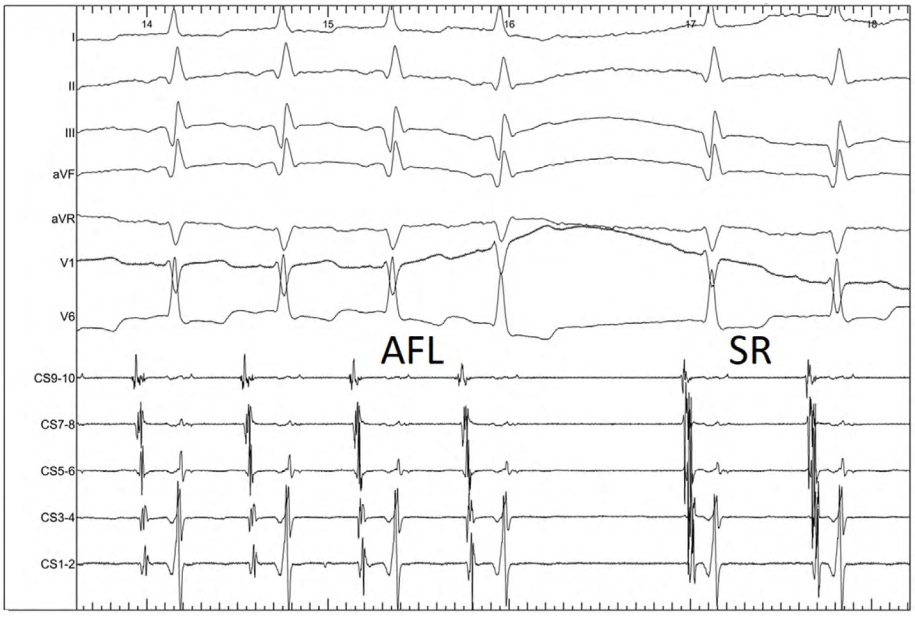

Figure 3. The surface and intracardiac electrocardiographic recordings from the coronary sinus. The recorded moment of termination of atrial flutter (AFL) and return of sinus rhythm (SR)

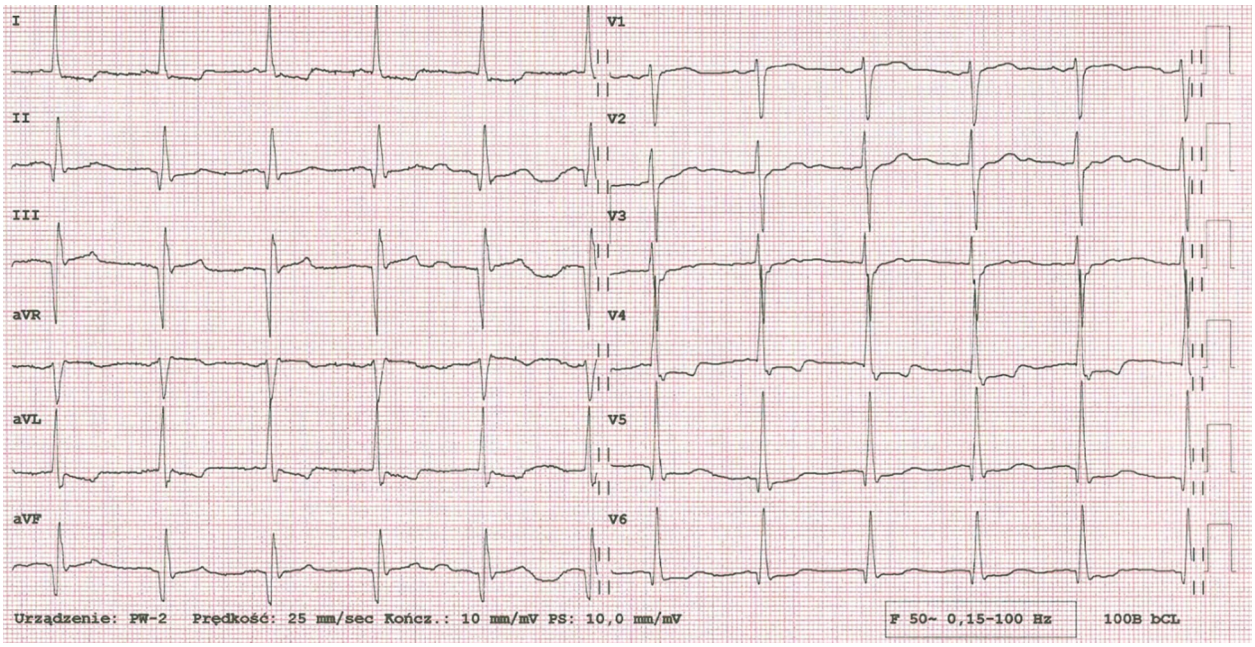

Figure 4. The 12-lead surface electrocardiographic recording. Sinus rhythm after ablation. Attention should be drawn to the low voltage P waves

103/min. and did not correspond to the P waves of sinus rhythm, there was an isoelectric line between P waves, and the PQ interval was approximately 160 ms. Despite meeting the criteria for AT on the ECG, the electrophysiological study ruled out such a diagnosis and definitively diagnosed slow AFl.

In the rare clinical situation presented here, the slowed conduction of electrical excitation within the lateral part of the right atrium was likely the result of a history of MI or the result of cannulation of the right atrium during coronary artery bypass surgery.

The case presented here provided significant diagnostic difficulties in differentiating AT from AFl. According to the European Society of Cardiology guidelines, emergency treatment for AT includes pharmacotherapy with adenosine (recommendation class IIa, level B), beta-blockers, non-dihydropyridine calcium channel antagonists (recommendation class IIa, level C). Other antiarrhythmics such as flecainide, propafenone, amiodarone can be considered (recommendation class IIb, level C). In the case of hemodynamic instability or ineffectiveness of pharmacotherapy, electrical cardioversion is recommended (recommendation class I, level B). In chronic treatment, if tachycardia-induced cardiomyopathy is present, the highest class of recommendations is percutaneous ablation (recommendation class I, level B). The guidelines do not recommend anticoagulant treatment in AT patients [2]. In contrast, the use of thromboprophylaxis in the case of AFl is similar to that in patients with atrial fibrillation due to the risk of formation of thrombotic material, which is reflected in important clinical decisions based on the differentiation of these arrhythmias (recommendation class I, level B) [2]. In patients with heart failure, there is often a clear link between the presence of arrhythmias and cardiovascular decompensation. Therefore, antiarrhythmic treatment — including pharmacotherapy, electrical cardioversion or ablation — should not be delayed [3]. In the case of the coexistence of diagnosed heart failure with reduced ejection fraction, there are important limitations in terms of pharmacotherapy. Heart failure with reduced ejection fraction is a contraindication to the use of diltiazem and verapamil [3].

Conflict of interest

None declared.

Streszczenie

W niniejszym tekście przedstawiono opis przypadku pacjenta, u którego wykonano ablację „atypowego" trzepotania przedsionków. W powierzchniowym zapisie elektrokardiograficznym początkowo rozpoznano częstoskurcz przedsionkowy. W wyniku badania i mapowania elektrofizjologicznego z wykorzystaniem modułu CARTOPRIME zweryfikowano diagnozę, rozpoznając „atypowe" trzepotanie przedsionków, którego fala propagacji w prawym przedsionku spełniała kryteria rozpoznania typowego trzepotania przedsionków.

Słowa kluczowe: arytmia, trzepotanie przedsionków, ablacja, badanie elektrofizjologiczne.

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Complications of percutaneous coronary intervention in 70 years old patient with multivessel coronary artery disease and prostate cancer

Powikłania przezskórnej interwencji wieńcowej u 70-letniego pacjenta z wielonaczyniową chorobą wieńcową i rakiem prostaty

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Abstract

A 70-year-old man with chronic coronary artery disease (CAD), disseminated atherosclerosis, heart failure with mildly reduced ejection fraction, hypertension, diabetes mellitus type 2 and prostate cancer was admitted to the Department of Cardiology because of low effort tolerance for further cardiological assessment before chemotherapy of prostate cancer. Due to the progression of CAD in coronarography, he was consulted at the Heart Team meeting and classified for two-step percutaneous coronary intervention (PCI). After PCI of the left anterior descending artery, he had a myocardial infarction with the ejection fraction decreased from 43% to 28%.

Key words: percutaneous coronary intervention, multivessel coronary artery disease, artherosclerosis, heart failure, myocardial infarction, oncology, prostate cancer

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Introduction

The multivessel disease is characterized by significant stenosis ≥ 50 % in at least two major coronary arteries. The strategies of revascularization are percutaneous coronary intervention (PCI) and coronary artery bypass grafting and both are effective in extending human lives. The selection of a method should be discussed in every case separately [1]. Every invasive treatment is connected with risk and adverse effects. One of them is myocardial infarction type 4a which is related to coronary procedure events and includes elevation of cardiac troponin greater than 5 times the 99th percentile, electrocardiogram and imaging examination changes within 48 hours after PCI. In these patients, the optimal pharmacotherapy to reduce cardiovascular risk in the future must be established.

Case report

A 70-year-old man with chronic coronary artery disease (CAD), after past myocardial infarction, with disseminated atherosclerosis and heart failure (HF) with mildly reduced ejection fraction (EF) was admitted to the Department of Cardiology for further cardiological assessment before chemotherapy of prostate cancer due to a decrease of effort tolerance.

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Furthermore, this patient had a history of hypertension, diabetes mellitus (DM) type 2 and prostate cancer diagnosed a few months earlier. In 2004 he underwent PCI of the right coronary artery (RCA) with 1 drug-eluting stent implantation in a course of inferior myocardial infarction

On the hospital admission, the patient was haemodynamically stable and denied any chest pain. However, he reported low exercise tolerance and shortness of breath after walking about 50 meters and periodically feelings of heart palpitations. Physical examination showed heart rhytm 65/min, blood pressure 114/68 mm Hg, and normal vesicular breath sounds without pulmonary congestion. Laboratory tests revealed elevated level of N-terminal pro-B-type natriuretic peptide 585.7 pg/mL, HbA1C 9.4% and normal concentration of high-sensitive troponin 17 ng/L. In electrocardiogram there was regular sinus rhythm, features of enlargement of the left atrium and previous inferior myocardial infarction. The echocardiography examination showed a mildly decreased ejection fraction of the left ventricle (EF 43%), left ventricle hypertrophy, grade 1 diastolic dysfunction (impaired relaxation), mild mitral and tricuspid regurgitation.

The patient underwent the coronarography which revealed progression of CAD, left anterior descending coronary artery (LAD) with 90% stenosis, ramus circumflexus dexter with 99% stenosis. He was consulted at the Heart Team meeting and classified for PCI of LAD and to repeat PCI of RCA. PCI of LAD and diagonal artery with rotablation and implantation 2 drug-eluting stent stents were made urgently. The procedure was technically difficult because of advanced, massive atherosclerosis in coronary arteries. After PCI the patient reported a new pain in the chest; also, an elevated level of highly sensitive troponin was observed, which was typical for the acute coronary syndrome. Myocardial infarction type 4a was recognized (after PCI) which was also confirmed by depressed ST segment in the inferior and lateral leads in electrocardiogram. An echocardiographic examination showed a reduction of left ventricular ejection fraction to 28% and new regional myocardial contractility abnormalities. The patient was consulted with an invasive cardiologist and was classified to continue conservative treatment because of the high risk of the repeated invasive procedure.

The patient received typical complex pharmacotherapy for acute coronary syndrome and heart failure. Furthermore, after consultation with a diabetologist, the treatment of DM was modified — the insulin therapy was maintained and added were flozin and metformin. The left ventricular ejection fraction improved to 41% during hospitalization. After improvement of vital signs, the patient was discharged from the hospital without severe contraindications to chemotherapy for prostate cancer.

Conclusions

A patient with multivessel CAD, before planned chemotherapy of prostate cancer, was qualified for PCI LAD, which conflicted with acute myocardial infarction type 4a. Due to prostate cancer, oncological treatment was a priority and after an improvement of the left ventricular ejection fraction, the patient should immediately have chemotherapy without planned new percutaneous intervention.

Discussion

Cardiotoxicity is a very common adverse action of cancer chemotherapy and a frequent cause of death [2]. One of the first reports about cardiotoxicity in oncological treatment was proved already in 1973 [3]. It revealed most of the ten abnormalities in electrocardiography or congestive HF. Echocardiographic measurement of ejection fraction is important to assess systolic function before and after chemotherapy [4, 5]. Some patients have a higher risk of HF during oncological therapy. The patient-related risk factors are age, sex, hypertension, HF, previous cardiovascular disease, obesity and DM [6, 7]. In this case report, the patient had chronic CAD and HF with reduced EF which was a poor prognosis. Due to the decrease of EF after PCI of LAD, the patient was disqualified from PCI RCA and only pharmacotherapy treatment of chronic ischaemic heart disease was possible. The most important is to value the risks and benefits of chemotherapy in patients with cardiovascular diseases and HF. It is proved, however, that benefits of chemotherapy outweighed the risk of cardiac dysfunction [8].

Conflict of interest

None declared.

Funding

None.

Streszczenie

Siedemdziesięcioletni mężczyzna z przewlekłą chorobą wieńcową (CAD), rozsianą miażdżycą, niewydolnością serca z umiarkowanie obniżoną frakcją wyrzutową, nadciśnieniem tętniczym, cukrzycą typu 2 i rakiem prostaty został przyjęty do Kliniki Kardiologii z powodu niskiej tolerancji wysiłku w celu dalszej oceny kardiologicznej przed chemioterapią raka prostaty. Po wykonaniu koronarografii, która ujawniła progresję CAD, pacjent został skonsultowany na spotkaniu *Heart Team* i zakwalifikowany do dwuetapowej przezskórnej interwencji wieńcowej (PCI). Po PCI gałęzi przedniej zstępującej lewej tętnicy wieńcowej wystąpił zawał serca oraz obniżenie frakcji wyrzutowej lewej komory z 43% do 28%.

Słowa kluczowe: przezskórna interwencja wieńcowa, wielonaczyniowa choroba wieńcowa, miażdżyca, niewydolność serca, zawał serca, onkologia, rak prostaty

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A rare case of coexistence of a patent ductus arteriosus and a congenital carotid arteriovenous fistula in a 7-year-old girl

Rzadki przypadek współistnienia przetrwałego przewodu tętniczego i wrodzonej przetoki tętniczo-żylnej szyjnej u 7-letniej dziewczynki

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Abstract

A 7-year-old girl, who underwent a hemodynamically significant patent ductus arteriosus closure at the age of 3 months, was diagnosed with a systolic gradient in the aortic lumen of approximately 40 mm Hg in Doppler examination, caused by prominence of the occluder. Additionally, an increase in the left ventricle and pulmonary artery sizes were established. Due to unclear symptoms, cardiac catheterization was performed, concluding that the implant was correctly positioned and did not obstruct blood flow, yet revealing features of increased left-right flow and pulmonary hypertension, suggesting the presence of an intracranial arteriovenous fistula. The performed computed tomography excluded a cerebral fistula and revealed an abnormal connection between the branch of the left external carotid artery and the left internal jugular vein. The fistula was interventionally closed with coils and no residual leakage was observed after the procedure. The girl was discharged home in a good condition.

Key words: arteriovenous fistula, carotid artery, patent ductus arteriosus, interventional treatment

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Introduction

Vascular fistulas, both congenital and acquired, are very rare (about 1% of all heart defects), appearing in the process of angiogenesis, abnormal arterio-arterial, arterio-venous or veno-venous connections, and may be located in various parts of the body. The most common fistulas are located in the systemic, coronary or intrapulmonary

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circulation. In all of the cases, the capillary circulation is bypassed [1, 2]. A systemic arterio-venous (A-V) fistula located in the brain (e.g., Galen's vein malformation) can lead to intracranial bleeding caused by a rupture of the thin wall of the fistula [3]. Large fistulas can lead to heart failure due to significant left-right shunt. Symptoms such as systolic-diastolic murmur above the fistula, rapid pulse, high systolic-diastolic pressure difference, hyperkinetic apical beat, gallop rhythm and tachycardia can be found. Doppler echocardiography, computed tomography and/or magnetic resonance imaging are essential in the diagnosis [4, 5]. The modern and safest method of treating fistulas is the interventional implantation of various endovascular devices into the lumen of the fistula [6]. In the case of very large intrapulmonary fistulas, cardiosurgical treatment is also considered [1, 6, 7].

Case report

A 7-year-old girl underwent a hemodynamically significant patent ductus arteriosus (PDA) closure with an Amplatzer Duct Occluder at the age of 3 months. Recent control echocardiography revealed left ventricular enlargement, dilatation of the pulmonary trunk and indentation of the implant into the aortic lumen, causing narrowing and flow velocity acceleration. The patient was admitted for diagnosis. Physical examination only exposed a soft systolic murmur at the left edge of the sternum. The echocardiographic image indicated a mild form of coarctation of the aorta, while the implant slightly extended into the aortic lumen. The Doppler examination at the isthmus site, revealed an acceleration of flow with a maximum gradient of 40 mm Hg, with no features of "coarctation-type" flow. Due to the reported chest pain that intensified during exercise, the patient was qualified for cardiac catheterization. During the procedure, direct measurements and angiography showed no evidence of aortic coarctation requiring dilatation, the implant was in the correct position and no leakage at the vascular level was observed (Figure 1), however, the examination revealed pulmonary hypertension (systolic blood pressure 39 mm Hg; mean 30 mm Hg). The hemodynamic image indicated the presence of an intracranial fistula however a contrast-enhanced computed tomography imaging of the head exposed no presence of a fistula in this region. Therefore, the area of the examination was extended to the neck and only then the A-V fistula was visualized in the subcranial region, between the branch of the left external carotid artery and the left internal jugular vein. A loud systolic-diastolic murmur was heard in the neck near the lower region of the left ear (Figure 2). The girl was qualified for fistula closure (Figure 3) and an interventional, effective closure was performed using coils (Figure 4).

Discussion

The simultaneous occurrence of two different congenital extracranial vascular fistulas in the same child, i.e., PDA and the carotid A-V fistula, is extremely rare, while iatrogenic fistulas acquired after catheterization of the internal jugular vein are more often diagnosed in the neck [7–10]. A fistula can be diagnosed clinically based on a characteristic systolic-diastolic murmur at the site of the fistula. Until

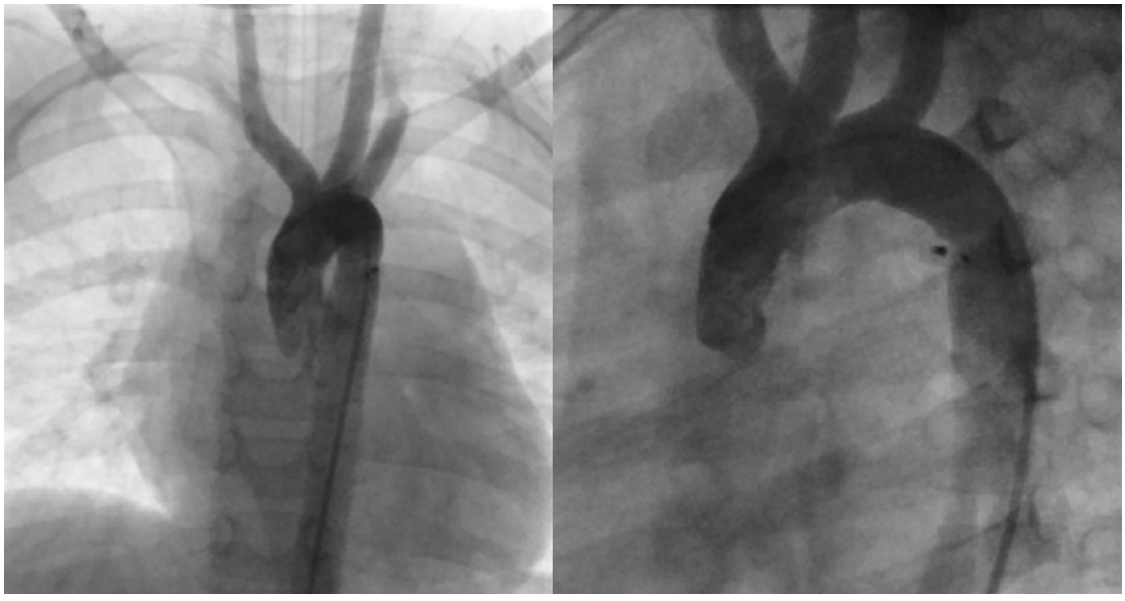

Figure 1. Aortography. Implant inside persistent ductus arteriosus in the correct position. Aortic coarctation was excluded

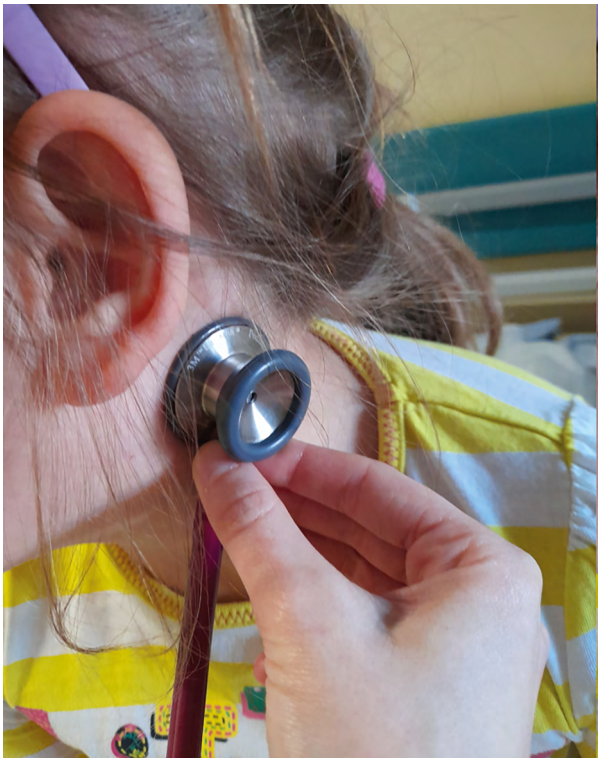

Figure 2. After placing the stethoscope in the area near the lower region of the left ear, a loud systolic-diastolic murmur was heard (photo with the consent of the patient and mother)

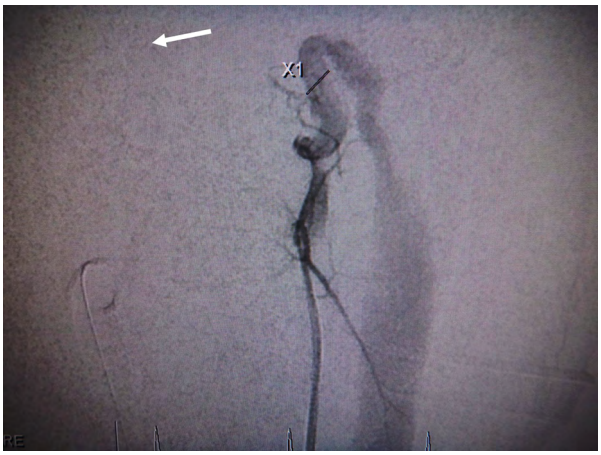

Figure 3. Arteriography. A catheter placed in the left external carotid artery. Visible arteriovenous fistula between the branch of the left external carotid artery and the left internal jugular vein (arrow)

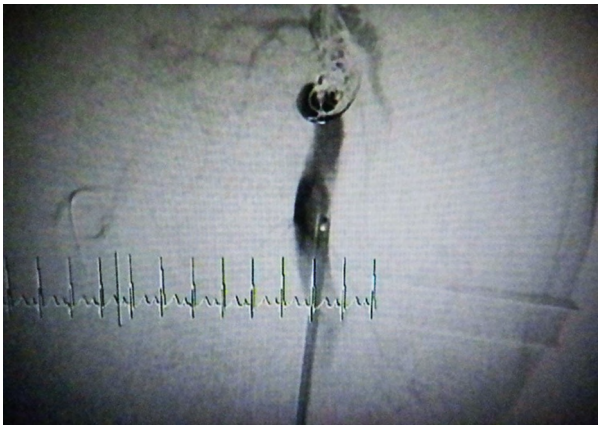

Figure 4. Arteriography. A catheter placed in the left external carotid artery. A fistula tightly closed with a coil occluder

the age of 7, the girl was in good condition and showed no disturbing symptoms until an abnormal image appeared in the aortic arch and PDA occluder implantation site examinations. It is possible to speculate, that if not for the cardiac catheterization performed to explain the causes of left ventricular enlargement, the vascular fistula in the neck would probably not have been diagnosed for a long time. On the other hand, the fistula could have been suspected after auscultatory examination, since one of the symptoms was a loud continuous systolic-diastolic murmur in the area of the lower pole of the left ear. An in-depth analysis of the course of treatment of the patient allowed to establish, that in some cases a more insightful auscultatory examination, such as auscultation of the cranio-jugular area, may expose symptoms of initially unexpected medical conditions.

Conclusions

Carotid arteriovenous fistulas in children are very rare and diagnosed at random. Hearing a continuous murmur of the fistula with a stethoscope may facilitate diagnosis. The treatment of choice is the interventional closure of the fistula.

Conflict of interest

None declared.

Funding

None.

Streszczenie

U 7-letniej dziewczynki, której w 3. miesiącu życia zamknięto implantem istotny hemodynamicznie przetrwały przewód tętniczy, w badaniu metodą Dopplera stwierdzono wpuklenie się okludera do światła aorty i jej przewężenie z gradientem skurczowym około 40 mm Hg. Ponadto powiększył się wymiar lewej komory oraz poszerzyła się tętnica płucna. Z powodu niejasnych objawów wykonano cewnikowanie serca — stwierdzono, że implant jest prawidłowo umieszczony i nie powoduje utrudnienia przepływu krwi. Wykluczono koarktację aorty, natomiast ujawniono cechy zwiększonego przepływu lewo-prawego z cechami nadciśnienia płucnego, sugerujące obecność wewnątrzczaszkowej przetoki tętniczo-żylnej. Po wykonaniu tomografii komputerowej wykluczono przetokę mózgową, uwidoczniono nieprawidłowe połączenie pomiędzy gałęzią lewej tętnicy szyjnej zewnętrznej a lewą żyłą szyjną wewnętrzną. Przetokę zamknięto interwencyjnie za pomocą cewek, po zabiegu nie zaobserwowano przecieku resztkowego. Pacjentkę wypisano ze szpitala w stanie dobrym.

Słowa kluczowe: przetoka tętniczo-żylna, tętnica szyjna, przetrwały przewód tętniczy, leczenie interwencyjne

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Latent complete atrioventricular block in a patient with Wolff–Parkinson–White syndrome and fast paroxysmal atrial fibrillation

Utajony całkowity blok przedsionkowo-komorowy u chorego z zespołem Wolffa–Parkinsona–White'a oraz napadowym migotaniem przedsionków z szybką czynnością komór

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Abstract

The Wolff–Parkinson–White (WPW) syndrome is very rarely complicated with a complete atrioventricular block (AVB). In this case, the ventricle is activated entirely through the accessory pathway and the QRS complex is wide and exhibits maximum pre-excitation features. This case report describes a history of a 68 years old male with symptomatic WPW syndrome, episodes of rapid atrial fibrillation and complete atrioventricular block.

Complete heart block in WPW syndrome should be suspected in the presence of a very wide, torn QRS, PJ interval > 0.27 s and paroxysmal atrial fibrillation without atrioventricular re-entrant tachycardia episodes.

Key words: Wolff–Parkinson–White syndrome, complete atrio-ventricular block, paroxysmal atrial fibrillation, case report

Folia Cardiologica 2023; 18, 2: 91–94

Introduction

In Wolff–Parkinson–White (WPW) syndrome the complete atrioventricular block (AVB) occurs very rarely [1–3]. In this case, the ventricle is activated entirely through the accessory pathway and the QRS complex is wide and exhibits maximum pre-excitation features.

Case report

We report the case of a 68 years old male with symptomatic for many years WPW syndrome. Recurrent episodes of atrial fibrillation with rapid ventricular rate, up to 230 bpm and subsequent pre-syncope, were observed in recent months. On admission, the patient had atrial fibrillation with intermittent pre-excitation (Figure 1).

In addition, the patient had hypertension complicated by moderate left ventricular hypertrophy (interventricular septum 16 mm, left ventricular ejection fraction 55%), generalized atherosclerosis and type II diabetes mellitus. The electrocardiogram during sinus rhythm had a pattern of pre-excitation compatible with a left free wall accessory pathway, a PR interval of 118 ms, QRS duration of 198 ms, an R pattern in lead V1, and an S pattern with slurring in lead aVL (Figure 2).

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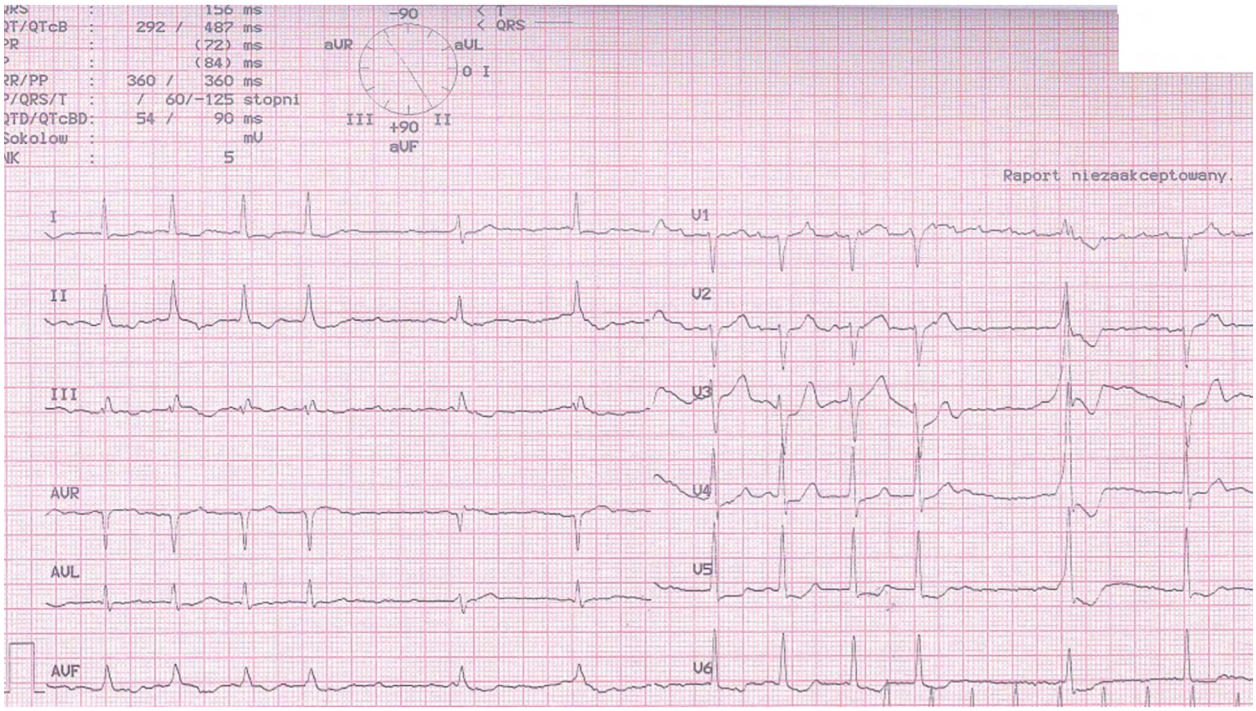

Figure 1. Atrial fibrillation with intermittent pre-excitation

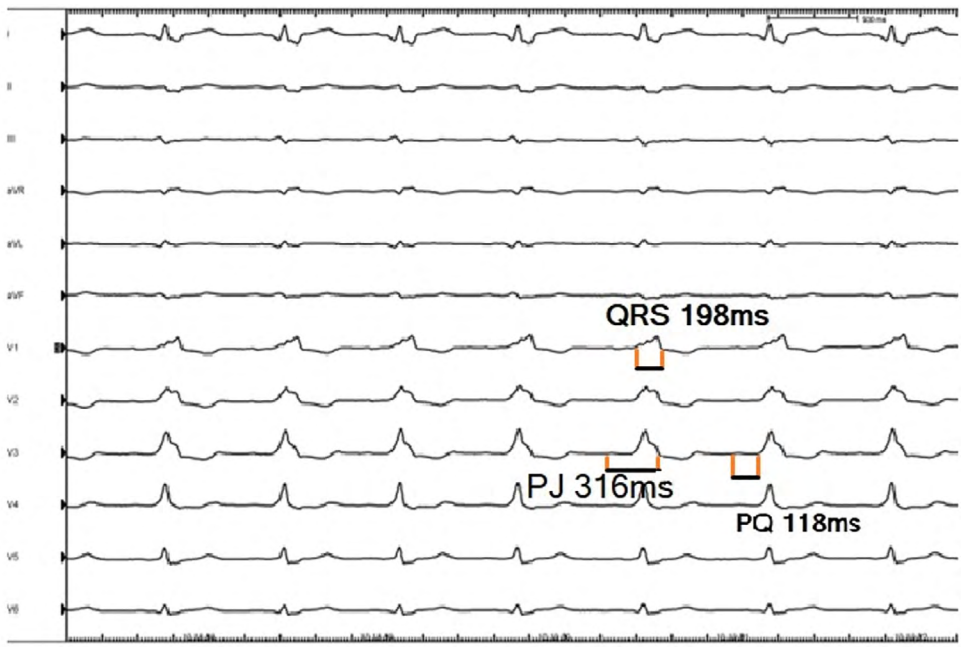

Figure 2. Electrocardiogram with preexcitation at the beginning of ablation

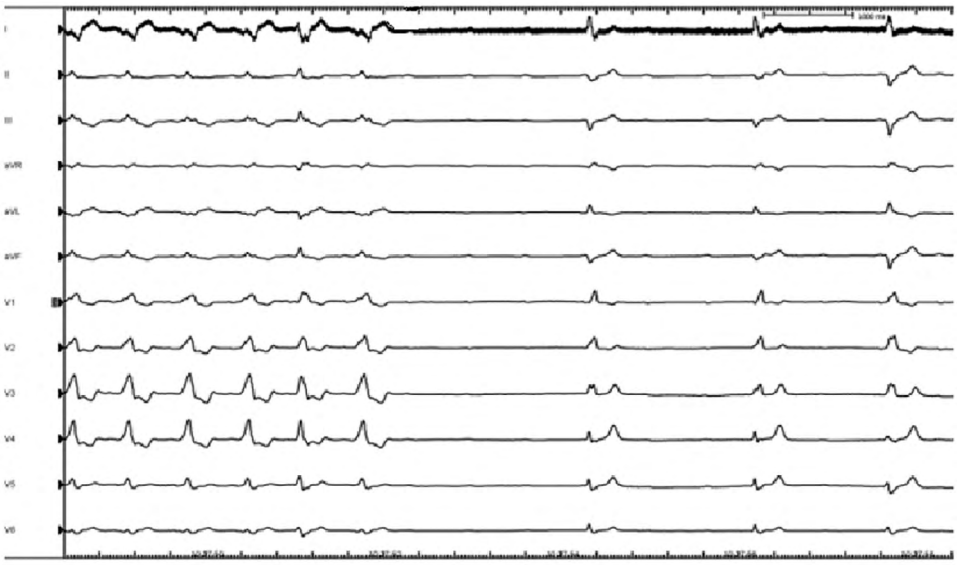

Figure 3. Electrocardiogram during ablation, the disappearance of preexcitation and complete atrioventricular block

The PJ interval was 316 ms, which was indicating the complication of the WPW syndrome with the AVB. Electrophysiology study revealed very short retrograde refraction ≤ 200 ms, and antegrade < 300 ms. Shorter coupling during the electrophysiology study was not examined due to the high risk for atrial fibrillation provocation. Atrioventricular re-entrant tachycardia (AVRT) was not induced. Subsequently, the ablation procedure was performed using a 7F, 4 mm tip ablation catheter, which was introduced from the right femoral artery and placed across the aortic valve at the mitral annulus on the free wall of the left ventricle. In the third second of radiofrequency energy application, pre-excitation disappeared, and a complete atrioventricular block occurred with a ventricular escape rhythm of 30 bpm (Figure 3). It is noteworthy that, the QRS during preexcitation was wider in comparison with QRS duration during ventricular escape rhythm (198 ms vs. 160 ms). Within the same day dual chamber pacemaker with a ventricular lead placed in the right ventricular outflow tract was implanted. The recurrences of the preexcitation were observed on the day following the ablation procedure and later on, what is more, episodes of fast atrial fibrillation were also repeatedly detected. After 2 months, the ablation procedure was repeated, resulting in permanent accessory pathway destruction. During the 30-month follow-up, no recurrences of preexcitation and rapid atrial fibrillation were noted. After 3 months of constant atrio-ventricular pacing, the patient was hospitalized due to exacerbation of heart failure.

Discussion

The diagnosis of complete heart block in WPW syndrome should be suspected in the presence of a very wide, torn QRS with paroxysmal atrial fibrillation but without AVRT. The PJ interval expands between the beginning of the P wave and the ending of the QRS complex and represents the time interval from the onset of atrial depolarization to the complete ventricular activation time. The PJ normal value is less than 0.27 s. The prolongation of the PJ interval is the electrocardiographic indicator of the first/third-degree AVB [4] or bundle branch block [5]. In the present case prolonged PJ interval, the disappearance of AVRT episodes and very wide, torn QRS during AF were indicating complete AV block within WPW syndrome. Changing the sequence of activation from the base of the left ventricle through the accessory pathway, to the right ventricle outflow tract (*via* pacing lead) may have caused a worsening of cardiac function in the study patient. Another factor responsible for heart failure *de novo* symptoms might be a substantial percentage of right ventricular pacing in the setting of complete atrioventricular block causing "pacemaker-induced cardiomyopathy".

Conflict of interest

None declared.

Streszczenie

Zespół Wolffa–Parkinsona–White'a (WPW) rzadko bywa powikłany blokiem przedsionkowo-komorowym III stopnia. W takiej sytuacji komory pobudzane są wyłącznie poprzez impuls przechodzący przez drogę dodatkową, zespół QRS jest szeroki i wykazuje cechy maksymalnej preekscytacji. W poniższym przypadku przedstawiono historię 68-letniego pacjenta z objawowym zespołem WPW, epizodami migotania przedsionków z szybką czynnością komór oraz całkowitym blokiem przedsionkowo-komorowym.

Całkowity blok przedsionkowo-komorowy u chorych z WPW powinien być podejrzewany w przypadku występowania bardzo szerokiego, zawężlonego zespołu QRS, odstępu PJ trwającego > 0,27 s, a także u pacjentów z napadowym migotaniem przedsionków bez epizodów nawrotnego częstoskurczu przedsionkowo-komorowego.

Słowa kluczowe: zespół Wolffa–Parkinsona–White'a, całkowity blok przedsionkowo-komorowy, napadowe migotanie przedsionków, opis przypadku

Folia Cardiologica 2023; 18, 2: 91–94

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Sprawozdanie i fotorelacja z VIII Konferencji czasopisma *Folia Cardiologica* 21–22.04.2023

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Collegium Medicum, Uniwersytet Jana Kochanowskiego w Kielcach
I Klinika Kardiologii i Elektroterapii, Świętokrzyskie Centrum Kardiologii, Wojewódzki Szpital Zespolony w Kielcach

W dniach 21–22 kwietnia, jak co roku od 8 lat, gościliśmy naszych czytelników i lekarzy zainteresowanych problematyką diagnostyki i terapii kardiologicznej w Warszawie na dwudniowej konferencji czasopisma. Pomimo ogromnej konkurencji, w tych dniach mieli bowiem swoją konferencję w stolicy kardiolodzy inwazyjni (*Warsaw Course on Cardiovascular Interventions — WCCI*), echokardiografiści (POL-ECHO), a w Krakowie miało miejsce duże wydarzenie Interna 2023 (XXII Krajowa Konferencja Szkoleniowa TIP), frekwencja dopisała, a co najważniejsze, słuchacze mieli dużo pytań, więc po każdej niemal sesji rozwijała się ożywiona dyskusja.

Wytyczne ESC przedstawione były tak, aby lekarz praktyk nie miał problemów z ich interpretacją, sesja: „Holistyczne podejście do pacjenta kardiologicznego, więcej pytań niż odpowiedzi?" ze znakomitymi wykładami profesorów Krzysztofa J. Filipiaka, Artura Mamcarza, Marcina Barylskiego, Marcina Wełnickiego i doktora Daniela Śliża uzmysłowiły nam, jak ważne jest uwzględnienie wielu schorzeń i okoliczności towarzyszących choremu „kardiologicznemu". Uznanie słuchaczy i duże zainteresowanie wzbudziły również znakomite warsztaty: hemodynamiczne prowadzone przez prof. Jacka Legutko i elektrokardiograficzne prezentowane przez prof. Krzysztofa Szydło. Lekarze

Fotografia 1. W biało-czerwonych barwach czasopisma ☺: Krzysztof J. Filipiak i Beata Wożakowska-Kapłon

Adres do korespondencji: prof. dr hab. n. med. Beata Wożakowska-Kapłon, Collegium Medicum, Uniwersytet Jana Kochanowskiego w Kielcach, aleja IX Wieków Kielc 19A, 25–317 Kielce, e-mail: bw.kaplon@poczta.onet.pl

Fotografia 2. Sesja przypadków w „Klinicznym kalejdoskopie kardiologicznym” i prowadzący sesję: Marcin Wełnicki, Katarzyna Starzyk, Marcin Barylski i Radosław Bartkowiak

Fotografia 3. Agnieszka Woronowicz-Chróściel przedstawia „Różne oblicza ubytku w przegrodzie międzyprzedsionkowej”

Pracowni Echokardiografii I Kliniki Kardiologii i Elektroterapii poprowadzili warsztaty echokardiograficzne, a zespół kieleckich elektrofizjologów w ponad godzinnej sesji przedstawił: „Nowoczesną diagnostykę i terapię zaburzeń rytmu” i próbował odpowiedzieć na pytanie: kiedy należy kierować chorego do elektrofizjologa. Mieliśmy również okazję dowiedzieć się, jakie są praktyczne wskazania do zastosowania ambulatoryjnego 24-godzinnego monitorowania ciśnienia tętniczego w diagnostyce chorych z nadciśnieniem tętniczym.

Fotografia 4. Wykładowcy po udanej sesji, stoją od lewej: Olga Jelonek, Michał Bączek, Radosław Bartkowiak, Paweł Kośmider, Agnieszka Major, Beata Wożakowska-Kapłon, Konrad Strzębała, Katarzyna Starzyk, Przemysław Dąbkowski, Paweł Wałek

Fotografia 5. Pierwszy dzień konferencji za nami, zespół I Kliniki Kardiologii i Elektroterapii Świętokrzyskiego Centrum Kardiologii, stoją od lewej: Paweł Kośmider, Michał Bączek, Beata Wożakowska-Kapłon, Olga Jelonek, Ireneusz Domański-Giec, Katarzyna Starzyk, Agnieszka Major, Konrad Strzębała i Paulina Putowska

Fotografia 6. Po sesji elektrofizjologicznej, grono wykładowców i prowadzących, od lewej stoją: Krzysztof Szydło, Michał Bączek, Beata Wożakowska-Kapłon, Paweł Kośmider, Przemysław Dąbkowski, Paweł Wałek, Maciej Młodnicki

Fotografia 7. Wykładowcy i prowadzący sesji Klubu 30 w komplecie, stoją od lewej: Mariusz Tomaniak, Krzysztof Ozierański, Katarzyna Holcman, Agata Bielecka-Dąbrowa, Agnieszka Kapłon-Cieślicka, Agata Tymińska, Justyna Sokolska, Mateusz Sokolski, Michał Tkaczyszyn

Bardzo duże zainteresowanie i oddźwięk wzbudziła sesja KKK, czyli „Kliniczny kalejdoskop kardiologiczny", zaprezentowana przez lekarzy I Kliniki Kardiologii i Elektroterapii Świętokrzyskiego Centrum Kardiologii w formie sześciu przypadków klinicznych, które nawet dla wytrawnych kardiologów stanowiły nie lada wyzwanie. *Last but not least,* znakomite sesje dwóch z dwunastu wspierających czasopismo sekcji PTK: sesja Sekcji Farmakoterapii Sercowo-Naczyniowej: „Indywidualizacja postępowania u pacjentów po ostrym zespole wieńcowym", sesja Sekcji Farmakoterapii Sercowo-Naczyniowej oraz sesja Klubu 30 PTK: „Rzadkie choroby serca czy raczej... zbyt rzadko diagnozowane?". Za rok obiecujemy nie mniej ambitne i wartościowe spotkanie dla czytelników i sympatyków pisma i już teraz zapraszamy do Warszawy w dniach 26–27 kwietnia 2024 roku na kolejną konferencję czasopisma *Folia Cardiologica*.

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