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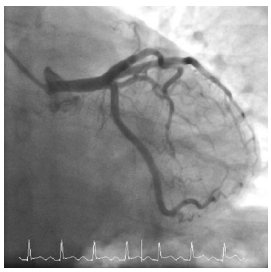

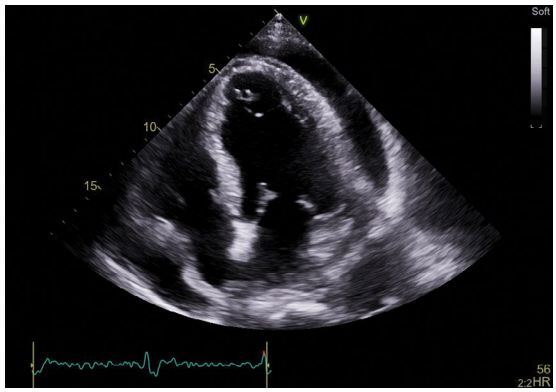

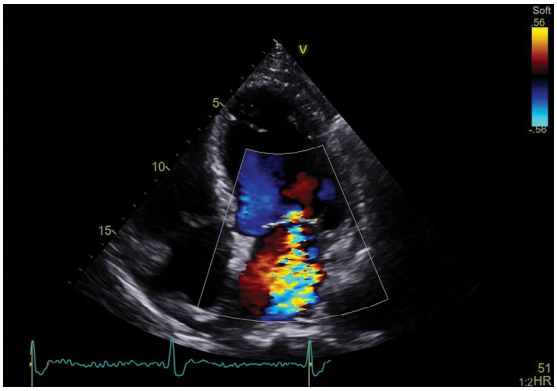

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Carotid arterial stiffness in type 2 diabetic patients

Sztywność tętnic szyjnych u pacjentów z cukrzycą typu 2

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Abstract

Introduction. Functional carotid arterial changes expressed by arterial stiffness indices represent subclinical organ damage in subjects with type 2 diabetes mellitus (T2DM). There are still controversies to what extent diabetes *per se* influences arterial stiffness and what is the contribution of other atherosclerotic risk factors in arterial stiffness pathophysiology. The aim of the study was to assess carotid arterial stiffness in patients with uncomplicated T2DM. We examined the relationship of classical cardiovascular risk factors and haemoglobin A1 and arterial stiffness indices in diabetes.

Material and methods. The study group consisted of 168 subjects: 84 subjects with T2DM (34 M, 50 F, mean age 55.8 ± 7.9 years) and 84 healthy patients (60 M, 24 F, mean age 54.3 ± 7.0 years). From carotid arteries ultrasound — high-resolution echo-tracking (eT) arterial stiffness parameters were evaluated: β, Ep, AC, AI, PWV-β.

Results. β, Ep, AI, PWV-β were higher in patients with T2DM in the comparison with control group. In the group of T2DM in stepwise multivariate analysis of arterial stiffness indices the following models were achieved with only significant variables: $\beta = 1.8 + 0.096 \times PP + 0.07 \times age$; $R^2 = 0.166$, $Ep = 16.7 + 1.852 \times PP$; $R^2 = 0.286$, $AC = 1.9 - 0.005 \times \times SBP - 0.007 \times HR + 0.14 \times$ smoking cigarettes; $R^2 = 0.165$, $AI = 18.0 - 0.80 \times BMI + 0.40 \times age$; $R^2 = 0.147$, PWV-β = $= -0.4 + 0.77 \times SBP - 0.72 \times MAP - 0.50 \times PP + 0.03 \times HR$; $R^2 = 0.235$.

Conclusions. T2D constitutes the strong independent determinant of arterial stiffness. In patients with T2DM the independent determinants of arterial stiffness parameters were age, SBP, MBP, PP, HR, BMI and smoking cigarettes. Not only glycemic control but also a multifactorial anti-risk strategy might play an important role in the prevention of the development of vascular stiffness and subclinical target organ damage in diabetes.

Key words: arterial stiffness, diabetes mellitus, glycated haemoglobin

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Introduction

Diabetes mellitus is associated with early and accelerated atherosclerosis and an increased risk of cardiovascular morbidity and mortality [1]. Pathophysiological mechanisms underlying these associations are not completely understood. Diabetes affects the cardiovascular system through two main mechanisms: atherosis which refers to lipid deposition in the vasculature to form intimal plaques, while sclerosis refers to vessel stiffening [2].

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Arterial changes include increased intima–media thickness (IMT), smooth muscle hypertrophy, collagen accrual and cross-linking, fibrosis and inflammation [3]. These changes are aggravated by advanced glycation end-products (AGE), irreversibly glycated proteins which stimulate systemic inflammation, oxidative stress, fibrosis, extracellular matrix remodeling, tissue injury modulation and lipid deposition in the arterial wall [4]. Elevated blood glucose concentrations favour AGE formation. Thus AGE might play a significant role in arterial stiffness. Increased arterial stiffness may be an important pathway linking diabetes to increased cardiovascuar risk [5]. It is known that increased arterial stiffness predicts the development of cardiovascular disease in type 2 diabetes mellitus (T2DM) [6]. Haemoglobin A_{1c} is an AGE that serves clinically as a marker of average glycemia in patients with diabetes.

There have been studies investigating the association between the classical risk factors and arterial stiffness in diabetes mellitus. However, the role of glycated haemoglobin (HbA_{1c}) control is still unclear [7, 8]. After analyzing the largest and longest-running study of T2DM (UKPDS, United Kingdom Prospective Diabetes Study), it is still unknown whether glucose control reduces the patient's risk of cardiovascular disease (CVD) [9]. In the present study we evaluated the impact of the T2DM on arterial stiffness and the impact of the classical risk factors and HbA_{1c} on carotid arterial stiffness in diabetic patients. We examined which of them [age, sex, smoking, blood pressure, body mass index (BMI), total cholesterol level — CH, low--density lipoprotein (LDL), high-density lipoprotein (HDL)] and HbA_{1c} could act as determinants of arterial stiffness in diabetes mellitus.

Material and methods

The study group consisted of 84 subjects with T2DM, 34 males and 50 females, mean age 55.8 ± 7.9 years. The control group consisted of 84 age-matched healthy subjects, 24 females and 60 males. Diabetics were treated by oral hypoglicaemic agents in 74%, by statins — in 91%. All patients underwent a comprehensive clinical examination, ECG, echocardiography, vascular assessments and evaluation of biological parameters. Only patients with a normal left ventricular (LV) systolic function (EF > 55%) and without cardiomyopathy, pericardial disease or valve dysfunctions were enrolled. Patients with evidence of ischaemic heart disease [a history of angina, a history of myocardial infarction, Q waves on electrocardiography (ECG) and regional wall motion abnormalities on echocardiography] were not eligible for the study. The protocol was approved by the local research ethics committee and each subject gave informed consent.

Demographic data

General data was obtained through a structured interview. Weight and height were measured according to the standard protocol, and each patient's body mass index (BMI) was calculated.

Laboratory determinations

Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), blood triglicerydes (TG), level of glycated hemoglobin (HbA_{1c}) were evaluated by standard techniques.

Blood pressure phenotypes

Blood pressure was measured while the patient was in the sitting position with the use of a standard sphygmomanometer on the left arm after a 5-minute rest. The first and fifth phases of Korotkoff sounds were used for systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. The mean blood pressure (MBP) was calculated as the mean pulse pressure added to one-third of the DBP. Pulse pressure (PP) was defined as the difference between SBP and DBP.

Echocardiography

A detailed two dimensional Doppler echocardiogram (Alpha 10 Hitachi-Aloka, Japan) was recorded for all the patients. M-mode measurements of end diastolic wall thickness [of interventricular septum (IVS) and posterior wall (PW)] and cavity diameter [LV end-diastolic diameter (EDD)] were used to calculate LV mass (LVM) by the formula introduced by Devereux et al. [10] and indexed to body surface area (BSA) to obtain a LV mass index (LVMI). Left ventricular ejection fraction was assessed in each subject using the Teichholz method.

Integrated assessment of arterial structure and function

Vascular ultrasound of the right common carotid artery was performed with an Alpha 10 Hitachi-Aloka machine equipped with an integrated and automated ultrasound, Doppler and echo-tracking system. Intima media thickness (IMT) was determined according to the established standards as the distance from the leading edge of the first echogenic line to the second echogenic line, with the media-adventitia interface [11]. Images of the thickest point within 10 mm from the common carotid artery (CCA) to the carotid bulb were saved and then measured. After clear visualization of the intima-media complex of both the anterior and posterior arterial wall in its longitudinal axis with a maximal internal diameter, an echo-tracking sample was positioned at the end of the intima, with a 1 kHz sampling rate for continuous detection of carotid diameter changes. In experimental

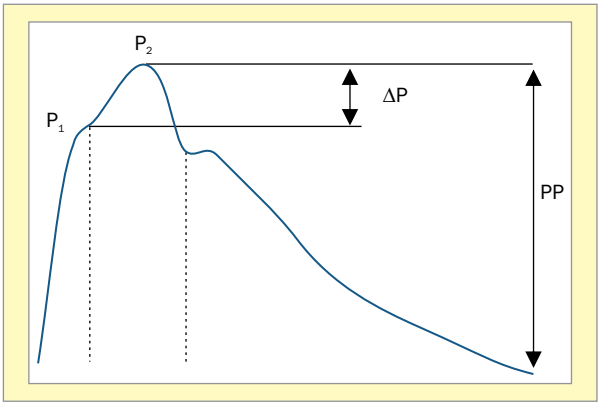

Figure 1. Augmentation index (AI) — method of calculation (source [1]); PP — pulse pressure, P1 — first systolic peak, P2 — second systolic peak, ΔP = P2 – P1

studies, diameter changes are very similar to intravascular pressure changes, which enables the automatic conversion of the carotid diameter waveform changes into arterial pressure waveforms by calibrating its peak and minimal values to systolic and diastolic brachial blood pressures [12]. The relationship of pressure-diameter is thought to be linear [12]. Three to five beats were averaged to obtain a representative waveform. The following arterial stiffness parameters were evaluated on-line [12, 13]:

β — beta stiffness index, as the ratio of the natural logarithm of systolic / diastolic blood pressure to the relative change in diameter:

$\beta = \ln (Ps/Pd/[(Ds - Dd)/Dd]$,

where: ln — the natural logarithm, Ps — systolic blood pressure, Pd — diastolic blood pressure, Ds — arterial systolic diameter, Dd — arterial diastolic diameter;

Ep — epsilon, Young modulus, pressure-strain elasticity modulus:

$Ep = (Ps - Pd)/[(Ds - Dd)/Dd]$;

AC — arterial compliance, calculated from the arterial cross area and blood pressures:

$AC = \pi(Ds \times Ds - Dd \times Dd)/[4 \times (Ps - Pd)]$;

PWV-β — one-point pulse wave velocity, calculated from the time delay between two adjacent distension waveforms from a water hammer equation with the use of β — the stiffness parameter:

$PWV\text{-}\beta = \sqrt{(\beta P/2\rho)}$,

where P — diastolic blood pressure, ρ — blood density (1050 kg/m^3).

From the parameters of wave reflection — augmentation index (AI) was calculated as:

AI = ΔP/PP, which is illustrated in Figure 1.

The blood pressure of the right arm was measured by an automated cuff sphygmomanometer with the patient being in the supine position for 10 minutes. The reproducibility of these measurements has been reported elsewhere [14]. An original example of arterial stiffness parameter examination by high resolution echo-tracking system derived from the right common carotid artery is presented in Figure 2.

Statistical analysis

Mean and standard deviations were calculated for the quantitative variables and percentages for qualitative variables. All variables were not normally distributed and therefore the differences between the groups were tested by the Mann-Whitney test for quantitative variables and by the chi-square test for the percentages of qualitative variables. Statistical significance was set at $p < 0.05$ (two-sided tests) and for multiple testing we used a statistical significance of $p < 0.01$. A multivariable logistic regression analysis was conducted considering the occurrence of arterial stiffness as a dependent variable. All the variables presenting a significant value < 0.25 at univariate analysis were included in the model. The stepwise forward method was used and odds ratios (OR) with 95% confidence interval (CI) were calculated. The model was evaluated with the Hosmer-Lemeshow test.

Results

Clinical characteristics of all patients is presented in Table 1.

All patients had normal IMT values (< 0,9 mm) and preserved LV systolic function (EF > 55%). Carotid arterial stiffness parameters like β, Ep, AI, PWV-β were higher in patients with T2DM in the comparison with control group (Table 2).

Significant linear correlations (table 3) were found between β and age ($r = 0.258$, $p = 0.001$), IMT ($r = 0.87$, $p = 0.009$), LVMI ($r = 0.177$, $p = 0.035$), SBP ($r = 0.280$, $p = 0,01$), MAP ($r = 0.227$, $p = 0.039$) and PP ($r = 0.268$, $p = 0.014$); between Ep and age ($r = 0.254$, $p = 0.002$), IMT ($r = 0.3$, $p = 0.006$), SBP ($r = 0.534$, $p < 0.001$), DBP ($r = 0.337$, $p = 0.001$), MAP (0.496, $p < 0.001$), PP ($r = 0.44$, $p < 0.001$) and HR ($r = 0.226$, $p = 0.015$); between AC and SBP ($r = -0.454$, $p < 0.001$), DBP ($r = -0.333$, $p = 0.002$), MAP ($r = -0.428$, $p < 0.001$), PP ($r = -0.366$, $p = 0.001$), heart rate (HR) ($r = -0.306$, $p = 0.005$) and smoking cigarettes ($r = 0.272$ $p = 0.013$); between AI and age ($r = 0.212$, $p = 0.011$), BMI ($r = -0.380$, $p = 0.001$) and HR ($r = -0.343$, $p = 0.003$); between PWV-β and age ($r = 0.222$, $p = 0.005$), IMT ($r = 0.293$, $p = 0.008$), SBP ($r = 0.552$, $p < 0.001$), DBP ($r = 0.467$, $p = 0.001$), MAP ($r = 0.539$, $p < 0.001$), PP ($r = 0.357$, $p = 0.001$) and HR ($r = 0.333$, $p = 0.003$). In the group with T2DM stepwise multivariate analysis (Snedecor's F distribution) of arterial stiffness indices the following models were achieved with only significant variables [age; SBP; PP; MBP; HR; smoking cigarettes, BMI):

$\beta = 1.8 + 0.096 \times PP + 0.07 \times age$; $R^2 = 0.166$,

$Ep = 16.7 + 1.852 \times PP$; $R^2 = 0.286$,

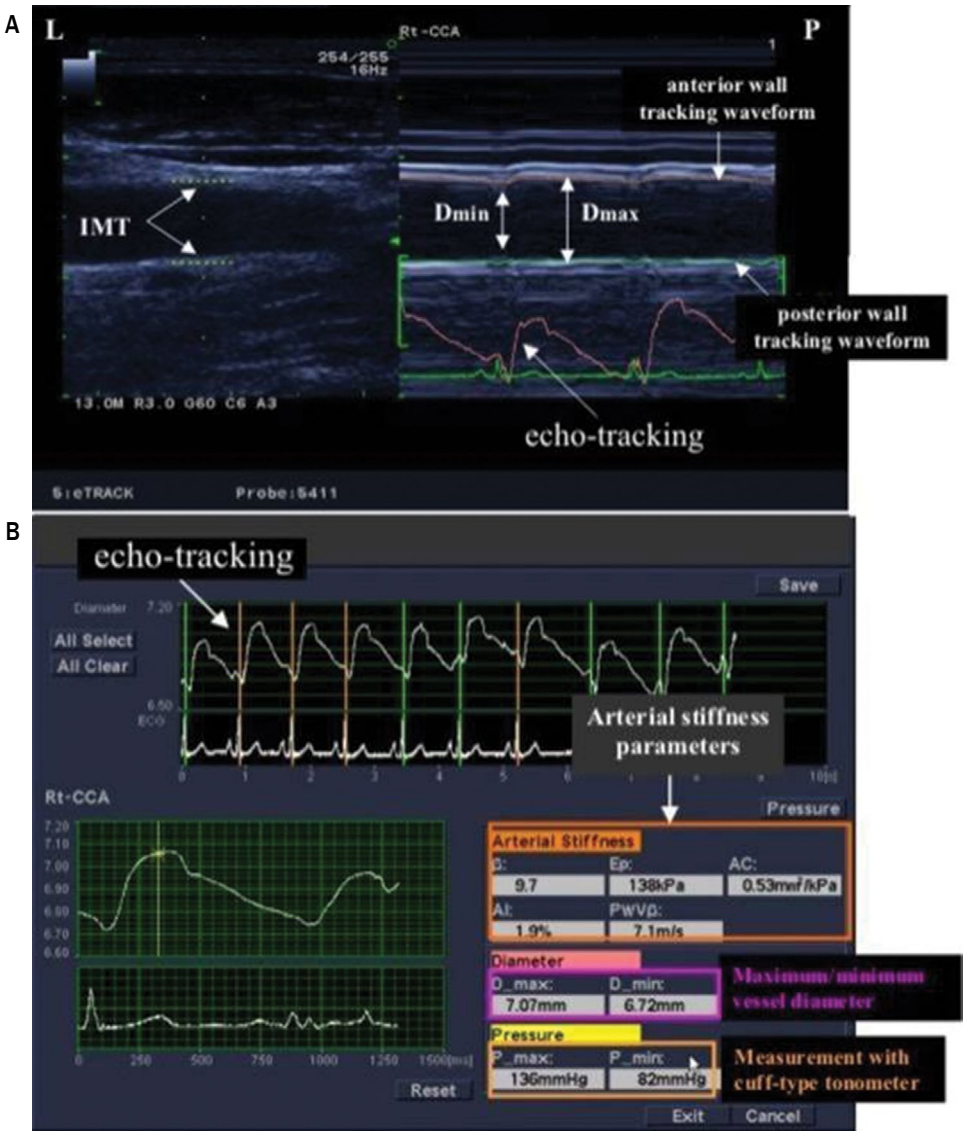

Figure 2A, B. Left (L): B-mode visualisation of right common carotid artery. Right (P): echo-tracking computed curve of dynamic diameter carotid artery. Lower: arterial stiffness parameters: β — beta; Ep — epsilon; AC — arterial compliance; PWV-β — one-point pulse wave velocity; AI — augmentation index

AC = 1.9 – 0.005 × SBP – 0.007 × HR + 0.14 × smoking cigarettes; $R^2 = 0.165$,

AI = 18.0 – 0.80 × BMI + 0.40 × age; $R^2 = 0.147$,

PWV-β = –0.4 + 0.77 × SBP – 0.72 × MAP – 0.50 × PP + 0.03 × HR; $R^2 = 0.235$.

Discussion

Cardiovascular diseases constitute the main cause of death in diabetes. Increased arterial stiffness is one of the key mechanisms of augmented cardiovascular risk in T2DM patients.

Hyperglycemia in T2DM stimulates the formation of advanced glycation end products (AGEs). The AGEs cross-links within the vascular wall further exacerbate vascular stiffness and large artery atherosclerosis [3]. The mechanisms by which high levels of glycaemia might lead to arteriopathy might become clear through recent studies. Accumulated AGE products have been known to be related to glycation and preferential oxidation of LDL and further uptake by macrophages to create foam cells [15] and finally glycaemia increases atherosclerosis [16]. The atherosclerotic process consists of two different aspects: atherosis (structural process) and sclerosis (functional process).

Table 1. Clinical characteristics of patients

Variable	Control group (C) N = 84	Diabetes (D) N = 84	K vs. D p
Age [years old]: $\bar{x}$ ± SD	54.3 ± 7.0	55.8 ± 7.9	0,086[a]
Sex:			< 0.001[b]
• females [%]	24 (28.6%)	50 (59.5%)	
• males [%]	60 (71.4%)	34 (40.5%)	
Body mass index (BMI) [kg/m^2]: $\bar{x}$ ± SD	25.9 ± 3.8	30.3 ± 4.7	< 0.001[b]
Heart rate (HR) [min^{-1}]: $\bar{x}$ ± SD	71 ± 11	72 ± 9	0.290[c]
Systolic blood pressure (SBP) [mm Hg]: $\bar{x}$ ± SD	128 ± 14	136 ± 18	0.003[c]
Diastolic blood pressure (DBP) [mm Hg]: $\bar{x}$ ± SD	77 ± 9	75 ± 9	0.120[c]
Pulse pressure (PP) [mm Hg]: $\bar{x}$ ± SD	52 ± 9	61 ± 14	< 0.001[c]
Total cholesterol [mg/dL]: $\bar{x}$ ± SD	230 ± 37	198 ± 39	< 0.001[d]
Low-density lipoproteins (LDL) [mg/dL]: $\bar{x}$ ± SD	146 ± 29	116 ± 34	< 0.001[d]
High-density lipoproteins (HDL) [mg/dL]: $\bar{x}$ ± SD	59 ± 18	49 ± 12	0.003[a]
Triglycerides (TG) [mg/dL]: $\bar{x}$ ± SD	127 ± 98	161 ± 82	0.001[a]
Glucose [mg/dL]: $\bar{x}$ ± SD	98 ± 15	147 ± 62	< 0.001[c]
Cigarette smoking: yes	40 (47.6%)	18 (21.4%)	< 0.001[b]
Creatinine [mg/dL]: $\bar{x}$ ± SD	0.83 ± 0.15	0.87 ± 0.19	0.544[c]
C-reactive protein (CRP) [mg/L]: $\bar{x}$ ± SD	1.61 ± 1.27	1.62 ± 1.59	0.985[c]
Ejection fraction (EF) [%]: $\bar{x}$ ± SD	69.5 ± 8.4	69.2 ± 7.0	0.811[c]
Left ventricular mass (LVM) [g]: $\bar{x}$ ± SD	154 ± 50	222 ± 60	< 0.001[c]
Intima media complex (IMT) [mm]: $\bar{x}$ ± SD	0.54 ± 0.15	0.67 ± 0.15	< 0.001[a]

[a]The Mann-Whitney U test; [b]Pearson's chi-squared test; [c]Student's *t*-test; [d]Fisher exact test; SD — standard deviation

Table 2. Carotid arterial stiffness indices in control and diabetes group

Variable	Control group (C) N = 84	Diabetes (D) N = 84	K vs. D p
β [–]: $\bar{x}$ ± SD	7.59 ± 2.53	10.04 ± 3.15	< 0.001[a]
Ep [kPa] : $\bar{x}$ ± SD	104.9 ± 41.1	137.2 ± 50.6	< 0.001[a]
AC [mm^2/kPa] : $\bar{x}$ ± SD	0.66 ± 0.23	0.70 ± 0.26	0.332[a]
AI [%]: $\bar{x}$ ± SD	20.01 ± 12.80	16.78 ± 13.34	0.035[a]
PWV-β [m/s]: $\bar{x}$ ± SD	6.1 ± 1.1	6.8 ± 1.2	< 0.001[b]

[a]The Mann-Whitney U test; [b]Student's *t*-test; β — beta stiffness index; SD — standard deviation; Ep — epsilon; AC — arterial compliance; AI — augmentation index; PWV-β — one-point pulse wave velocity

IMT reflects structural changes and arterial stiffness indices are functional markers [17]. There are many methods to assess systemic and regional arterial stiffness, such as applanation tonometry and mechanotransduction with the evaluation of 'gold standard' carotid-femoral pulse wave velocity [5, 6, 14, 18]. Echo-tracking systems, especially new high resolution ones, may provide easy-to-measure local arterial stiffness parameters in the detection of early functional arterial changes that precede vascular structural remodelling. European Society of Cardiology (ESC) experts recommend local arterial stiffness measurements for pathophysiologic studies [19]. Echo-tracking of carotid arteries has been shown as a convenient method to measure arterial stiffness parameters [13, 18, 20]. In the present study, similarly to Avgeropoulou et al. [21] the mean values of arterial stiffness parameters like β, Ep, AI, PWV-β were significantly higher in diabetic patients comparing to control subjects . In patients with diabetes mellitus the independent determinants of carotid arterial stiffness parameters were age (of β stiffness, AI), systolic blood pressure (of AC,

Table 3. Linear regression correlation coefficients of carotid arterial stiffness indices in type 2 diabetic group

Variable	β	Ep	AC	AI	PWV-β
Age [years]	**r = +0.258***	**r = +0.254***	r = –0.119	**r = +0.212***	**r = +0.222***
	p = 0.001	**p = 0.002**	NS	**p = 0.011**	p = 0.005
Duartion of type 2 diabetes [years]	r = +0.200	r = +0.233	r = –0.234	r = +0.019	r = +0.242
	NS	NS	NS	NS	NS
HbA_{1c} [mmol/mol]	r = +0.054	r = +0.075	r = +0.021	r = –0.131	r = +0.058
	NS	NS	NS	NS	NS
Total cholesterol [mmol/L]	r = –0.067	r = –0.066	r = +0.101	r = +0.060	r = –0.048
	NS	NS	NS	NS	NS
IMT [mm]	r = +0.287	r = +0.300	r = +0.063	r = –0.224	r = +0.293
	p = 0.009	p = 0.006	NS	NS	p = 0.008
LDL-cholesterol [mmol/L]	r = –0.138	r = –0.133	r = +0.166	r = +0.071	r = –0.135
	NS	NS	NS	NS	NS
HDL-cholesterol [mmol/L]	r = +0.163	r = +0.126	r = –0.159	r = +0.167	r = +0.131
	NS	NS	NS	NS	NS
Triglycerides [mmol/L]	r = –0.077	r = –0.010	r = +0.001	r = –0.198	r = +0.040
	NS	NS	NS	NS	NS
BMI [kg/m^2]	r = –0.042	r = +0.034	r = –0.013	**r = –0.380***	r = +0.025
	NS	NS	NS	**p = 0.001**	NS
LVMI [g/m^2]	**r = 0.177***	r = +0.009	r = –0.051	r = +0.066	r = +0.006
	p = 0.035	NS	NS	NS	NS
SBP [mm Hg]	r = 0.280*	r = +0.534*	**r = –0.454***	r = –0.070	**r = +0.522***
	p = 0.010	p < 0.001	**p < 0.001**	NS	**p < 0.001**
DBP [mm Hg]	r = + 0.142	r = +0.377*	r = –0.333*	r = –0.051	r = +0.467*
	NS	p = 0.001	p = 0.002	NS	p = 0.001
MAP [mm Hg]	r = 0.227*	r = + 0.496*	r = –0.428*	r = –0.062	r = +0.539*
	p = 0.039	p < 0.001	p < 0.001	NS	p < 0.001
PP [mm Hg]	**r = 0.268***	**r = +0.440***	**r = –0.366***	r = – 0.058	**r = +0.357***
	p = 0.014	**p < 0.001**	**p = 0.001**	NS	**p = 0.001**
HR [min^{-1}]	r = +0.207	r = +0.266*	r = –0.306*	r = –0.343*	r = + 0.333*
	NS	p = 0.015	p = 0.005	p = 0.003	p = 0.003
Cigarette smoking (1 — yes, 0 — no)	r = –0.129	r = –0.148	**r = +0.272***	r = +0.074	r = –0.144
	NS	NS	**p = 0.013**	NS	NS

*p < 0.05 considered significant; β — beta stiffness index; Ep — epsilon; AC — arterial compliance; AI — augmentation index; PWV-β — one-point pulse wave velocity; HbA_{1c} — glycated haemoglobin; IMT — intima-media complex; LDL; HDL; BMI — body mass index; LVMI — left ventricular mass index; SBP — systolic blood pressure; DBP — diastolic blood pressure; MAP — mean blood pressure; PP — pulse pressure; HR —heart rate

PWV-β), mean blood pressure (of PWV-β), pulse pressure (of β stiffness, Ep, PWV-β), heart rate (of AC, PWV-β), body mass index (AI) and smoking cigarettes (AC) but not HbA_{1c}.

There are still controversies to what extent diabetes *per se* influences arterial stiffness and what is the contribution of other atherosclerotic risk factors into arterial stiffness pathophysiology in diabetes. Some authors have indicated the main role of hypertension and age in arterial stiffness increase in diabetic patient [22]. Based on the meta-analysis of 77 studies with 26,970 patients included Cecelja et al. [23] documented diabetes as the independent determinant of arterial stiffness in about half of the studies while age and hypertension — in 90% of them. It has been suggested that co-existing hypertension may

mask the independent influence of diabetes on arterial stiffness. Our data are consistent with the studies in which age and blood pressure were the main determinants of arterial stiffness [24]. Tanakouchi et al. [25] showed that age and systolic blood pressure were significantly correlated with PWV in patients with non-insulin-dependent diabetes mellitus. Also Takahara [26] proved that age and systolic blood pressure had a significant impact on PWV in type 2 diabetes subjects. Age exposes the aortic wall to degenerative phenomena such as collagen accumulation, fragmentation of elastic fibers and calcification of the media responsible for the increase in aortic rigidity [27]. Long-standing arterial pulsation in the central artery has a direct effect on the structural matrix proteins, collagen and elastin in the arterial wall, disrupting muscular attachments and causing elastin fibers to fatigue and fracture [28]. This would explain why age and blood pressure are the major determinants of arterial stiffness [29].

Several studies also reported an independent association between HR and arterial stiffness [24]. The underlying mechanism is still unknown. Those studies indicated that the rate of elastin fractures depends on the number of stress cycles, that is, the number of heartbeats experienced which may explain the relationship between HR and arterial stiffness [25]. Consistently with our data, some studies reported that BMI was associated with arterial stiffness measured by baPWV [24]. In our study, we observed no significant association between HbA_{1c} levels and arterial stiffness indices in patients with T2DM. These data are consistent with Taniwaki et al. [30], who showed that HbA_{1c} was not an independent risk factor for arterial stiffness parameters (baPWV) in diabetic subjects. Kumeda et al. [31] also reported that in hemodialysis patients HbA_{1c} was not correlated with baPWV. Seong-Woo Choi et al. [7] showed that HbA_{1c} was not associated with baPWV in Korean T2DM patients. The exact reasons for these results are unknown. The first reason might be that arterial stiffness is strongly related to the ageing process [32] so that the ageing effect might have been so great that the effects of hyperglycemia may be covered. The second reason might be that almost all patients have been treated by statins which have a potential confounding effect on the association between hyperglycemia and arterial stiffness. Also, the usefulness of HbA_{1c} in T2DM has been questioned for more than 15 years [33]. Abnormal glycaemia may lead to the development of atherosclerosis in diabetes after many years of the disease. The results of the study by Larsen et al. [34], who had observed the metabolic control in patients with type 1 diabetes for 18 years, revealed the relationship between the mean values of HbA_{1c} and the progression of atherosclerotic changes in carotid and coronary arteries. As HbA_{1c} reflects the mean values of glycaemia for 3 months preceding the evaluation, it might not be an ideal parameter for the long follow-up of glycemic status. Standl i Ceriello [35] proved that sudden and acute glycemic changes as well as postprandial glycaemia are the most toxic factors for endothelium. Also HbA_{1c} does not express glycemic alterations and low serum glucose levels, which are known as factors modifying endothelium function. Postprandial glycaemia and glycemic spikes are thought to be a more predictive independent risk factor for cardiovascular diseases in T2DM than HbA_{1c} level [36, 37].

We did not observe significant correlations of carotid arterial stiffness indices and cholesterol levels. It is worth noting that almost all diabetic subjects in our study were treated with statins. The results of studies on the influence of statins on arterial stiffness are controversial [38, 39]. The lack of the association between carotid arterial stiffness and lipids in intriguing while taking into consideration documented relationship of cfPWV and atherosclerotic plaques [40, 41]. This may be explained by the lack of the impact of classical risk factors on the early stages of atherosclerotic process [42].

Limitations of the study

The study population was relatively small, only Caucasian and well-educated, which limits the generalisability of our findings . Blood pressure values used to calculate carotid eT arterial stiffness indices were measured over the brachial artery, which tends to overestimate carotid pressures due to central to peripheral blood pressure amplification. This is especially important in young subjects, but may have less relevance due to the mean age of our study patients, which was 57 ± 10.4 years. However, studies have shown a significant correlation between central and brachial blood pressure measurements [43] and many epidemiological studies use brachial artery blood pressure to estimate carotid artery stiffness.

Conclusions

In patients with T2DM, the independent determinants of carotid arterial stiffness parameters were age, systolic blood pressure, mean blood pressure, pulse pressure, heart rate, body mass index and smoking cigarettes but not HbA_{1c}. Not only glycemic control but also multifactorial anti-risk strategy (antihypertensive therapy, change of lifestyle) might play an important role in preventing the development of vascular stiffness and subclinical target organ damage in diabetes.

Statement of human and animal rights

All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 [5].

Statement of informed consent

Informed consent was obtained from all patients for being included in the study.

Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Wstęp. Zmiany czynnościowe tętnic szyjnych, które są wyrażone jako wskaźniki sztywności, są marekrem subklinicznego uszkodzenia narządowego u chorych na cukrzycę typu 2 (T2DM). Istnieją kontrowersje co do tego, w jakim stopniu cukrzyca *per se*, a w jakim stopniu inne czynniki ryzyka miażdżycy wpływają na sztywność naczyń. Celem pracy była ocena sztywności tętnic szyjnych u pacjentów z niepowikłaną T2DM. Autorzy zbadali zależność między klasycznymi czynnikami ryzyka sercowo-naczyniowego i wartością hemoglobiny A1 a sztywnością tętnic w cukrzycy.

Materiał i metody. Badaną grupę stanowiło 168 chorych, w tym 84 pacjentów z T2DM (34 M, 50 K, średnia wieku 55,8 ± 7,9 roku) oraz 84 zdrowe osoby stanowiące grupę kontrolną (60 M, 24 K, średnia wieku 54,3 ± 7,0 roku). Metodą *echo-tracking* (Ep) oceniono sztywność tętnic szyjnych za pomocą następujących parametrów: β, Ep, AC, AI, PWV-β.

Wyniki. U chorych z T2DM wartości wskaźników sztywności tętnic szyjnych (β, Ep, AC, AI, PWV-β) były istotnie wyższe niż u osób z grupy kontrolnej. W grupie z T2DM w analizie regresji wielokrotnej uzyskano natępuące istotne modele parametrów sztywności: $\beta = 1{,}8 + 0{,}096 \times PP + 0{,}07 \times age$; $R^2 = 0{,}166$, $Ep = 16{,}7 + 1{,}852 \times PP$; $R^2 = 0{,}286$, $AC = 1{,}9 - 0{,}005 \times SBP - 0{,}007 \times HR + 0{,}14 \times$ liczba papierosów, $R^2 = 0{,}165$, $AI = 18{,}0 - 0{,}80 \times BMI + 0{,}40 \times$ a wiek; $R^2 = 0{,}147$, PWV-$\beta = -0{,}4 + 0{,}77 \times SBP - 0{,}72 \times MAP - 0{,}50 \times PP + 0{,}03 \times HR$; $R^2 = 0{,}235$

Wnioski. Cukrzyca typu 2 jest silnym niezależnym czynnikiem sztywności tętnic. U pacjentów z T2DM niezależnymi determinanatmi parametrów sztywności tętnic były wiek, SBP, MBP, PP, HR, BMI oraz palenie papierosów. Nie tylko kontrola glikemii, ale także wieloczynnikowa strategia prewencyjna może odgrywać istotną rolę w zapobieganiu rozwojowi sztywności naczyń oraz subklinicznym uszkodzeniom narządowym w cukrzycy.

Słowa kluczowe: sztwyność naczyń, cukrzyca. hemoglobina glikowana

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The electrocardiographic characteristics of the P-wave with incomplete Bachmann's bundle block in patients with atrial fibrillation

Charakterystyka elektokardiograficzna załamka P u pacjentów z niepełnym blokiem pęczka Bachmanna i migotaniem przedsionków

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Abstract

Introduction. The structural and functional changes in atrial myocardium create a substrate for atrial fibrillation (AF). The pathophysiology includes enlargement of the cavities and replacing the cardiomyocytes with connective tissue. That slows down the conduction velocity and creates focal conduction block zones and slow conduction areas promoting the re-entrant circuits. The described changes influence the duration and amplitude of the P-wave in the electrocardiography.

Materials and methods. The study group consisted of 54 patients diagnosed with AF. There were 19 women and 35 men, aged 65.8 ± 10.0 years. 22 patients had paroxysmal AF, in sinus rhythm during the examination and 32 had persistent AF, in whom the direct current cardioversion was performed to achieve sinus rhythm.

Results. In patients with persistent atrial fibrillation the P-wave duration after the restoration, the sinus rhythm was significantly longer in comparison to patients with paroxysmal atrial fibrillation (159.2 ± 14.3 vs. 171.2 ± 16.6 ms, p = 0.006). The patients with persistent AF showed higher positive amplitude in lead V1 as well as higher negative amplitude than patients with paroxysmal AF; positive amplitude: 0.053 ± 0.023 vs. 0.084 ± 0.040 mV, p = 0.002, negative amplitude: 0.045 ± 0.018 vs. 0.075 ± 0.037 mV, p = 0.001.

Conclusions. In patients with incomplete Bachmann's bundle block and atrial fibrillation, the duration of the P-wave is prolonged, more in the persistent arrhythmia group. The P-wave duration and morphology correlate with the interatrial conduction disturbances. The P-wave morphology changes indicate the causal relationship of AF rather with interatrial conduction delay than with left atrial hypertrophy.

Key words: P-wave duration, P-wave morphology, incomplete Bachmann's bundle block, atrial fibrillation

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Introduction

The structural and functional changes in atrial musculature create a substrate for atrial fibrillation [1]. Those pathologies result from numerous diseases such as ischaemic heart disease, hypertension or heart defects, in particular mitral valve disease [2]. Over time and the ongoing course of pathophysiological changes, the atria enlarge and cardiomyocytes are replaced by connective tissue [3, 4]. The slowing down the activation wave of conduction velocity creates focal conduction block zones and slow conduction areas promoting the formation of re-entrant circuits. Those changes promote arrhythmia maintenance. The influence for described disturbances contributes to the functional isolation of left atrium and pulmonary veins junctions leading ultimately to the creation of trigger zones for atrial fibrillation [5].

The structural changes described above contribute to the formation of arrhythmogenic foci perpetuating premature beats and atrial rhythms. Rapid arrhythmias induce local changes in the refractory period of the atrial muscle, which increases the possibility of re-entrant rhythms and leads to the persistence of atrial fibrillation [6]. The impact of arrhythmic paroxysms on further enlargement of the left atrium by increasing left ventricle filling pressure leads to consecutive structural changes.

All of this affects the duration and amplitude of the P-wave in the electrocardiography (ECG). The slow muscle conduction and the left atrium enlargement causes the prolongation of the P-wave duration [7]. In hypertension patients, the pressure overload can induce atrial musculature hypertrophy with an increase in P-wave amplitude (increase in negative deflection in lead V1) [8]. However, a reduction in the number of cardiomyocytes and an increase in the amount of connective tissue can decrease the generated potential and thus decrease the P-wave amplitude. The profound changes in intra- and interatrial conduction are related to the Bachmann's bundle block. Changes in activation time significantly affect the P-wave duration and morphology [9]. The detailed assessment of those parameters has not been extensively studied in the literature so far.

The authors aimed at the assessment of the P-wave characteristics in patients with atrial fibrillation and incomplete Bachmann's bundle block.

Material and methods

The study group consisted of 54 patients with atrial fibrillation. There were 19 women and 35 men, aged 65.8 ± 10.0 years. The presence of essential co-morbidities and coexisting anti-arrhythmic treatment was also assessed. The patients' population was divided into 22 patients with paroxysmal atrial fibrillation and 32 patients with a persistent form of arrhythmia, in whom the electrical cardioversion was performed to re-establish sinus rhythm. All the patients were treated with beta-blockers and some of them received propafenone or amiodarone. In patients with persistent AF, the precise estimation of the arrhythmia was not possible, but to narrow the time span included only the patients with episodes lasting from 1 to 6 months. The clinical characteristics of the study group were presented in Table 1.

The P-wave parameters: duration and amplitude in particular leads were measured using LabSystem™ Pro EP Recording System from Boston Scientific. The P-wave duration was measured in all leads at a paper speed of 200 mm/s and enhancement 64–128. The P-wave morphology was assessed in lead II to recognize the typical pattern of the incomplete Bachmann's bundle block. The amplitudes

Table 1. The clinical characteristics of the study groups

Parameter	Patients with paroxysmal AF	Patients with persistent AF	p
Number	22/54	32/54	0.382
Sex	8 F, 14 M	9 F, 23 M	0.390
Age	67.8 ± 7.2	64.8 ± 10.2	0.321
HT	20/22	30/32	0.874
IHD	6/22	4/32	0.197
HF	2/22	3/32	0.924
DM	4/22	7/32	0.668
CKD	4/22	2/32	0.192
Metoprolol	7/22	14/32	0.290
Amiodarone	3/22	3/32	0.458
Propafenone	12/22	25/32	0.457

AF — atrial fibrillation; F — female; M — male; HT — hypertension, IHD — ischaemic heart disease, HF — heart failure; DM — diabetes mellitus; CKD — chronic kidney disease

Table 2. The P-wave parameters in patients with paroxysmal and persistent atrial fibrillation (AF)

Parameter	Patients with paroxysmal AF	Patients with persistent AF	p
Number of patients	22	32	–
P-wave duration [ms]	**159.2 ± 14.3**	**171.2 ± 16.6**	**0.006**
Peak separation [ms]	58.3 ± 13.9	62.1 ± 18.7	0.56
Amplitude II lead [ms]	0.137 ± 0.164	0.109 ± 0.040	0.613
Amplitude V1 lead positive [ms]	**0.053 ± 0.023***	**0.084 ± 0.040#**	**0.002**
Amplitude V1 lead negative [ms]	**0.045 ± 0.018***	**0.075 ± 0.037#**	**0.001**

Differences between positive and negative phases in lead V1: in patients with paroxysmal AF: *p = 0.16, in patients with persistent AF: #p = 0.062

of the P-wave were measured in lead II. The incomplete Bachmann's bundle block was recognized if the separation of the right and left atrial P-wave peaks was significant (more than 40 ms). The positive and negative phases in lead V1 were also measured. To avoid the measurement inaccuracies, all the measurements were repeated 5 times and presented as the mean value.

In the group of patients with persistent AF, the direct current cardioversion was done (in general anaesthesia under propofol 1 mg/kg and fentanyl 50 μg given intravenously) to restore the sinus rhythm. A single 300 J shock was successful in all patients.

The study protocol was accepted by the local Bioethical Committee at Wroclaw Medical University.

Statistical analysis

The continuous variables are presented as a mean and standard deviation. The comparisons were performed with the parametrical Students *t*-test or non-parametrical U Mann-Whitney test for independent variables. The dependent variables comparisons were performed using the Students *t*-test for dependent variables or Wilcoxon paired test. Each categorical variables were presented as numbers and/or percentages. The comparisons were performed with the chi-square test. The correlations between the studied parameters were performed using Pearson's correlations coefficient or Spearman's rank correlation according to the statistical properties of the data.

P values less than 0.05 were considered significant.

Results

In patients with persistent atrial fibrillation, the P-wave duration after the restoration of sinus rhythm was significantly longer in comparison to patients with paroxysmal atrial fibrillation. The parameters and morphology of the P-waves were shown in Table 2.

The persistent arrhythmia group had more pronounced interatrial conduction abnormalities even if it didn't reach the statistical significance. The P-wave amplitude in lead II was not significantly different between the groups as was the separation of the peaks in this lead. The patients with persistent AF showed higher positive amplitude in lead V1 as well as higher negative amplitude than patients with paroxysmal AF. The comparison of positive and negative V1 deflection in both groups according to atrial fibrillation form indicated the borderline statistical difference in persistent AF (p = 0.061).

The P-wave duration correlated with the separation of the right and left atrial peaks in lead II slightly differently in patients with paroxysmal (A) and persistent (B) atrial fibrillation, which was depicted in Figure 1.

There was a strong positive correlation between the positive and the negative amplitudes in lead V1 in patients with persistent atrial fibrillation. This correlation was shown in Figure 2. This coherence did not reach statistical significance in patients with paroxysmal AF.

After the combination of the parameters for the whole group studied the strong positive interrelation between the positive and negative deflections in lead VI was maintained what was presented in Figure 3.

To illustrate the complexity of P-wave morphology in a patient with incomplete Bachmann's bundle block the typical example of the ECG tracings was presented in Figure 4.

Discussion

The main result of this study in patients with incomplete Bachmann's bundle block is the effect of sustained arrhythmia episodes on the prolongation of the P-wave duration. This indicates strongly the direct influence of atrial fibrillation-induced intra-atrial and inter-atrial conduction disorders and, most probably, atrial enlargement. The phenomenon should be associated with an increased left ventricle filling pressure and could be dependent on the ventricular rate [10]. Data on the effect of AF on muscle conduction are more scarce and non-equivocal, but if the prolongation of the P-wave is taken into account in combination with the peak separation of the lead II P-wave, one can draw the obvious conclusion that the prolongation is related to the interatrial conduction delay. As the subgroups

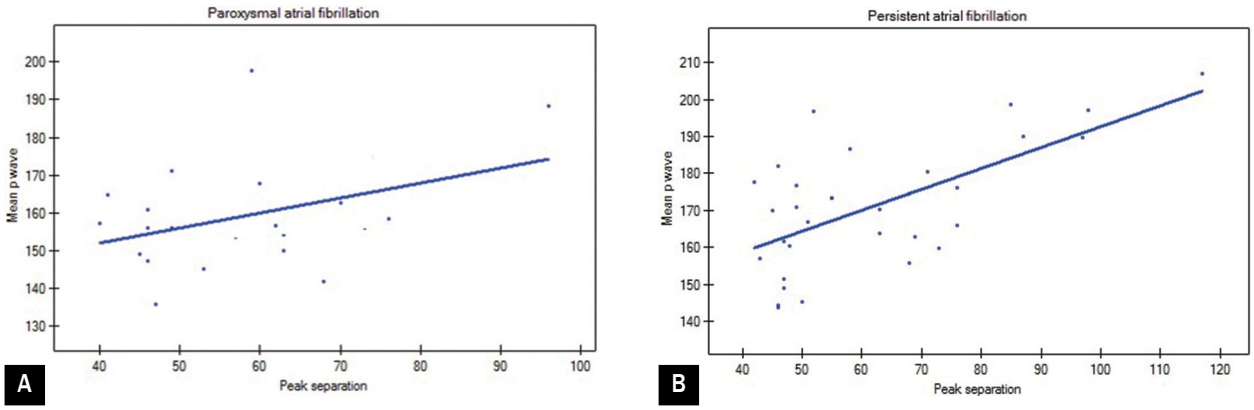

Figure 1. The mean P-wave duration correlation with peak separation in lead II in patients with paroxysmal (**A**) (r = 0.39, p = 0.07) and persistent (**B**) (r = 0.59, p = 0.00207) atrial fibrillation

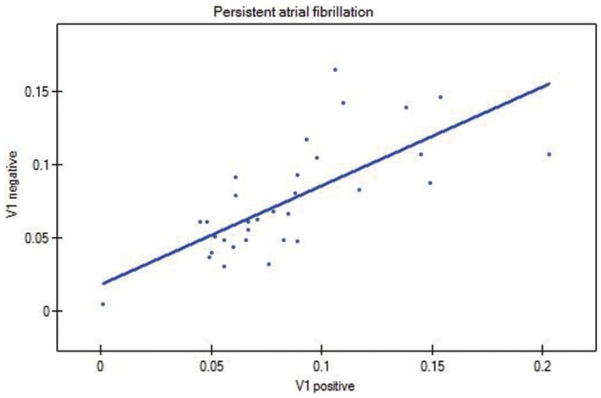

Figure 2. A strong correlation (r = 0.766, p < 0.000001) between positive and negative amplitude in lead V1 in patients with persistent atrial fibrillation

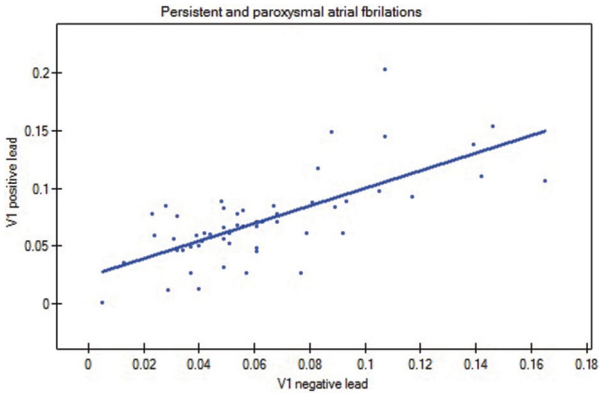

Figure 3. A strong positive correlation (r = 0.663, p < 0.000001) between positive and negative amplitude in lead VI in the whole group of patients

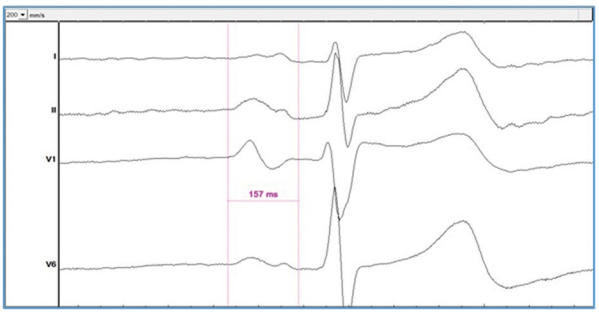

Figure 4. The complexity of P-wave morphology in a patient with incomplete Bachmann's bundle block (paper speed 200 mm/s, enhancement 64×)

of patients with both paroxysmal and persistent atrial fibrillation are similar according to age, comorbidities, gender distribution and anti-arrhythmic treatment, all of which could affect the duration of the P-wave, it indicates that this study's results are related only to the absence or presence to the prolonged episode of arrhythmia [1]. This observation seems to be directly in line with the clinical observation, contained in the term "AF begets AF" created by Wijffels et al. [11]. The direct current cardioversion did not affect by itself on the P-wave duration in one study, indicating the stable values of the P-wave parameter immediately after the procedure and on the next day [12].

Another important result from this study is the statistically significant relationship between the duration of the P-wave and the distance of the right and left atrium peaks in lead II. In both studied patients group, the positive correlation was maintained, in the persistent AF patients the correlations coefficient was higher. Although the difference in the separation of the peaks in both groups was statistically insignificant, the separation itself influenced the P-wave duration. It seems justified to conclude, that the prolongation of the P-wave is mainly due to conduction delay in the Bachmann's bundle and not to the atrial muscle dimensions [13]. While there are no similar papers

yet, and the only one pointed to similar conclusions [14], this is consistent with the literature on Bachman's bundle conduction disorders [8, 9, 13].

The precise methodology for the measurement of the P-wave duration used in this study is the same one the authors adopted a few years ago to show the lack of P-wave dispersion — a parameter which is related to the inaccuracy of the measurement [15]. This approach is qualitatively different from the other researchers' methodology [16]. It is nevertheless in line with the suggestions of Bayes de Luna contained in the paper "Diagnosis of interatrial block", where it was explicitly pointed out to precisely measure the P-wave from the earliest beginning to the latest end [17].

Another novel issue resulting from this research is the increase in the amplitudes of the positive and negative phase of the V1 lead P-wave in patients with persistent AF. The direct comparison of the deflections in the studied groups indicate the more advanced disease in the patients with a persistent form of arrhythmia and most probably reflects the more enlarged left atria during ongoing arrhythmia. But this reflects also the known electrocardiographic pattern. In physiology, the overlap of the negative phase (left atrium) with the positive phase (right atrium) influences both amplitudes. The significant extension of the left atrium muscle mass increases the negative deflection in V1, with a negative predominance in this lead [8]. In both groups, in lead V1, the positive deflection is greater than the negative one, even if only barely visable. It gives rise to a hypothesis that among the patients with incomplete Bachmann's bundle block the interatrial conduction delay could be the cause of atrial fibrillation and not hypertrophy of the muscle [3, 18]. The analysis of our entire population indicates this electrocardiographic phenomenon to be true cause it's difficult to assume the atrial fibrillation results in or is a result of right atrial hypertrophy/enlargement.

Study limitations

An essential limitation of this study is a relatively small population of patients. The already mentioned very precise measurement method used in the study including the electrophysiological recording system could affect the camparibility with other researcher's results. As the duration of persistent AF was not possible to be precisely established, the hypothesis, that the prolongation of the P-wave duration is caused mainly by the arrhythmia itself, should be assessed with caution. This constitutes the main limitation of this research, even if the available data contradict such correlation [12].

Conclusions

In patients with incomplete Bachmann's bundle block and atrial fibrillation, the duration of the P-wave is prolonged, significantly more in the persistent arrhythmia group.

The P-wave duration and morphology correlate with the interatrial conduction disturbances.

The P-wave morphology changes indicate the causal relationship of AF rather with interatrial conduction delay than with left atrial hypertrophy.

Funding

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Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Wstęp. Zmiany strukturalne i czynnościowe mięśnia przedsionków tworzą podłoże dla migotania przedsionków (AF). Patofizjologia obejmuje powiększanie jam przedsionków i zastępowanie kardiomiocytów tkanką łączną. Spowalnia to prędkość przewodzenia, tworzy strefy lokalnego bloku oraz obszary zwolnionego przewodzenia sprzyjające rytmom nawrotnym. Opisane zmiany wpływają na czas trwania i amplitudę załamka P w elektrokardiografii.

Materiał i metody. Badana grupa składała się z 54 pacjentów ze zdiagnozowanym AF. Stanowiło ją 19 kobiet i 35 mężczyzn w wieku 65,8 ± 10,0 lat. U 22 pacjentów stwierdzono napadowe AF, a u 32 pacjentów występowała przetrwała forma arytmii i wykonano u nich kardiowersję prądem stałym w celu uzyskania rytmu zatokowego.

Wyniki. U pacjentów z przetrwałym AF czas trwania załamka P po przywróceniu rytmu zatokowego był znacznie dłuższy niż u pacjentów z napadowym AF (159,2 ± 14,3 *vs.* 171,2 ± 16,6 ms; p = 0,006). Pacjenci z przetrwałym AF wykazywali wyższą amplitudę dodatnią w odprowadzeniu V1, a także bardziej ujemną amplitudę w tym odprowadzeniu niż pacjenci z napadowym AF; amplituda dodatnia: 0,053 ± 0,023 *vs.* 0,084 ± 0,040 mV; p = 0,002, amplituda ujemna: 0,045 ± 0,018 *vs.* 0,075 ± 0,037 mV; p = 0,001.

Wnioski. U pacjentów z niepełnym blokiem wiązki Bachmanna i AF czas trwania załamka P jest wydłużony, bardziej w grupie przetrwałej arytmii. Czas trwania załamka P i morfologia korelują z zaburzeniami przewodzenia międzyprzedsionkowego. Zmiany morfologii załamka P wskazują na związek przyczynowy AF raczej z opóźnieniem przewodzenia międzyprzedsionkowego niż z przerostem lewego przedsionka.

Słowa kluczowe: czas trwania załamka P, morfologia załamka P, niepełny blok pęczka Bachmanna, migotanie przedsionków

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Pathophysiology of atrial fibrillation. Systemic review

Patofizjologia migotania przedsionków. Przegląd systemowy

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Abstract

Atrial fibrillation (AF) is an arrhythmia in which chaotic electrical signals are generated in the atria. AF can be classified as first episode AF, paroxysmal AF, persistent AF, long-standing persistent AF and permanent AF. Hence, AF is one of the biggest problems of contemporary health care (due to severe complications like thromboembolic disease and huge expenses associated with the treatment).

The pathophysiology of the AF includes a triggered activity in the myocardium and also left atrial enlargement (LAE), and remodelling of the atria that may result in an interatrial block (IAB). IAB is prolonged conduction between the atria and is diagnosed in electrocardiography (ECG) when P-wave duration ≥ 110 ms. Other ECG changes coexisting with IAB, LAE and also remodelling of the atria are attributed to P-wave dispersion ≥ 40 ms, and a P-wave terminal force in V1 ≤ –0.04 mm/s.

Remodelling of the atria leads to structural, cellular and hormonal changes. At the cellular scale — mitochondrial size and count are enlarged. A neurohormonal imbalance is also related to arrhythmia. An increased level of atrial natriuretic peptide, B-type natriuretic peptide, angiotensin II, transforming growth factor-β_1 are observed in the case of cellular and ion channels changes.

Atrial fibrillation is a significant problem posed to modern health care. In light of these remarks, an effective way of AF prevention needs to be found. In any case, further research in this field is needed.

Key words: atrial fibrillation, interatrial block, electrocardiography

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Introduction

Atrial fibrillation (AF) is an abnormal and irregular heart rhythm in which electrical signals are generated chaotically throughout the atria. AF can be classified as first episode AF, paroxysmal AF (that terminates < 7 days), persistent AF (AF sustained > 7 days and early persistent AF — lasting less than 3 months), long-standing persistent AF (> 12 months in duration), permanent AF (accepted by the patient and physician) [1]. One of the most frequently identified predictors of AF is a visibly prolonged time for the signal to appear. It is called the interatrial block (IAB).

Due to the fact that AF is the most common type of heart arrhythmia, it is one of the biggest problems of the present health care system. AF is reported to cost the United States alone approximately $6 billion each year. Reports indicate that people who suffer from AF spend an additional $8,705 more per year than people without AF [2, 3]. It is estimated, that by 2050 AF will have affected 6–12 million people in the USA and 17.9 million people in

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Europe by 2060 [4]. Currently, AF affects circa 2.9% of the world's adult population. Among people within the range of 60–69 years old — AF occurs in 4.2% of the population. AF affects also 9.7% of the population from the age group of 70–79, whereas if focusing on people from within the group of 80–89 years old, AF is noted to affect 13,4% of them [5]. Hence, LAE and remodelling may result in AF. In electrocardiography (ECG) it can be demonstrated as an interatrial block (IAB).

Interatrial block — definition and consequences

The interatrial block is prolonged conduction between the atria. It is diagnosed in ECG when P-wave duration ≥ 110 ms IAB is caused by the enlargement of the atria and atrial remodelling. Impulse delay or blockage is often in the Bachmann bundle (BB) [6]. The first research in this field was done by Bachmann et al. in 1916 — interatrial conduction delay was pointed out and its importance as leading to atrial fibrillation was mentioned [7]. IAB is an important sign because it has serious implications. First of all, IAB is associated with AF, but the role of the interatrial block as a predictor of other arrhythmias has not been evaluated [8]. Secondly, remodelling of the left atrium could lead to cerebral stroke, even if AF is not manifested [9]. That is why, in 2005, Lorbar et al. showed an 80% prevalence of IAB in patients with embolic stroke [10].

Drawing an analogy to other conduction delays, IAB may be graded as first, second and third degrees. IAB can also be divided into partial and advanced type of block [11] The normal transit time for electrical impulses generated in the sinus node has to be conducted throughout the atria in less than 110 ms. The first degree IAB (or partial IAB) is a conduction delay across the Bachmann's bundle with P-wave duration > 110 ms. The impulse goes with a delay through the normal conduction pathway. IAB with notched P-wave in leads I, II, III and aVF is considered to represent partial or first degree. In ECG P-wave morphology in lead V1 in partial IAB often comes with a negative mode (or a biphasic mode), however, left atrial enlargement cannot be diagnosed. The second degree of IAB is diagnosed when the first component of the P-wave maintains its spatial orientation while the final component of IAB shows noticeable modifications in its morphology and duration (this state is also seen with atrial aberrancy), but conditions of the third degree of IAB are not met. The third degree of IAB is a complete block in the normal interatrial conduction pathway. The impulse goes through an alternate pathway with caudocranial activation of the left atrium. ECG biphasic (±) (±) P-wave in the inferior leads is therefore produced. A negative P-loop is more evident than in partial IAB, what is often intensified by coexistent left atrial enlargement. That is why the terminal negative loop of P-wave in lead V1 is wider as compared with a partial IAB. Due to the underlying fibrosis, the positive mode of P-waves in leads II, III and aVF may sometimes not be well seen [11–13].

Remodelling of the atria

Remodelling can be divided into three separate types: electrical, structural, and autonomic. Structural remodelling is the cause of electrical dissociation between local conductive heterogeneity and muscle fibre bundles. This state results in the re-entry of waves and arrhythmias. Due to the fact, that structural remodelling is usually irreversible, it is important to start treatment very early [14]. Various aetiologies of remodelling should be considered to understand this process (changes in intracellular structure in cardiac muscle, left atrium enlargement as a cause of fibrosis, *etc.*) because changes in the cardiac tissue structure depend on the mechanism of atrial remodelling. In the case of atrial fibrillation myocyte, myolysis is additionally observed [15]. In the case of left atrial enlargement associated with heart failure — fibrosis and myocyte cellular hypertrophy are observed [16]. Fibrosis of the atrium plays a great role in the pathogenesis of AF. Daccarett et al. [17] confirm this point of view in research performed with magnetic resonance imaging (MRI). They declare, that the extent of fibrosis is an independent risk factor of cerebral stroke.

Cellular changes in atria remodelling

At the cellular scale — mitochondrial size and count are enlarged. These changes appear after about one week of continued atrial fibrillation [18]. What is more, in the case of healthy myocytes, visible domination of myosin heavy chain (α isoform) can be observed, while in the cases of heart failure the amount of myosin heavy chain β isoform increase. This is the reason for a decrease of left atrial systole connected with higher left atrium (LA) volume. These factors are directly responsible for LA remodelling. After this stage is completed, degenerative changes in myocyte cells can be found *e.g.* cellular oedema, the nucleus pyknosis and in consequence — lower count of myocyte cells [19]. Furthermore, to create a re-entry wave — a refractory period should be shortened, conduction should be delayed and the conduction itself should be elongated. A great role in these changes plays ion-channel remodelling [20]. The atrial refractory period depends on the duration of electric potential because sodium (Na^+) channels are inactivated in the depolarization phase and need repolarization to –60 mV. The duration of electric potential is determined by a balance between inward current (especially of Ca^{2+}) and outward flow of K^+. The electric remodelling of ion channels can lead to shortening of both the resting potential in a cardiomyocyte and the refractory period. Rapid left atrial activation (during AF), reduces the inward current

Ca^{2+} of type L, and escalates the outward flow of K^{+}. If the new impulse strikes into the unidirectional block, re-entry wave can be developed, which also leads to atrial fibrillation [21]. These changes are the main symptoms leading to AF propagation [22, 23].

Left atrial enlargement — mechanisms and implications

Zacà et. al. confirms that the increased size of the LA predicts atrial fibrillation. What is more, LA progressive enlargement is examined as a predictor of arrhythmic recurrences [23]. On the other hand atrial enlargement can be a result of atrial fibrillation [24]. In ECG left atrial enlargement is diagnosed by P-wave duration > 120 ms long, or alternatively, the time between its two peaks ≥ 40 ms and by positive-negative (or negative) P-wave in V1 [25]. An increased anterior-posterior (AP) diameter of left atrium signed in M-mode echocardiography is related to increase AF incidence by 40% [26] An AP diameter over 5 cm, when sinus rhythm is maintained, characterizes a 4-fold increase risk of AF [27]. Additionally, LA volume is a clear indicator of AF in patients with cardiomyopathy, and the same set of parameters apply to elderly patients [28]. Unfortunately, the relationship between planimetric diameters and the volume of LA is not linear [29]. The volume of AF is found to be a better tracer of atrial fibrillation determined by left atrium volume index (LAVI) — LA volume measured by biplane area length method and indexed to body surface area (BSA) [30]. Left atrial enlargement contributes to the growth and preservation of arrhythmia, which is called electrophysiological remodelling. On the other hand, arrhythmia leads to structural changes in the cardiac muscle [31]. That is why maintained sinus rhythm is responsible for a decrease of left atrial diameter and contractility enhancement [32]. Xiong et al. suggest there are no differences of LA diameters before cardioversion and directly after the procedure [33]. Nevertheless, measurements performed one month after the appearance of sinus rhythm showed a decrease of AP diameter of LA. What is more, the reduction of an LA size is related to Interventricular septal motion [34]. Tops et al. [34] proved that electrical cardioversion or cardiac ablation leads to a decrease of an LA size, however, the recurrence of AF undermines these changes. Another research demonstrates LA volume decrease after effective cardiac ablation from 59 to 50 mL in cases of patients, who maintain sinus rhythm for 3 months. In the case of patients who are after the ineffectual procedure, the recorded LA volume increases from 63 mL to 68 mL. Restored sinus rhythm results with a reduction of left atrial AP diameter from 4.5 ± 0.3 cm to 4.2 ± 0.2 cm ($p < 0.001$), and a reduction of the LA area from 29.4 ± 5.1 cm^2 immediately after electrical cardioversion to 26,7 ± 4,6 cm^2 after 6-month follow-up [35]. Similar results are presented by Reant et al. [35] who proved that in the case of patients with persistent AF, after the restitution of sinus rhythm in 11-month follow-up occurred decrease LA area from 28,7 ± 2,8 cm^2 to 21,7 ± 2,1 cm^2 (24% reduction). Unfortunately, patients with an AF recurrence during the 11-month follow-up achieved a 7% reduction in the LA area. What is more, in a situation of a recurrent, paroxysmal AF after effective cardiac ablation a 19% reduction of the LA area is possible (from 23.5 ± 4.8 cm^2 to 19.0 ± 3.8 cm^2), while, in cases, after electrical substrate elimination is not total — the reduction of the LA area is successful in the case of only 5% of the total number of patients. These findings prove that even short duration of AF stimulates LA remodelling. On the other hand, maintenance of sinus rhythm is helpful in averting pathological remodelling, even if AF is recurrent. Furthermore, there is a relationship between electrical and structural remodelling [36]. Surgical procedures (*e.g.* Maze procedure) also conduce to a decrease of LA area and the associated advantageous biochemical changes [37].

Neurohormonal imbalance in the interatrial block

Neurohormonal changes play a significant role in atrial remodelling. An increased level of atrial natriuretic peptide (ANP), B-type natriuretic peptide (BNP), angiotensin II, transforming growth factor-β_1 (TGF-β_1) are all related to both cellular and ion channels changes [38]. In the case of patients with AF, the level of C-reactive protein (CRP) is elevated, as well as tumour necrosis factor, interleukins and cytokines. The significance of inflammation in the pathogenesis of cardiac muscle remodelling is verified by relations between LA volume and the level of CRP in peripheral blood [39]. Due to the fact, that there is a relationship between elevated CRP and metalloproteinases activation (by oxygen free radicals), inflammation is found to influence LA fibrosis. That leads to a left atrium enlargement and a deterioration of the LA mechanic function. Moreover, CRP stimulates the expression of the receptors of end glycation products, which, in turn, leads to hardening of arterial walls and the cardiac wall. That is the reason for left atrial distention to appear in the case of patients who suffer from diabetes mellitus [39].

Electrocardiographic changes

ECG is a crucial diagnostic instrument in cases of atrial fibrillation, interatrial block and enlargement of the atria. P-wave terminal force shows early delay in left atrial conduction (it is a product of the amplitude and the duration of the terminal phase of P-wave in lead V1). It has been observed when left atrium is dilated [32]. The P-wave initial portion in the same lead is significantly greater

in patients with left atrial overload. These patients are more likely to develop AF in the future. P-wave duration ≥ 125 ms, P-wave dispersion ≥ 40 ms, and a P-wave terminal force in V1 ≤ –0.04 mm/s are good clinical risk factors of the atrial fibrillation recurrence, post pulmonary vein isolation (PVI) in the case of patients with paroxysmal atrial fibrillation [40]. De Bacquer et al. [15] checked baseline P-wave items in patients aged 55 to 74 years and apparently healthy at baseline. 40 of them developed AF within the 10-year period and they were compared retrospectively with those of 120 matched controls. Broad maximum P-waves (> 120 ms) at baseline were observed in 70% of the patients with AF and it referred to 41% of controls (p = 0.002). P-wave duration proved to be a significant risk marker (independent of other risk factors like blood pressure, body mass index etc.). Ishida et al. [16] checked the incidence of AF in patients with marked LA overload (diagnosed by P-wave inversion in lead V1) which was 15-fold higher than that in control patients (19% of patients with LAO developed AF versus 81 % who did not. In the control group under discussion — AF was developed by 1.3% of patients). On the other hand, no significant difference was seen between the AF and non-AF groups with regard to the area, duration, and amplitude of the P-wave terminal portion in lead V1. Alternatively, these ECG changes were significantly greater in the AF group than in the non-AF group. In view of this, the area of the P-wave initial portion was independently associated with the development of AF (hazard ratio 4.02, 95% confidence interval 1.25–17.8; p = 0.018) [16].

Conclusions

Atrial fibrillation is a significant problem posed to modern health care. Huge expenses incurred by AF occurrences together with thromboembolic implications of AF create a pressing need for finding a way to prevent the condition. Well-known risk factors *e.g.* LAE, age, previous history of AF do not reduce the range of problems related to the prevention of AF. The pathophysiology of the AF includes LAE, remodelling of the atria (that may result in interatrial block) and also triggers activity in cardiac muscle. P-wave duration ≥ 125 ms, P-wave dispersion ≥ 40 ms and a P-wave terminal force in V1 ≤ –0.04 mm/s appear to be good clinical factors indicating interatrial conduction delay and left atrial enlargement. In any case, further research in this field is needed.

Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Migotanie przedsionków (AF) jest arytmią, która charakteryzuje się chaotycznymi impulsami elektrycznymi generowanymi w przedsionkach serca. Można wyróżnić pierwszy epizod AF, napadowe AF, przetrwałe AF, przetrwałe długotrwające AF, utrwalone AF. Obecnie jest to jeden z największych problemów ochrony zdrowia (np. ze względu na ciężkie powikłania zakrzepowo-zatorowe i ogromne nakłady finansowe związane z ich leczeniem).

Mechanizm powstania AF polega na wyzwalaniu aktywności miokardium na podłożu powiększenia i przebudowy przedsionków. Mogą one skutkować blokiem przewodzenia (IAB) lub wydłużonym czasem przewodzenia między przedsionkami (LAE). Diagnostyka LAE jest możliwa na podstawie elektrokardiogramu (EKG), gdy czas trwania załamka P przekracza 110 ms. Inne zmiany w EKG współwystępujące z blokiem śródprzedsionkowym, powiększeniem lewego przedsionka, a także związane z remodelingiem przedsionków są wyrażone poprzez dyspersję P ≥ 40 ms i iloczyn amplitudy i czasu trwania fazy ujemnej P w odprowadzeniu V1 ≤ –0,04 mm/s.

Remodeling przedsionków prowadzi do strukturalnych, komórkowych i hormonalnych zmian. W skali komórkowej zwiększają się liczba i rozmiar mitochondriów. Zaburzenie równowagi neurohormonalnej również jest związane z arytmią. Podwyższone stężenia przedsionkowego peptydu natriuretycznego, peptydu natriuretycznego typu B, angiotensyny II, transformującego czynnika wzrostu β_1 towarzyszą zmianom komórkowym i modyfikacjom kanałów jonowych.

Migotanie przedsionków to bardzo ważny problem, będący wyzwaniem współczesnej ochrony zdrowia. W związku z tym należy poszukiwać metod efektywnej prewencji tej arytmii.

Słowa kluczowe: migotanie przedsionków, blok śródprzedsionkowy, elektrokardiografia

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ST-segment elevation myocardial infarction in 27-year-old pregnant women

Zawał serca z uniesieniem odcinka ST u 27-letniej kobiety w ciąży

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Abstract

A pregnant woman diagnosed with a myocardial infarction is an extremely rare case in clinical practice. Obesity and nicotinism are well-known risk factors for cardiovascular events that are significant especially in the population of younger women. In this paper, the authors present the case of a 27-year-old woman who suffered an ST-segment elevation myocardial infarction in early pregnancy. Percutaneous coronary intervention constitutes the basis for treatment in such cases. The problem is the optimal adjustment of pharmacotherapy, safety for the developing foetus should also be included in its planning.

Key words: myocardial infarction, STEMI, pregnancy, smoking, obesity

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Case report

A 27-year-old obese smoking female patient, who was diagnosed with inferior wall myocardial infarction with ST elevation, was transferred from the hospital emergency department to a haemodynamic laboratory. A few hours before the admission, during a meeting with friends, the patient suddenly felt chest pain, for the first time in her life. The pain gradually decreased over a few dozen minutes. At the time of admission, the patient described it as chest discomfort. The physical examination revealed a regular heart rate of 117/min, blood pressure 140/77 mm Hg, systolic murmur at the apex, class II obesity — body mass index (BMI) 35 kg/m^2. No family history. The electrocardiography (ECG) revealed sinus tachycardia with a frequency of 119/min, ST-elevation typical of Pardee's sign, by 3 mm in inferior wall leads, and ST depression up to 2.5 mm in the I, aVL, V2–V4 leads (Figure 1).

The patient received 300 mg of acetylsalicylic acid, 180 mg of ticagrelor and 100 mg of enoxaparin. She had coronary angiography performed immediately, which revealed a peripheral obstruction of the posterolateral branch of the right coronary artery (probably of embolic aetiology) (Figures 2, 3). The patient was qualified for urgent angioplasty of the infarct-related artery. Glycoprotein IIb/IIIa inhibitor (eptifibatide) was included in the treatment; an attempt was made to carry out vessel revascularisation and multiple balloon inflations were performed. As a result, the flow was slightly improved [TIMI (Thrombolysis In Myocardial Infarction) 1].

Laboratory tests showed dynamic changes in the troponin I concentration, determined using the high-sensitivity method (maximum concentration 17,164.4 ng/L), which is typical of infarction, and hyperlipidemia [total cholesterol 195 mg/dL, LDL (low-density lipoprotein)-cholesterol 141 mg/dL]. The normal activity of protein C and S and antithrombin III was observed. The patient also had genetic tests carried out, which did not show any factor V Leiden or prothrombin G20210A mutation or any mutations in both alleles of *F2*, *F5* and *MTHFR* genes.

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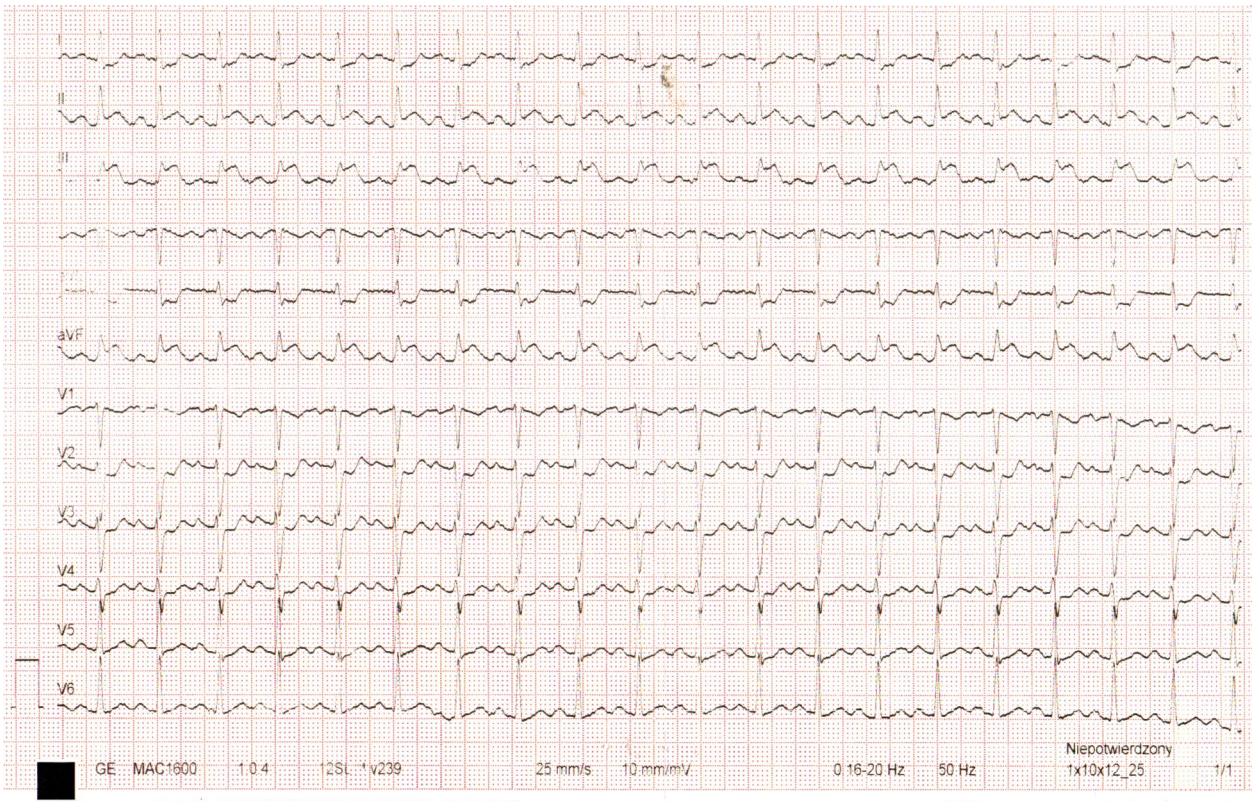

Figure 1. Electrocardiogram (ECG) performed on admission to the clinic

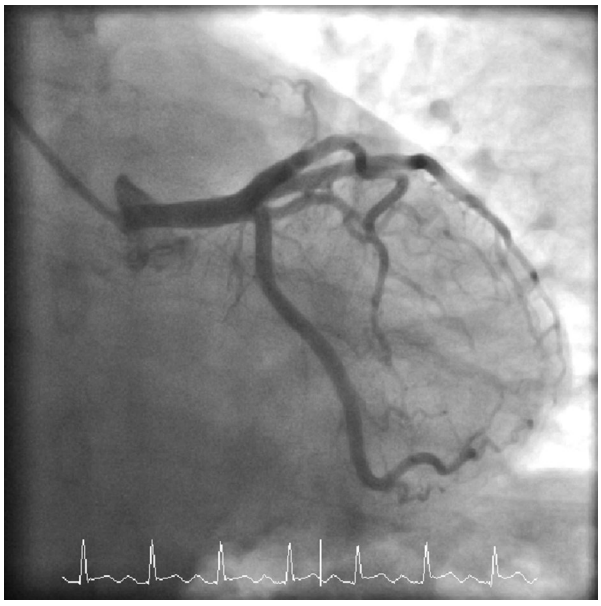

Figure 2. Angiogram of normal left coronary artery

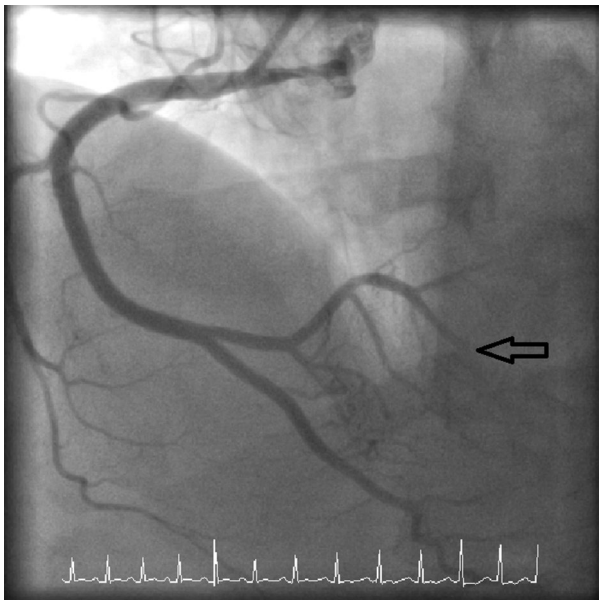

Figure 3. Angiogram of right coronary artery. The arrow indicates the place of obstruction of the posterolateral branch.

Echocardiography revealed hypokinesis of the basal segment of the inferior wall, left ventricular ejection fraction was 55%. Transesophageal echocardiography did not show any obvious signs of the atrial septal defect or embolic material in the left atrial appendage.

No signs of thrombosis in superficial and deep veins in the lower extremities were observed in the Doppler ultrasound. The 48-hour ECG monitoring using the Holter method recorded sinus rhythm with an average frequency of 79/ /min, without episodes of supraventricular arrhythmia. The

abdominal ultrasound showed no significant deviations. Due to the fact that the patient had missed her period and her suggestion that she could be pregnant, a gynecological ultrasound was performed and confirmed the possibility of early pregnancy. This was also confirmed by a typical increase in the human chorionic gonadotropin (hCG) concentration. Therefore, the therapy was immediately modified and the patient stopped taking statin and ticagrelor.

No complications were observed during the hospitalisation. After the discharge, the patient was treated with enoxaparin in a therapeutic dose, acetylsalicylic acid at a dose of 75 mg and bisoprol at a dose of 2.5 mg. She gave birth to a healthy female newborn weighing 3,150 g who was delivered by caesarean section performed in the 40th week of pregnancy.

Discussion

Ischemic heart disease is rare in pregnancy (prevalence is between 2.8–6.2 per 100,000 deliveries). However, it is becoming more and more common nowadays because women decide to become mothers at a later age when they already have comorbidities. [1] Coronary heart disease accounts for > 20% of all maternal deaths due to cardiac causes. As far as the aetiology of the disease is concerned, nonatherosclerotic causes dominate, including pregnancy-related spontaneous coronary artery dissection (43%), cases with normal large coronary arteries observed in coronary angiography (18%) and coronary artery thrombosis (17%) [2]. The risk of acute myocardial infarction (AMI) is also increased by some obstetric conditions (pre-eclampsia, thrombophilia, postpartum haemorrhage, blood product transfusion and postpartum infection) [3]. Compared to the older population (over the age of 55), the main risk factors in younger women include smoking and obesity [4]. It was also observed that the V Leiden, prothrombin *G20210A* gene and *MTHFR C667T* mutation increases the risk of myocardial infarction and stroke, especially at an early age [5].

Pregnancy-related acute coronary syndrome (ACS)/AMI occurs most frequently in the third trimester [ST-segment elevation myocardial infarction (STEMI) 25%, non-ST-segment elevation myocardial infarction (NSTEMI) 32%] or in the postpartum period (STEMI 45%, NSTEMI 55%). The clinical picture is the same as in the general population [1]. In case of STEMI, percutaneous coronary intervention (PCI) is preferred. The morbidity and mortality associated with AMI in pregnancy outweighs the potential teratogenic risk of coronary angiography. Although it is important to avoid unnecessary maternal and foetal exposure during pregnancy, this should not discourage doctors from performing recommended life-saving procedures [3, 6]. Young women with STEMI have worse prognosis than young men and they are more often rehospitalised within the next 6 months. This is due to the fact that they do not receive full pharmacological treatment and they are less frequently treated with invasive methods [4]. In pharmacotherapy, small doses of acetylsalicylic acid, beta-blockers and nitrates seem to be safe for the mother and the foetus, whereas angiotensin-converting enzyme inhibitors and statins are contraindicated [2, 7]. Information on the safety of glycoprotein IIb/IIIa inhibitors is derived from individual case reports. The use of those drugs during pregnancy should be limited; if they are administered to a patient in whom the labour begins, caesarean section should be performed to minimise bleeding complications in the child [3, 8].

Conclusions

The treatment of pregnant women with acute myocardial infarction poses a considerable challenge. In case of STEMI, the basic method is percutaneous revascularisation. As far as possible pharmacotherapy is concerned, it is possible to use acetylsalicylic acid, beta-blockers and nitrates, whereas statins and angiotensin-converting enzyme inhibitors are contraindicated. Full pharmacological treatment should be sought in accordance with the existing guidelines to improve prognosis in this group of patients.

Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Kobieta w ciąży z rozpoznaniem zawału serca to przypadek niezwykle rzadki w praktyce klinicznej. Otyłość i nikotynizm są dobrze znanymi czynnikami ryzyka zdarzeń sercowo-naczyniowych, które mają znaczenie zwłaszcza w populacji młodszych kobiet. W pracy omówiono przypadek 27-latki, którą hospitalizowano z powodu zawału serca z uniesieniem odcinka ST we wczesnej ciąży. Podstawą leczenia w takich przypadkach jest przezskórna interwencja wieńcowa. Problem stanowi optymalne dopasowanie farmakoterapii, a w jej planowaniu należy uwzględnić również bezpieczeństwo dla rozwijającego się płodu.

Słowa kluczowe: zawał serca, STEMI, ciąża, nikotynizm, otyłość

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Zawał serca z uniesieniem odcinka ST u 27-letniej kobiety w ciąży

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Streszczenie

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Słowa kluczowe: zawał serca, STEMI, ciąża, nikotynizm, otyłość

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Opis przypadku

Dwudziestosiedmioletnia, otyła, paląca tytoń pacjentka została przekazana ze szpitalnego oddziału ratunkowego do pracowni hemodynamicznej z rozpoznaniem zawału ściany dolnej z uniesieniem odcinka ST. Kilka godzin przed przyjęciem, w czasie spotkania ze znajomymi, nagle pojawił się u niej po raz pierwszy w życiu ból w klatce piersiowej, stopniowo zmniejszający swoje nasilenie w ciągu kilkudziesięciu minut. W chwili przyjęcia ból ten określany był przez chorą jako dyskomfort w klatce piersiowej. W badaniu przedmiotowym stwierdzono miarową czynność serca 117/min, ciśnienie tętnicze 140/77 mm Hg, skurczowy szmer na koniuszku, otyłość II stopnia — wskaźnik masy ciała (BMI, *body mass index*) 35 kg/m^2. Wywiad rodzinny był nieobciążony. W elektrokardiogramie (EKG) stwierdzono tachykardię zatokową o częstości 119/min, uniesienie odcinka ST o typie fali Pardeego o 3 mm w odprowadzeniach znad ściany dolnej oraz obniżenie odcinka ST do 2,5 mm w odprowadzeniach I, aVL, V2–V4 (ryc. 1).

Chora otrzymała 300 mg kwasu acetylosalicylowego, 180 mg tikagrelolu oraz 100 mg enoksaparyny. W wykonanej w trybie natychmiastowym koronarografii stwierdzono obwodową niedrożność gałęzi tylno-bocznej prawej tętnicy wieńcowej (prawdopodobnie o etiologii zatorowej; ryc. 2, 3). Pacjentkę zakwalifikowano do pilnej angioplastyki tętnicy dozawałowej. Do leczenia włączono inhibitor receptora glikoproteiny IIb/IIIa (eptifibatid), podjęto próbę udrożnienia naczynia, wykonano wielokrotne inflacje balonem. Uzyskano niewielką poprawę przepływu (TIMI [*Thrombolysis In Myocardial Infarction*] 1).

W badaniach laboratoryjnych obserwowano typową dla zawału serca dynamikę zmian stężenia troponiny I oznaczanej metodą wysokoczułą (maksymalne stężenie wynosiło 17 164,4 ng/l) oraz hiperlipidemię (stężenie cholesterolu całkowitego 195 mg/dl, stężenie cholesterolu frakcji LDL [*low-density lipoprotein*] 141 mg/dl). Stwierdzono prawidłową aktywność białek C i S oraz antytrombiny III. Wykonano badania genetyczne, w których nie wykryto mutacji czynnika V Leiden ani wariantu 20210A genu protrombiny, mutacji w obu allelach genów *F2*, *F5* oraz *MTHFR*.

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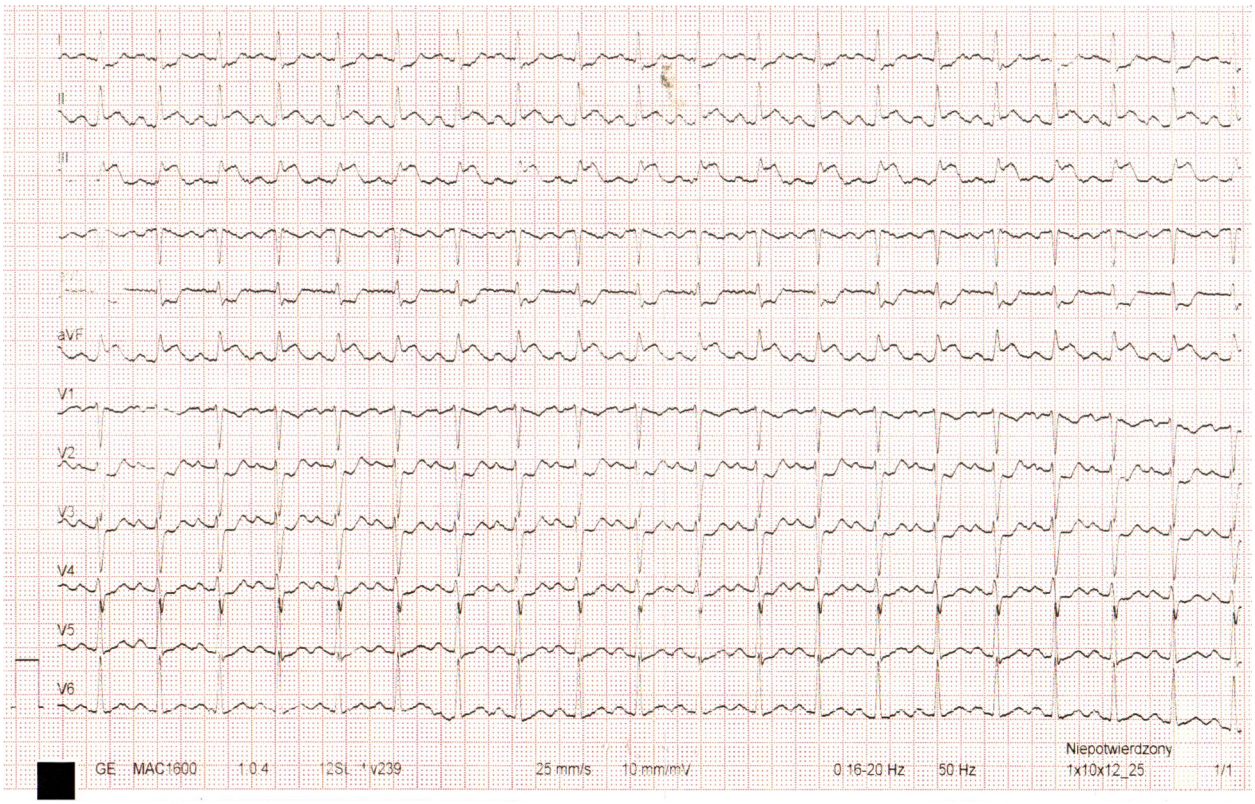
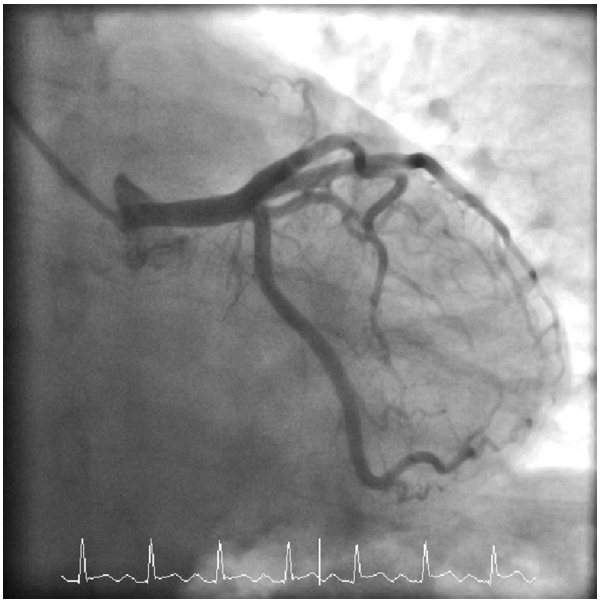
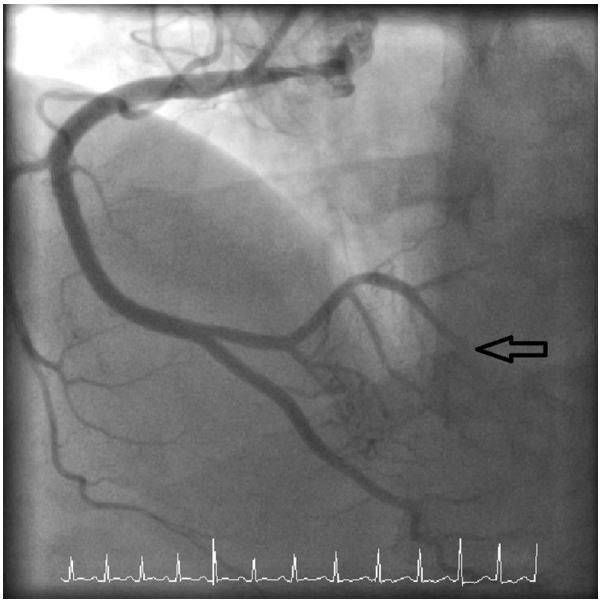


Rycina 1. Badanie elektrokardiograficzne przy przyjęciu chorej do kliniki



Rycina 2. Obraz angiograficzny prawidłowej lewej tętnicy wieńcowej



Rycina 3. Obraz angiograficzny prawej tętnicy wieńcowej. Strzałka wskazuje miejsce niedrożności gałęzi tylno-bocznej

W badaniu echokardiograficznym stwierdzono hipokinezę segmentu przypodstawnego ściany dolnej, a frakcja wyrzutowa lewej komory wynosiła 55%. W echokardiografii przezprzełykowej nie uwidoczniono ewidentnych cech ubytku w przegrodzie międzyprzedsionkowej ani materiału zatorowego w uszku lewego przedsionka.

W badaniu ultrasonograficznym (USG) metodą Dopplera nie wykazano cech zakrzepicy w układzie żył powierzchownych i głębokich kończyn dolnych. W 48-godzinnym monitorowaniu EKG metodą Holtera zarejestrowano rytm zatokowy o średniej częstości 79/min, bez napadów arytmii nadkomorowej. W USG jamy brzusznej nie wykazano istotnych odchyleń. Ze względu na opóźniające się wystąpienie krwawienia miesięcznego oraz sugestię chorej, że może być w ciąży, wykonano USG ginekologiczne, w którym stwierdzono możliwość wczesnej ciąży. Potwierdzono to również, stwierdzając typowy przyrost stężenia gonadotropiny kosmówkowej (hCG, *human chorionic gonadotropin*). Niezwłocznie zmodyfikowano terapię. Odstawiono statynę i tikagrelor.

Przebieg hospitalizacji był niepowikłany. Po wypisaniu chora była leczona enokasaparyną w dawce terapeutycznej, kwasem acetylosalicylowym w dawce 75 mg oraz bisoprololem w dawce 2,5 mg. Ciążę zakończono przez cięcie cesarskie w 40. tygodniu jej trwania, urodzeniem zdrowego noworodka płci żeńskiej o masie 3150 g.

Dyskusja

Choroba niedokrwienna serca jest rzadko spotykana w okresie ciąży (częstość występowania wynosi między 2,8 a 6,2/100 tys. porodów). Staje się jednak coraz bardziej powszechna, ponieważ kobiety decydują się na macierzyństwo w późniejszym wieku, gdy występują już u nich choroby współistniejące [1]. Choroba wieńcowa odpowiada za ponad 20% wszystkich zgonów matek z przyczyn sercowych. Dominuje etiologia niemiażdżycowa, w tym samoistne rozwarstwienie tętnicy wieńcowej związane z ciążą (43%), przypadki z prawidłowymi dużymi tętnicami wieńcowymi w ocenie koronarograficznej (18%) oraz zakrzepica w tętnicach wieńcowych (17%) [2]. Niektóre stany położnicze dodatkowo zwiększają ryzyko wystąpienia ostrego zawału serca (AMI, *acute myocardial infarction*) (stan przedrzucawkowy, trombofilia, krwotok poporodowy, transfuzja produktu krwi i zakażenie poporodowe) [3]. W porównaniu z kobietami starszymi (> 55. rż.) u młodszych częściej występują jako czynniki ryzyka nikotynizm oraz otyłość [4]. Stwierdzono także, że obecność mutacji czynnika V Leiden, genu protrombiny *G20210A* i mutacji *MTHFR C667T* zwiększają ryzyko wystąpienia zawału serca i udaru mózgu, zwłaszcza w młodym wieku [5].

Wystąpienie ostrego zespołu wieńcowego (ACS, *acute coronary syndrome*)/AMI związanego z ciążą zdarza się najczęściej w III trymestrze (zawał serca z uniesiem odcinka ST [STEMI, *ST-segment elevation myocardial infarction*] 25%, zawał serca bez uniesienia odcinka ST [NSTEMI, *non-ST-segment elevation myocardial infarction*] 32%) lub w okresie poporodowym (STEMI 45%, NSTEMI 55%). Obraz kliniczny jest taki sam jak w populacji ogólnej [1]. W STEMI preferuje się przezskórną interwencję wieńcową (PCI, *percutaneous coronary intervention*). Zachorowalność i śmiertelność związana z AMI w okresie ciąży przeważa nad potencjalnym teratogennym ryzykiem angiografii wieńcowej. Chociaż ważne jest, aby unikać niepotrzebnego narażenia dla matki i płodu podczas ciąży, to nie powinno to zniechęcać lekarzy do wykonania wskazanych procedur ratujących życie [3, 6]. Rokowanie u młodych kobiety ze STEMI jest gorsze niż u młodych mężczyzn; są częściej rehospitalizowane w ciągu 6 miesięcy. Jest to spowodowane faktem, że nie otrzymują pełnego leczenia farmakologicznego i są rzadziej leczone inwazyjnie [4]. W farmakoterapii małe dawki kwasu acetylosalicylowego, beta-adrenolityki i nitraty wydają się bezpieczne dla matki i płodu, natomiast inhibitory konwertazy angiotensyny i statyny są przeciwwskazane [2, 7]. Informacje dotyczące bezpieczeństwa inhibitorów receptora glikoproteiny IIb/IIIa pochodzą z opisów pojedynczych przypadków. Stosowanie tych leków w okresie ciąży powinno być ograniczone, a jeśli dojdzie do rozpoczęcia akcji porodowej u pacjentki, która je otrzymała, to należy wykonać cięcie cesarskie w celu zminimalizowania powikłań krwotocznych u dziecka [3, 8].

Podsumowanie

Leczenie ciężarnych kobiet z zawałem serca stanowi niemałe wyzwanie. W przypadku STEMI podstawę terapii stanowi rewaskularyzacja przezskórna. W farmakoterapii można stosować kwas acetylosalicylowy, beta-adrenolityki oraz nitraty, natomiast przeciwwskazane jest podawanie statyn oraz inhibitorów konwertazy angiotensyny. Należy dążyć do zastosowania pełnego leczenia farmakologicznego zgodnie z obowiązującymi wytycznymi, by poprawić rokowanie w tej grupie chorych.

Konflikt interesów

Autorzy nie zgłaszają konfliktu interesów.

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A percutaneous coronary intervention strategy for chronic total occlusion in a patient with severely impaired left ventricular systolic function. A wise move?

Przezskórna rewaskularyzacja wieńcowa przewlekle zamkniętej tętnicy wieńcowej u pacjenta z ciężką dysfunkcją skurczową lewej komory. Mądre posunięcie?

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Abstract

Percutaneous coronary intervention (PCI) for chronic total occlusion is a widely accepted revascularization procedure that accounts for around 10% of PCI procedures. Chronic totally occluded (CTO) coronary artery often is problematic, with most patients managed medically or referred for coronary artery bypass graft surgery due to the lack of standardized indication criteria. It has been shown that in patients with ischaemic heart failure left ventricular ejection fraction (LVEF) ≤ 35%, worse long-term outcome was related to the presence of CTO. However, it seems evident that improved survival and symptoms in patients with left ventricular dysfunction undergoing any myocardial revascularization is only achieved when viability is preserved. Here the authors present a case of CTO of the left anterior descending artery that was successfully treated with PCI resulting in subsequent left ventricular function improvement.

Key words: chronic total occlusion, CTO, coronary artery disease, percutaneous coronary intervention, complex PCI

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Introduction

The incidence of chronic total occlusion (CTO) in a Large French Registry (CARDIO-ARSIF) was 8.1% [1]. Undoubtedly, conscientious planning is crucial in CTO management, which includes treatment applicable to selected patients [2]. Percutaneous coronary intervention (PCI) for chronic total occlusion is a generally accepted revascularization procedure that accounts for around 10% of PCI procedures [3]. However, chronic totally occluded (CTO) coronary artery often is problematic, with the majority of patients managed medically or referred for coronary artery bypass graft surgery (CABG), due to the lack of standardized indication criteria., due to the lack of standardized indication criteria. Moreover, recent comparative studies of CTO PCI versus conservative treatment have shown ambiguous benefits of revascularization concerning both clinical and quality of life measures [4].

It has been shown that in patients with ischaemic heart failure left ventricular ejection fraction (LVEF) ≤ 35%, worse long-term outcome was related to the presence of CTO [5]. Although PCI might remain the only alternative to manage patients with low LVEF, a limited body of literature is available covering studies on outcomes of percutaneous CTO recanalization regarding viable myocardium. Nonetheless, in a study by Galassi et al. [6], LVEF improved significantly six months after successful CTO PCI in patients with LVEF ≤ 35%. Similarly, in a cardiac magnetic resonance study,

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including 29 CTO patients with LVEF ≤ 40%, Cardona et al. [7] showed that successful CTO PCI resulted in subsequent left ventricular function improvement. In the meta-analysis by Megaly et al. [8], which included studies using cardiac magnetic resonance (CMR) for quantification of volumes, demonstrated that successful CTO PCI was associated with a statistically significant increase in mean LVEF.

Even though regarding symptoms, patients with CTO particularly tend to adapt to their condition, they (not only those with low LVEF) frequently will experience dyspnoea more than from exertion angina. Galassi et al. [9] confirmed symptoms alleviation after CTO PCI: less dyspnoea in patients with low LVEF and less angina in those with preserved LVEF [6].

Nevertheless, here is presented a case of the CTO of the LAD that was successfully treated with PCI resulting in subsequent left ventricular function improvement.

Case report

In May 2019, a 52-year-old 20 pack-years smoker with no medical history presented for evaluation of chest pain radiating to the left upper extremity for about six months. Physical examination was grossly unremarkable. A surface electrocardiogram showed poor R-wave progression in V1–V3. Left ventricular function was severely diminished, with significant global wall motion abnormalities and a calculated ejection fraction of 27%.

He underwent coronary angiography due to the typical nature of his symptoms and high pre-test probability for coronary artery disease. Coronary angiogram revealed double-vessel coronary artery disease with a recanalized total occlusion of left anterior descending (LAD) and 80% occlusion of the proximal segment of the right coronary artery (RCA) (Figure 1). A drug-eluting stent (DES) was implanted in the RCA with adequate postprocedural TIMI (Thrombolysis In Myocardial Infarction) flow (Figure 2).

Subsequently, in June, the patient underwent CMR, which revealed viable myocardium in the LAD territory. European System for Cardiac Operative Risk Evaluation (EuroSCORE) II and J CTO score of 2.28% and 2 points, respectively. Therefore, it was decided that he would benefit after undergoing PCI for CTO of LAD. An antegrade wire escalating using Fielder FC wire with Finecross® microcatheter was attempted successfully. Predilatations with 1.5-×15-mm and 2.5-×20-mm balloons were performed. After an intracoronary injection of nitro-glycerine, two 3.0-×38-mm sirolimus-eluting coronary stents (CRE8 3.0 × 31 mm; 3.0 × 25 mm) were implanted at the lesion site in LAD resulting in TIMI III flow. During follow-up, the exercise capacity of the patient, as well as left ventricular function (40%), rapidly improved. Additionally, he was advised to quit smoking.

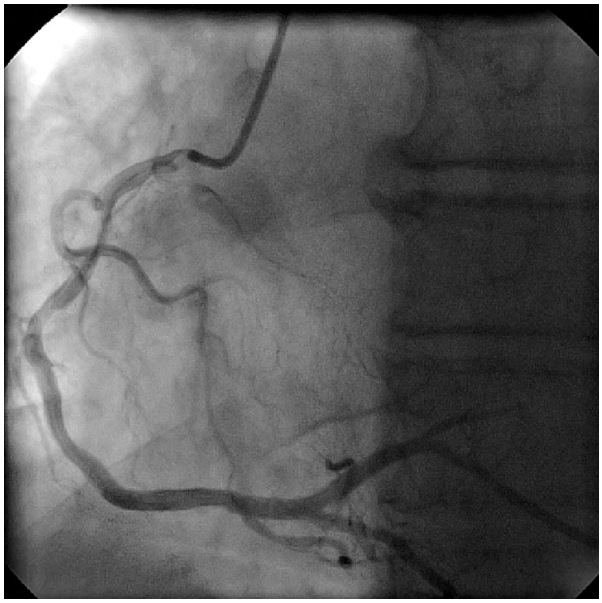

Figure 1. Left anterior oblique (LAO) caudal view showing right coronary artery 80% proximal occlusion

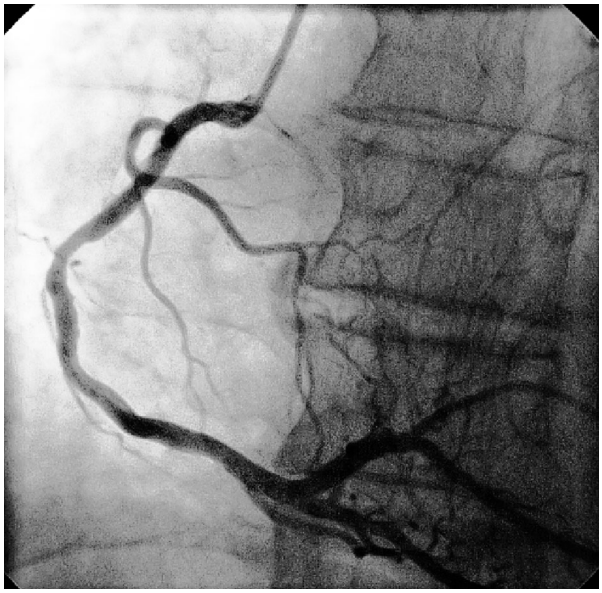

Figure 2. Left anterior oblique (LAO) caudal view showing post stenting of a proximal right coronary artery

Discussion and conclusion

Our findings are consistent with the generally accepted difference of at least 5% in LVEF as clinically significant left ventricular (LV) function improvement. Undeniably, according to the literature, percutaneous revascularization of CTOs in the setting of systolic dysfunction indeed

leads to improved LVEF and reduced adverse, resulting in positive clinical consequences. Accordingly, several observational studies showed that successful CTO revascularization is associated with improved quality of life [1]. However, it seems evident that the improvement of survival and symptoms in patients with left ventricular dysfunction undergoing any myocardial revascularization is only achieved when viability is preserved. This case study showed that despite significant radiation exposure and contrast dose, in high-risk patients, the advantages of CTO percutaneous procedure outweigh the disadvantages, primarily when performed by experienced PCI operators.

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Conflict of interest

The authors declare that there is no conflict of interest.

Streszczenie

Przezskórna angioplastyka (PCI) przewlekle zamkniętej tętnicy wieńcowej (CTO) jest powszechnie akceptowaną procedurą stanowiącą 10% zabiegów PCI. Przewlekłą okluzję tętnicy wieńcowej w większości przypadków, ze względu na brak jasnych wytycznych postępowania, leczy się zachowawczo lub kardiochirurgicznie poprzez operacyjne wszczepienie pomostów naczyniowych. Wykazano, że u pacjentów z kardiomiopatią niedokrwienną i frakcją wyrzutową lewej komory niższą lub równą 35% obecność CTO tętnicy wieńcowej wiąże się z gorszym rokowaniem. Oczywistym jest jednak, że korzyści w odniesieniu do przeżywalności i poprawy jakości życia u pacjentów z dysfunkcją skurczową lewej komory po rewaskularyzacji są osiągalne tylko wtedy, gdy zachowana jest żywotność mięśnia sercowego. Przytoczono przypadek kliniczny pacjenta po skutecznej PCI przewlekle zamkniętej gałęzi przedniej zstępującej lewej tętnicy wieńcowej z następczą poprawą funkcji skurczowej lewej komory.

Słowa kluczowe: przewlekle zamknięta tętnica wieńcowa, choroba wieńcowa, przezskórna rewaskularyzacja wieńcowa, złożona przezskórna rewaskularyzacja wieńcowa

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COVID-19 pandemic delays diagnosis and treatment of patients with acute coronary syndrome

Epidemia COVID-19 opóźnia proces diagnostyczno-leczniczy chorych z ostrym zespołem wieńcowym

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Abstract

We would like to present a case study of a 77-year-old patient who came to the hospital two weeks after an episode of severe chest pain due to fear of infection with SARS-CoV-2 virus. As a result of the delay. The patient developed numerous complications of myocardial infarction.

Key words: acute coronary syndrome, myocardial infarction, complications of myocardial infarction, rupture of the free wall of the left ventricle, ventricular tachycardia, mitral regurgitation

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Introduction

Acute coronary syndromes are associated with the risk of various types of early and late complications that worsen patients' prognosis. One of the most dramatic and with the highest mortality rates are severe ventricular arrhythmias and mechanical complications of myocardial infarction. The use of early revascularization resulted in a significant decrease in complications in acute coronary syndromes (ACS) [1, 2]. Fearing SARS-CoV-2 (severe acute respiratory syndrome-related coronavirus 2) infection, more than half of patients with myocardial infarction do not come to the hospital or arrive late, which significantly increases the risk of complications [3].

Case report

A 77-year-old patient was referred to the Department of Cardiology from the district hospital in a stable condition after suffering an episode of unstable ventricular tachycardia (VT) interrupted by electrical cardioversion. The patient reported chest pain that occurred two weeks before admission but did not go to the hospital at that time due to the coronavirus disease 2019 (COVID-19) pandemic. On admission, the patient was a feverish person with signs of pneumonia and negated the chest pain. The electrocardiogram (ECG) recorded a regular sinus rhythm of 80/min, disturbances in intraventricular conduction in the form of the right bundle branch block, and persistent ST-segment elevation in leads V2–V3. Laboratory tests showed a high concentration of inflammatory parameters and markers of myocardial necrosis without dynamic growth. Echocardiography showed akinesia of the inferior, inferolateral and lateral walls with a left ventricular ejection fraction of 45% and the presence of fluid in the pericardium with numerous streaks of fibrin up to 18 mm behind the lateral wall of the left ventricle (Figure 1). Based on the overall clinical picture, the patient was diagnosed with a previous myocardial

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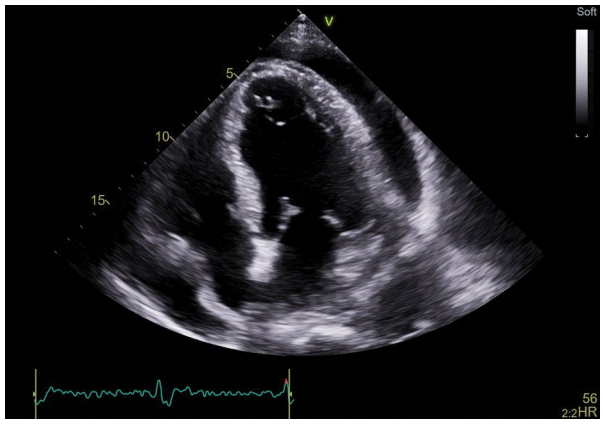

Figure 1. A significant amount of fluid in the pericardial sac as seen on a transthoracic echocardiographic examination

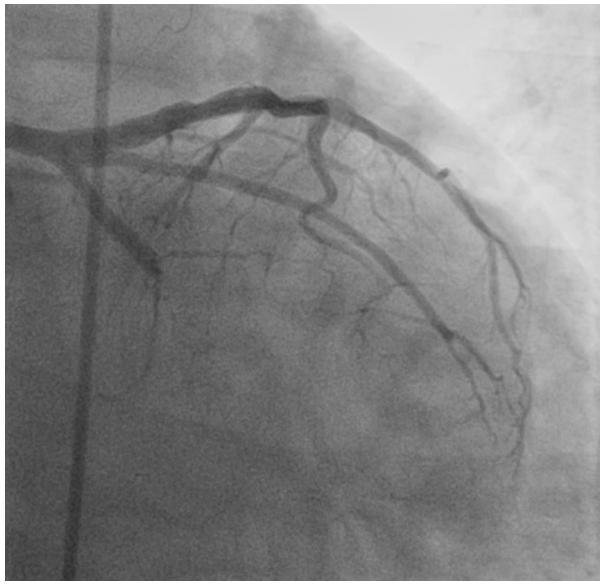

Figure 2. A branch that surrounds the left coronary artery closed in the middle segment

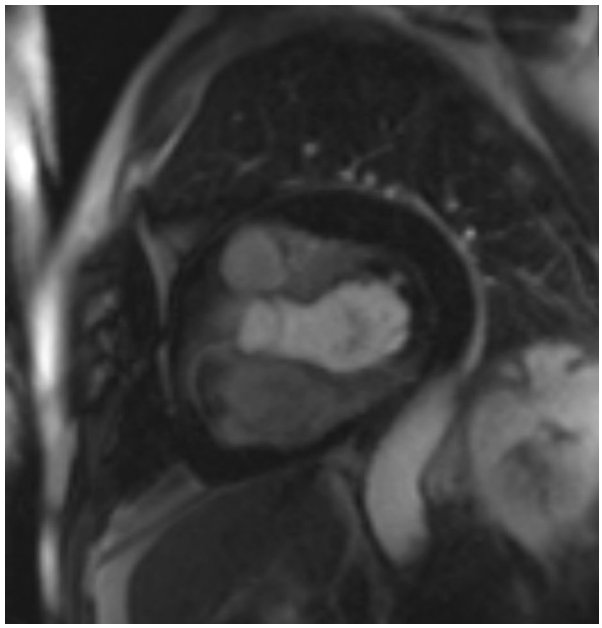

Figure 3. Magnetic resonance imaging with a slit-shaped, incomplete defect in the lateral wall of the left ventricle

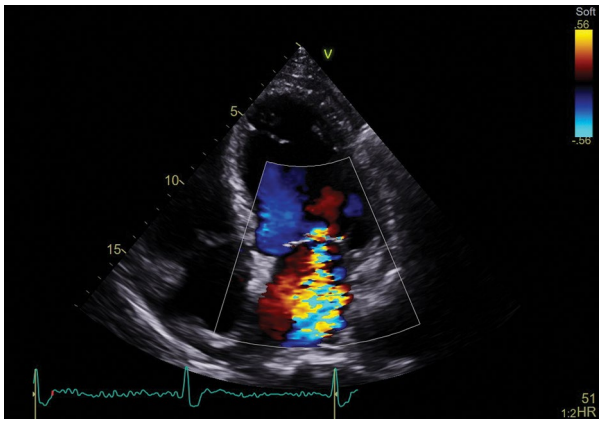

Figure 4. Severe functional mitral regurgitation

infarction and pneumonia, empirical antibiotic therapy was started, and the patient was not qualified for urgent coronary angiography. On the third day of hospitalization, he developed sustained ventricular tachycardia interrupted by an amiodarone infusion. The patient was qualified for coronary angiography, finding a closed circumferential branch, after a donation of two marginal branches, with a circumference showing from the collateral circulation, qualifying the patient for conservative treatment (Figure 2). After the procedure, recurrent ventricular tachycardias resistant to pharmacological treatment were observed; therefore, urgent ablation was performed to stop the arrhythmia. The control echocardiography showed fluid in the pericardial sac. Magnetic resonance imaging was performed, showing a fissured, incomplete defect in the lateral wall, filling with a contrast that might correspond to the perforation site, but no outflow of contrast to the pericardial sac was observed (Figure 3). Subsequent echocardiographic examinations showed a decreasing amount of fluid in the pericardial sacs. The stable patient was discharged home with an outpatient inspection in two weeks.

Follow-up

Follow-up echocardiography showed no haemodynamically significant amount of fluid in the pericardial sac but evidence of severe functional mitral regurgitation (Figure 4). The patient was consulted cardiosurgically and is currently awaiting mitral valve surgery.

Discussion

The reduced number of patients reporting myocardial infarction during the COVID-19 pandemic undoubtedly has a significant impact on the prognosis. An analysis carried out in Great Britain showed an increase in the total number of deaths during the pandemic, including deaths not related to SARS-CoV-2 infection [4]. Moreover, coronavirus infection may trigger acute coronary syndrome, similarly to the influenza virus, which may be associated with an even greater underestimation of the number of people who did not go to the hospital due to ACS [5].

Another factor that increases the risk of ACS complications is the lengthening of the time from the first contact with the health care system to revascularization. De Rosa et al. [6] analysed data from hospitalization of patients for myocardial infarction in Italy during the peak of the pandemic in March 2020. This study reported a 31.5% increase in the time to revascularization compared to March 2019. The increase in cases of patients with non-ST-elevation myocardial infarction (NSTEMI) treated conservatively was also observed, as well as a twofold increase in the number of serious complications [6].

Many hypotheses have sought to explain the reduction in hospitalization due to ACS during the COVID-19 pandemic. Stay-at-home orders and fear of SARS-CoV-2 infection likely discourage patients from seeking help from the healthcare system. In addition, the change in the organization of work in hospitals, including the creation of places and departments for the treatment of COVID-19 patients, may contribute to reduced availability of rapid diagnostics and treatment of cardiovascular diseases.

Conflict of interest

The authors declare no conflict of interest.

Streszczenie

Przedstawiono przypadek 77-letniego chorego, który w obawie przed zakażeniem SARS-CoV-2 zwlekał 2 tygodnie ze zgłoszeniem się do szpitala po epizodzie silnego bólu w klatce piersiowej. W wyniku opóźnienia w procesie diagnostyczno-leczniczym wystąpiły u nieego liczne powikłania zawału serca.

Słowa kluczowe: ostry zespół wieńcowy, zawał serca, powikłania zawału serca, pęknięcie wolnej ściany lewej komory, częstoskurcz komorowy, niedomykalność mitralna

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Epidemia COVID-19 opóźnia proces diagnostyczno-leczniczy chorych z ostrym zespołem wieńcowym

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Artykuł jest tłumaczeniem pracy: Bączek M, Starzyk K, Kośmider P, et al. The COVID-19 pandemic delays diagnosis and treatment of patients with acute coronary syndrome. Folia Cardiol. 2020; 15(5): 366–368. DOI: 10.5603/FC.2020.0053. Należy cytować wersję pierwotną

Streszczenie

Przedstawiono przypadek 77-letniego chorego, który w obawie przed zakażeniem SARS-CoV-2 zwlekał 2 tygodnie ze zgłoszeniem się do szpitala po epizodzie silnego bólu w klatce piersiowej. W wyniku opóźnienia w procesie diagnostyczno-leczniczym wystąpiły u nieego liczne powikłania zawału serca.

Słowa kluczowe: ostry zespół wieńcowy, zawał serca, powikłania zawału serca, pęknięcie wolnej ściany lewej komory, częstoskurcz komorowy, niedomykalność mitralna

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Wstęp

Ostre zespoły wieńcowe (ACS, *acute coronary syndromes*) są związane z ryzykiem wystąpienia różnego rodzaju wczesnych i późnych powikłań pogarszających rokowanie pacjentów. Do jednych z najbardziej dramatycznych i obarczonych wysoką śmiertelnością należą groźne komorowe zaburzenia rytmu serca oraz mechaniczne powikłania zawału serca. Zastosowanie wczesnej rewaskularyzacji spowodowało znaczące ograniczenie powikłań w ACS [1, 2]. W obawie przed zakażeniem SARS-CoV-2 (*severe acute respiratory syndrome-related coronavirus 2*) ponad połowa pacjentów z zawałem serca nie zgłasza się do szpitala lub przybywa do niego z opóźnieniem, co znacznie zwiększa ryzyko wystąpienia powikłań [3].

Opis przypadku

Pacjent w wieku 77 lat, w stabilnym stanie, po przebytym epizodzie niestabilnego częstoskurczu komorowego (VT, *ventricular tachycardia*) przerwanym kardiowersją elektryczną, został przekazany do kliniki kardiologii ze szpitala powiatowego. Chory podawał ból w klatce piersiowej, który wystąpił 2 tygodnie przed przyjęciem; nie zgłosił się wówczas do szpitala ze względu na pandemię COVID-19 (*coronavirus disease 2019*). W chwili przyjęcia gorączkował i przejawiał cechy zapalenia płuc, negował dolegliwości bólowe w klatce piersiowej. W zapisie elektrokardiologicznym (EKG) stwierdzono miarowy rytm zatokowy o częstości 80/min, zaburzenia przewodnictwa śródkomorowego pod postacią bloku prawej odnogi pęczka Hisa oraz przetrwałe uniesienie odcinka ST w odprowadzeniach V2–V3. W badaniach laboratoryjnych obserwowano wysokie stężenie parametrów zapalnych oraz markerów martwicy mięśnia sercowego bez dynamicznego narastania. W badaniu echokardiograficznym uwidoczniono akinezę ściany dolnej, dolno-bocznej oraz bocznej z frakcją wyrzutową lewej komory wynosząca 45% oraz obecność płynu w osierdziu z licznymi pasmami włóknika do 18 mm za ścianą boczną lewej komory (ryc. 1). Na podstawie całości obrazu klinicznego rozpoznano przebyty zawał serca oraz zapalenie płuc i włączono antybiotykoterapię empiryczną; nie

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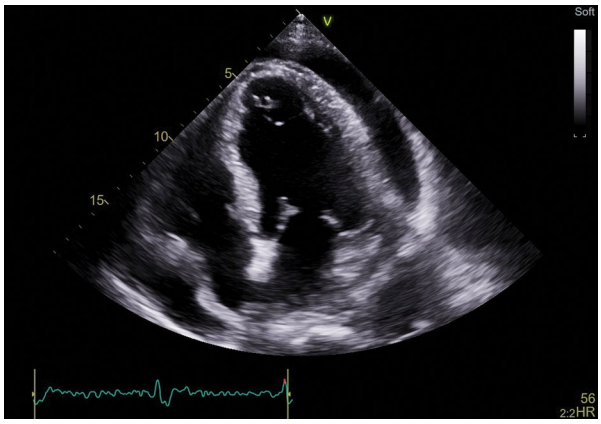
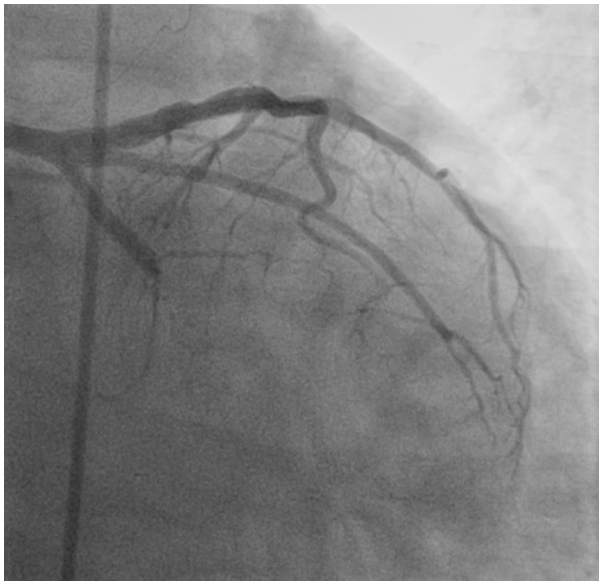
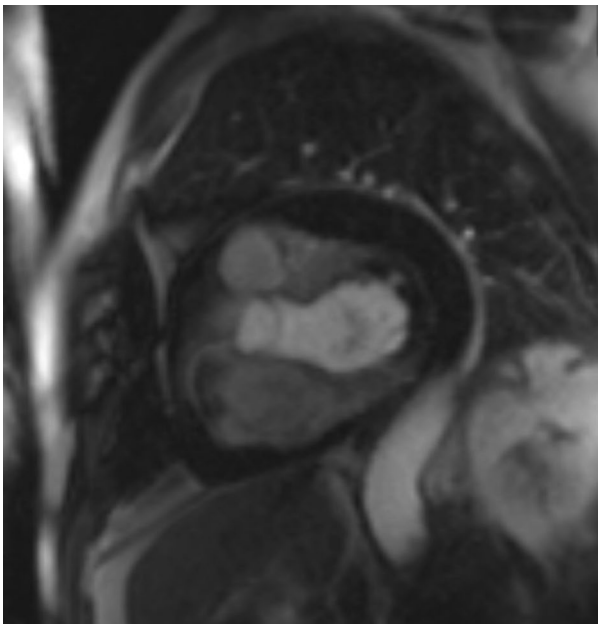


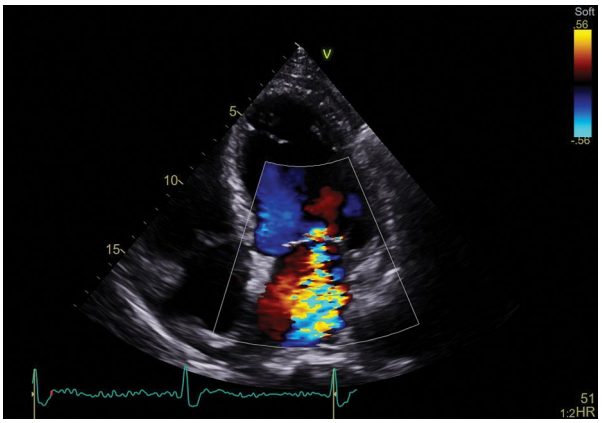
Rycina 1. Obecna istotna ilość płynu w worku osierdziowym uwidoczniona w przezklatkowym badaniu echokardiograficznym



Rycina 2. Zamknięta w segmencie środkowym gałąź okalająca lewej tętnicy wieńcowej



Rycina 3. Uwidoczniony za pomocą rezonansu magnetycznego szczelinowaty, niepełnościenny ubytek w obrębie ściany bocznej lewej komory



Rycina 4. Ciężka czynnościowa niedomykalność mitralna

zakwalifikowano chorego do koronarografii w trybie pilnym. W 3. dobie hospitalizacji wystąpił utrwalony VT przerwany podaniem wlewu amiodaronu. Chorego zakwalifikowano do koronarografii, stwierdzając zamkniętą, po oddaniu dwóch gałęzi marginalnych, gałąź okalającą z obwodem pokazującym się z krążenia obocznego, kwalifikując pacjenta do leczenia zachowawczego (ryc. 2). Po zabiegu obserwowano nawracające częstoskurcze komorowe oporne na leczenie farmakologiczne, dlatego w trybie pilnym wykonano ablację, uzyskując przerwanie arytmii. W kontrolnym badaniu echokardiograficznym utrzymywał się płyn w worku osierdziowym. Wykonano badanie rezonansu magnetycznego, uwidoczniając szczelinowaty, niepełnościenny ubytek w obrębie ściany bocznej wypełniający się kontrastem mogący odpowiadać miejscu perforacji, natomiast nie obserwowano wypływu kontrastu do worka osierdziowego (ryc. 3). W kolejnych badaniach echokardiograficznych uwidoczniono zmniejszającą się ilość płynu w worki osierdziowym. Chorego w stanie stabilnym wypisano do domu z zaleceniem kontroli ambulatoryjnej za dwa tygodnie.

Follow-up

W kontrolnym badaniu echokardiograficznym nie uwidoczniono istotnej hemodynamicznie ilości płynu w worku osierdziowym, natomiast obecne były cechy ciężkiej czynnościowej niedomykalności mitralnej (ryc. 4). Chorego konsultowano kardiochirurgicznie; obecnie czeka na zabieg plastyki zastawki mitralnej.

Dyskusja

Zmniejszona zgłaszalność pacjentów z zawałem serca w trakcie pandemii COVID-19 ma, bez wątpienia, znaczący wpływ na rokowanie. W analizie przeprowadzonej w Wielkiej Brytanii wykazano wzrost ogólnej liczby zgonów w trakcie pandemii, w tym również tych niezwiązanych z zakażeniem SARS-CoV-2 [4]. Co więcej, infekcja koronawirusowa może być czynnikiem wyzwalającym ACS, podobnie jak to się dzieje w przypadku wirusa grypy, co może być związane z jeszcze większym niedoszacowaniem liczby osób, które nie zgłosiły się do szpitala z powodu ACS [5].

Kolejnym czynnikiem zwiększającym ryzyko wystąpienia powikłań ACS jest wydłużenie czasu od pierwszego kontaktu z systemem ochrony zdrowia do rewaskularyzacji. De Rosa i wsp. [6] przeanalizowali dane z hospitalizacji pacjentów z powodu zawału serca we Włoszech w trakcie szczytu pandemii w marcu 2020 roku. W badaniu tym odnotowano wydłużenie czasu do rewaskularyzacji o 31,5% w porównaniu z marcem 2019 roku. Zaobserwowano również względny wzrost przypadków pacjentów z zawałem serca bez uniesienia odcinka ST (NSTEMI, *non-ST-elevation myocardial infarction*) leczonych zachowawczo oraz 2-krotny wzrost liczby poważnych powikłań [6].

Istnieje wiele hipotez tłumaczących ograniczenie liczby hospitalizacji z powodu ACS w trakcie pandemii COVID-19. Nakazy pozostania w domu oraz strach przed zakażeniem SARS-CoV-2 najprawdopodobniej zniechęcają pacjentów do szukania pomocy w systemie ochrony zdrowia. Ponadto zmiana organizacji pracy w szpitalach, w tym tworzenie miejsc oraz oddziałów zajmujących się leczeniem pacjentów z COVID-19, może się przyczyniać do zmniejszenia dostępności do szybkiej diagnostyki i leczenia chorób układu sercowo-naczyniowego.

Konflikt interesów

Autorzy nie zgłaszają konfliktu interesów

Piśmiennictwo

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Functional evaluation of the upper extremities after corrective surgery of congenital defects of the aortic arch in children depending on arterial vascularisation

Ocena funkcjonalna kończyn górnych po operacjach naprawczych wad wrodzonych łuku aorty u dzieci w zależności od unaczynienia tętniczego

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Abstract

Congenital heart defects are often accompanied by defects in great vessels, such as aortic coarctation with various forms of aortic arc hypoplasia, vascular rings and positional anomalies. Surgical treatment and the choice of optimal defect correction technique in the child may affect the restoration, maintenance or the arrest of blood supply by the aorta to arterial vessels given off by the aortic arch. Proper, closest physiological vascularisation of individual parts of the body and organs is necessary for proper development and maintenance of physiological function. The consequence of the use of various strategies for correcting congenital defects of the aorta and the use of available surgical techniques may be perfusion disorders, also in the field of vessels supplying upper limbs, most often the upper left limb.

In this article, we have attempted to develop a universal strategy for assessing the development and function of the upper extremities in children. It consists of cardiac methods of cardiovascular evaluation and physiotherapeutic function tests of the upper limbs adapted to the age of patients. Confirmation of the long-term importance of maintaining or restoring the blood supply by the aorta on the basis of objective function tests of the upper extremities may be an additional argument while choosing the optimal surgical method in the youngest children.

Key words: congenital heart defects, defects of the vessels of the aortic arch, pediatric cardiac surgery, coarctation of the aorta, hypoplasia of the aortic arch, cardiosurgical correction, evaluation of the upper extremities, muscle strength, EMG, AHA

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Introduction

In this article we attempt to develop a universal strategy for assessing the development and function of the upper extremities in children. It consists of cardiac methods of cardiovascular evaluation and physiotherapeutic function tests of the upper limbs, adapted to the age of patients. Confirmation of the long-term effects of maintaining or restoring the arterial blood supply originating from the aorta on the basis of objective function tests of the upper extremities may be an additional argument for choosing the optimal surgical method in the youngest children.

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Congenital heart and vascular abnormalities also concern structural abnormalities of vessels giving off by the aortic arch. This can cause abnormal blood flow and give clinical symptoms in the area of their atypical pattern and vascularisation, including the organs of the mediastinum, upper extremities and the head. The aim of surgical treatment of congenital heart defects is to correct all reparable anomalies that can be corrected. As a result, blood circulation is as close to the physiological standard as possible, regardless of the extent of primary, congenital defect. The correction of congenital defects also includes corrective procedures of the main artery and vessels giving off by its arch, *i.e.* vessels supplying the head. Vascular intervention usually changes their spatial orientation and position as well as in the blood supply of the corrected area. This fact may be important for the further functioning of both the arteries as well as the stuctures and organs vascularised by them.

The most common vascular-related congenital anomalies of the aortic arch include coarctation of the aorta with various forms of aortic arch hypoplasia, vascular rings and positional anomalies of the aorta and pulmonary artery in the mediastinum. The surgical technique selected for the treatment of these defects may affect the maintenance or restoration of the blood supply from the aorta to the orginating arteries, which is possible by changing its position, functional purpose, and spatial orientation. This may require an additional corrective procedure.

Traditional surgical strategies for the correction of congenital anomalies of the heart and mediastinal vessels have been developed to improve arterial blood circulation in the basic vascular structures, which is the main goal. The complex methods of the blood supply reconstruction to all originating vessels require modification of the surgical technique with an extension of the surgical field, duration of the procedure and degree of the intervention into the thoracic cavity. The patient's early age, *i.e.* neonatal period and infancy makes it even more difficult to make the decision to change the surgical strategy. Technical aspects such as low body weight and small size of a child may also cause difficulties. This is in contradiction to the rational premise that the simultaneous correction of all the reparable defects may have a positive impact on the child's further development.

Assumptions

Proper arterial vascularisation of tissue and organ is based on proper development and maintenance of physiological function. The maintenance of physiological vascularisation of the shoulder girdle and upper extremities in patients after cardiovascular correction procedures should therefore guarantee the proper development and function of these regions.

The anatomy of the defect and the way it is corrected cause hemodynamic effects, both on the total blood circulation in the body and on the blood flow in the carotid and vertebral vessels, directly adjacent to the region of the coarctation of the aorta and the hypoplastic aortic arch. The consequences of the use of various strategies for correcting the aortic coarctation and the use of a specific surgical technique, frequently with necessary arrest of blood flow, may include perfusion disorders, also in the field of vessels supplying upper limbs, usually the left upper limb.

As a result of the aortic arch anomaly with a limited blood supply originating from the aorta to the left subclavian artery, which is operated on in children (frequently in a newborn), the long-term consequence of the procedure may be limitation of the functional efficiency of the left upper extremity.

Aim of the study

To assess the late consequences of maintaining vascularisation by the aorta we have attempted to develop a universal strategy for assessing the development and function of the upper limbs in children after surgery of congenital heart and vascular defects, using the methods for cardiological assessment of the cardiovascular system and physiotherapeutic tests for functional evaluation of the upper extremities, which are age-appropriate and adapted to development of examined patients.

Confirmation of the long-term importance of maintaining or restoring the arterial blood supply originating from the aorta on the basis of objective, minimally invasive function tests of the upper limbs may be an additional, important argument in the discussion about the proper choice of optimal surgical strategy in the youngest children.

The most common congenital defects of the aortic arch and great arteries given off by the aortic arch — including initial anatomy and pathophysiology

Coarctation of the aorta with aortic arch hypoplasia

Coarctation of the aorta (CoA) is a congenital heart defect defined as aortic narrowing in the area of the aortic isthmus [1]. This defect occurs in approx. 0.4% of newborns and 6–8% of children with congenital heart defects [2]. It may occur as an isolated anomaly, or may be a component of other defects, resulting in a variety of clinical symptoms and threats to the whole body. In a newborn with critical CoA, only an early start of intensive treatment ensures their survival. This defect is classified as ductus-dependent critical one.

The basic literature describes two types of anatomy of both the aorta and aortic arch, treated as physiological forms. Type I, the most common variant (74.0–89.4% of the population), consists of three great vessels emerging from the aortic arch. The first vessel is the brachiocephalic trunk, which is divided into the right subclavian artery and the right common carotid artery. The second vessel is the left common carotid artery, and the last structure branching off from the aortic arch is the left subclavian artery. The second variant is so-called 'bovine trunk' (a profile of a bull) and occurs in 7.2–21.1% of the population [3]. Some authors also distinguish two subtypes of the second variant: 'long bovine trunk', which is type II-A, and 'short bovine trunk', which is type II-B [4]. It should be emphasized that both variants are physiological forms, with maintaining the proper blood flow, and all elements of the proper anatomy of the vessels.

In the congenital defect of the heart and vessels, called 'bovine trunk', the brachiocephalic trunk and the left carotid artery give off as a common vessel in the continuation of the ascending aorta. The proximal aortic arch does not develop in this anomaly. The continuation is the distal aortic arch with a relatively long shape reaching the region of the left subclavian artery and the aortic isthmus. In the form of a neonatal defect the peripheral flow of blood to the organs situated below the aortic arch is provided by an unobstructed arterial duct.

Vascular rings with the anomaly of the pattern of subclavian arteries

The subclavian artery ensures the proper arterial vascularisation of the upper extremity, which, after branching off from the brachiocephalic trunk on the right or distal aortic arch (on the left), becomes a strong axillary artery responsible for supplying the upper limb muscles with the shoulder girdle [5]. The axillary artery starts at the outer edge of the first rib. It ends at the height of the edge of the lower pectoralis major. The axillary artery is supported by a small number of branches connecting to the carotid branches of the subclavian artery and intercostal arteries.

A heart defect called a vascular ring contains many anatomical forms. From the point of view of the upper limb vascularisation, the anomalies of the origin and pattern of subclavian arteries, which incorrectly give off by the aortic arch on the opposite side of the extremity supplied, are of great importance. Such a position of arterial vessels causes compression symptoms of the mediastinal organs, including vitally important ones: trachea, bronchi, and esophagus. In the international nomenclature, the Latin term *arteria lusoria* has been adopted, which means the (right or left) aberrant artery.

Impression (compression) of vascular structures on mediastinal organs

The position of the aortic arch with its version, referred to as either left or right one, or in an intermediate position, specified clinically as 'zero' one may cause compression symptoms, additionally exacerbated by the basic hemodynamic parameters of the aorta: high blood pressure, high pulse wave with high blood flow volume and stiffness of its wall.

Patients with disadvantageous position and spatial orientation of the aorta in its initial, mediastinal segment show symptoms of impression, similar to those typical of the presence of a vascular ring.

Functional muscles of the upper extremities, responsible for physiological function of the limbs

The key muscles in the upper extremity function include rotator cuff, deltoid muscle, biceps and triceps, flexor and extensor groups of the wrist and hand [6]. The above-mentioned muscles also need vessels supplied from the aorta with an appropriate diameter and capacity to function properly. Adequate arterial vascularisation also seems to be a natural condition for the physiological development of the extremities, which is consistent with the growth of the whole body during childhood.

Cardiosurgical correction techniques in the treatment of vascular defects of the aortic arch

Standard methods of repair of aortic coarctation with hypoplasia of the aortic arch

The preferred treatment strategy today is an early repair of the aorta within the limits of its own healthy tissues. Coarctation of the aorta is often accompanied by various forms of underdevelopment (hypoplasia) of the aortic arch [7]. The traditional surgical treatment consists in the resection of the narrowing region with the removal of abnormal tissues; in some techniques — with the resection of the carotid and vertebral vessels, mainly the left subclavian artery. Performing the most popular corrective surgery in newborns and infants with low body weight makes it difficult or impossible to maintain the left subclavian artery, in which the blood flow is ensured by the presence of the collateral circulation. The sources of the collateral circulation for the severed subclavian artery include the internal thoracic artery and the vertebral artery — via the Circle of Willis, and vessels in the region of the shoulder girdle. Deprivation of vascularization by the aorta for such an important vessel as the subclavian artery, which supplies large muscle parts

of the upper extremity, predisposes to the formation and the symptoms of the so-called steal syndrome due to the backflow of blood in arteries. The symptoms of the steal syndrome, which are the most difficult to compensate, include neurological complications resulting from CNS hypoperfusion in the circle of Willis. The most common early symptoms in patients include headaches, dizziness and syncopes that are dangerous to life and health.

A modified technique of coarctation resection with narrowing of the aortic isthmus and proximal anastomosis by end-to-side method, with reconstruction of the blood supply to the left subclavian artery using a hypoplastic, distal segment of the aortic arch

Correction of the 'bovine trunk' anomaly in a newborn is possible by means of a standard lateral access. In the initial stage of the operation the blood supply to the left subclavian artery is reconstructed using a distal segment of the hypoplastic aortic arch, which smoothly changes into the left subclavian artery. By maintaining an infusion of prostaglandin and arterial duct patency, the brain and both upper limbs are supplied from heart while the lower part of the body is perfused from the duct. After ligation of the arterial duct, the descending aorta is closed with a vascular clamp and the narrowed aortic isthmus is extensively resected along with the periductal tissue, followed by — after lateral incision of the proximal part of the aortic arch at the base of the common brachiocephalic trunk and large malalignment of the descending aorta — performing the end-to-side anastomosis. An additional complication may be the anatomical variant including the subclavian artery origin in the area of the narrowed aortic isthmus or even below — from the periductal tissue, which requires independent transplantation of the left subclavian artery to the trunk of the left common carotid artery, with end-to-side anastomosis.

After this procedure with end-to-side anastomosis, the blood flow to the descending aorta is bypassed by a segment of the aortic arch and its hypoplasitic structure is used only to maintain continuity and to provide blood supply originating from the aorta to the left subclavian artery. The main advantage of this technique is that it can be performed without cardiopulmonary bypass, using lateral access, with the use of healthy, patient's own tissues and within their borders, while maintaining blood supply by the aorta to all arteries.

Clinical evaluation of a patient after aortic arch surgery

Diagnosis and imaging methods

The most important diagnostic issue is the initial anatomy of vascular anomalies. In patients with coarctation the intensity of aortic narrowing in the area of the aortic isthmus, aortic arch hypoplasia, as well as development and orientation of vessels giving off by the aortic arch are evaluated. Children with the vascular ring diagnosis require aortic arch identification and detailed evaluation of both the proximal segment of origin of carotid and vertebral vessels and their further pattern. In patients with symptoms of great vessel impression, the position of the aortic arch with its version, referred to as either left or right one, or in an intermediate position, specified clinically as 'zero' one, requires detailed imaging. In addition, the condition of the organs in the region of pressure is evaluated, with an assessment of the degree and permanence or irreversibility of their damage. Imaging tests are repeated many times during the postoperative period and are also the basis for the clinical assessment of a patient in a long-term follow-up.

Echocardiography

Two-dimensional echocardiography (ECHO) provides excellent imaging in the youngest children, newborns and infants. Currently, this is a standard examination performed in newborns with heart and vessel anomalies. Very often it is sufficient for the proper eligibility for surgical treatment. It is also a basic postoperative follow-up test.

Angiography

Traditional angiography (ANGIO) is used in case of echocardiographic doubts or in patients with unclear aortic arch anatomy, those with suspicion of additional anomalies and in older patients. Angiographic examination allows a precise assessment of most arterial vessels and blood flows but requires the administration of contrast agents.

Magnetic resonance imaging

The aortic arch and proximal segment of the descending aorta are well represented structures in magnetic resonance imaging (MRI). This method provides better 2D images and spatial reconstructions than any other non-invasive technology. MRI also allows detailed imaging of the vasculature in relation to other mediastinal structures. In this examination it is also possible to evaluate the condition of tissues and organs adjacent to the vessels.

Computed tomography

The main advantage of computed tomography (angio-CT) is the precise imaging in patients after implantation of metal stents and with high risk of forming aneurysms in the postoperative period and with contraindications to use MRI [8].

Methods of functional evaluation of the patient's upper extremities in the remote period after aortic arch vessel surgery

Prior to the anthropometric, biomechanical, and functional evaluation of the upper extremities, the functional hand

dominance should be determined. Information concerning right- or left-handedness is necessary to carry out a comparative analysis of extremities in terms of their efficiency or strength, especially in older children. Considering the obtained test results exclusively through the prism of the right and left side of the body may disrupt the process of deducing and be of no use to clinicians and patients, especially since the percentage of left-handed people is approximately 10% of the population [9]. One of the tools to determine the functional dominance of the upper limb in a child is the Peg Moving Task (PMT-5). The test consists in placing 5 sticks in the holes of a wooden board as soon as possible. The hand that completed the task in a shorter time is considered as dominant [10]. In older children, the dominant upper limb is the hand the child writes, uses a spoon, toothbrush, or scissors [11].

Examining the length and circumferences of the upper limb

Disturbance in the main arterial blood flow to the upper limb in adults usually results in the establishment of collateral circulatory system capable of maintaining limb life and function. In children, a similar dysfunction may adversely affect the nutrition of limb tissues, and thus disturb their growth and function [12]. In addition to measurment of the relative and absolute length of the upper limbs, it is useful to include segmental measurements of the arm and forearm to accurately determine the region of long bone growth disorder. Measurements of circumferences, particularly the first (R1) arm (short and long) one, second (R2) arm one and first (P1) forearm one, can provide information concerning soft tissue hypotrophy.

Hand grip strength (HGS) test

The Electro-Hydraulic Hand Dynamometer is acknowledged by the American Society of Hand Therapist as an objective tool for monitoring HGS in patients after cardiac surgery [13]. The standard protocol assumes the measurement of isometric force in a standing position [14], therefore, by reason of the test specificity and the possibility of a sudden increase in blood pressure, a sitting position with the arm slightly splayed from the body and the limb bent in the elbow joint up to 90° is recommended in cardiac patients (according to JAMAR protocol) [15]. The maximum hand grip must be maintained for at least 3 seconds [16]. The device is standardized and can be used in patients aged over 4 [17].

Muscle strength test

Biodex System PRO4 Dynamometer allows precise measurement of muscle strength moments under isometric (static), isokinetic (dynamic) and isotonic conditions in both healthy people and those in the rehabilitation process. The device can be used to objectively measure the strength for individual limb movements in the humeral [18] and elbow joints [19] within the full range of patient movements [20]. In addition, it is possible to determine the relationship between antagonistic muscle strength. The aim is to determine the proportion of muscle strength within a given joint [21, 22]. The measurement of muscle strength moments for both upper limbs makes it possible to directly compare them and determine the strength symmetry [21].

In patients after cardiac surgery it is not recommended to perform measurements under isometric conditions due to the excessive strain on the body by static work. In the rehabilitation process, measurements are frequently performed under isokinetic conditions [22, 23], in which the movements are performed with a constant, programmed by the researcher, value of angular velocity in the joint, and the generated resistance is adjusted individually to the patient's abilities.

The data used for the analysis is the maximum strength moment reached at a given angular velocity (absolute and relative values, *i.e.* related to the examined person's body weight), total work, maximum and average strength and fatigue index. The value of the strength moment may be affected by such factors as age, gender, BMI, or extremity dominance [24]. It is possible to adapt the dynamometer arms for children aged 6 and older [25].

Testing of electrical activity of muscles

Electromyography (EMG) is concerned with the study of changes in the electrical activity of muscles in particular time periods [26]. Surface electromyography (sEMG) is useful in complementing the diagnosis of neuromuscular diseases in children [27]. Due to possible postoperative neurological complications in cardiac patients, conducting a non-invasive examination using sEMG while performing selected motor tasks may provide information concerning the existing disorder and differences in muscle activation patterns [26]. Commonly used recommendations for placing electrodes on the skin are SENIAM guidelines [28].

To compare the examined patients to one another and possibly monitor the rehabilitation process, it is necessary to normalize the EMG signal amplitude, *i.e.* to compare the measured value with a reference value, which is obtained depending on the selected method. The MVIC (MVC) technique (maximum voluntary isometric contraction) assumes the execution of maximum isometric contraction [26]. The validity of this normalization technique should be considered in the case of cardiac patients in whom this could lead to deterioration of their health status. Other ways of normalization include obtaining of a reference value from the test, taking the maximum amplitude value of 100% or an average value, and it is also possible to measure the reference value for the conditions of known, non-maximum constant loading of the muscle group under static conditions [26].

The analysed data usually include the mean amplitude of the signal, the surface area under the signal envelope, the

mean and median frequency spectrum of EMG signal and the analysis of temporal characteristics of the muscle function.

Due to the ease of testing, electromyography has become a common tool to study the action potential of muscles, but it should not be equated with measuring muscle strength or power [26].

Infrared thermography imaging

The undisturbed function of the cardiovascular system is a precondition for appropriate development of the limb and maintaining its full functionality. The cardiovascular system is responsible i.a. for maintaining adequate temperature of tissues, thus infrared thermography imaging may be helpful in finding impaired blood supply in the upper limbs. They may manifest as a decrease in temperature of peripheral structures. Infrared thermography imaging, due to its minimal invasiveness and ease of use, is applied in body temperature tests in both adults and children [29]. This examination can only complement the basic tests, which assess blood flow in upper limb vessels. The existing literature does not contain clear indications for the use of infrared thermography and diagnostic protocols.

Small motor skills test

The assessment of the functional condition of the upper limb should be based on the small motor skills test. A commonly used tool is Assisting Hand Assessment (AHA). AHA measures and describes how children with one-sided disability use the affected hand to perform tasks/activities involving both hands. It consists of 22 parts subject to observation, which, depending on the quality of performance, are rated from 1 to 4 points [30]. AHA has been developed for children aged between 18 months to 12 years and allows monitoring of the rehabilitation process [31]. For children aged between 18 months and 5 years, the study involves observation of spontaneous play with toys requiring two hands (Small Kids AHA). A board game (School Kids AHA) has been developed for children aged 6–12 years, which provides an age-appropriate context for using the same toys as AHA for small children [32]. AHA is considered a useful clinical and research tool [33]. In 2017, the first tool assessing the hand function in children aged 3–12 months was developed — Hand Assessment for Infants (HAI). This tool consists of 31 parts assessed on a scale from 0 to 2 points depending on the quality of the presented infant's motor skills [34]. Both AHA and HAI are used in the diagnosis of children with neurological disorders, especially with cerebral palsy [30, 34].

Evaluation of psychomotor development of children after aortic arch surgery

In addition to the above instruments, which are strictly dedicated to the assessment of upper limb functions, there are a number of tools for comprehensive evaluation of the child's psychomotor development, the individual modules of which also cover the areas of small motor skills. In 2012, the American Heart Association (AHA) published a statement recommending continuous monitoring of the development of children and adolescents with congenital heart defects using standardized assessment tools. To determine the level of development of small motor skills and hand functions, AHA recommends using the following methods and tests: the Bayley Scales of Infant and Toddler Development — Third Edition, Bruininks-Oseretsky Test of Motor Proficiency — Second Edition (BOT-2), Grooved Peg Board, NEPSY-II, Peabody Developmental Gross Motor Scale (PDMS-II), Scales of Independent Behavior — Revised (SIB-R), Vineland Adaptive Behavior Scales — Third Edition (Vineland-3), Test of Visual-Motor Integration, Supplemental-Motor Coordination II (VMI) [35].

The Bayley Scales of Infant and Toddler Development (Bayley-III) is the "golden standard" in the assessment of infants and children with developmental disorders, as well as in monitoring the effectiveness of the therapeutic measures taken. The Bayley-III is a standardized tool for evaluating the development of children aged from 1 month to 42 months. The Bayley-III contains a subscale evaluating the development of small motor skills, *i.e.* precision hand movements. The subscale of precision motor skills is part of the overall assessment of a child's development but can also be treated as an independent part of the test. The evaluation of small motor skills is based on the examination of 66 characteristics and measures such abilities as hand grip quality, manual dexterity, handling of objects, visual-motor coordination, and movement dynamics. The scores obtained in the test are compared with adopted standards [36].

The above evaluation tools are also useful to comprehensively assess the motor deficits of the upper limbs, which may occur after cardiac surgery in the youngest patients.

Conclusions

Anthropometric, biomechanical and functional evaluation of upper extremities in children in long-term follow-up after surgery of aortic arch vascular anomalies allows an additional evaluation of the effectiveness and possible side effects of individual surgical methods.

Monitoring the development and efficiency of upper extremities in children treated for the most common congenital heart and vascular defects, with consequences in the form of blood supply disorders, should be based on a basic hemodynamic evaluation, as well as repetitive, non-invasive diagnostic tools and tests that check structural lesions within the upper limbs, as well as function tests, including, above of all, small motor skills.

Streszczenie

Wrodzonym wadom serca często towarzyszą wady wielkich naczyń, takie jak koarktacja aorty z różnymi formami hipoplazji łuku aorty, pierścienie naczyniowe i anomalie pozycyjne. Leczenie operacyjne i wybór optymalnej techniki korekcji wady u dziecka mogą wpływać na odtworzenie lub utrzymanie, lub wyłączenie odaortalnego dopływu krwi do naczyń tętniczych odchodzących od łuku aorty. Prawidłowe, najbliższe fizjologicznemu unaczynienie poszczególnych części ciała i narządów jest niezbędne do właściwego rozwoju i utrzymania funkcji. Konsekwencją zastosowania różnych strategii naprawy wad wrodzonych aorty i wykorzystania dostępnych technik operacyjnych mogą być zaburzenia perfuzji, także w zakresie naczyń zaopatrujących kończyny górne, najczęściej lewą kończynę górną.

Słowa kluczowe: wrodzone wady serca, wady naczyń łuku aorty, kardiochirurgia dziecięca, koarktacja aorty, hipoplazja łuku aorty, korekcja kardiochirurgiczna, ocena kończyn górnych, siła mięśniowa, EMG, AHA

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Ocena funkcjonalna kończyn górnych po operacjach naprawczych wad wrodzonych łuku aorty u dzieci w zależności od unaczynienia tętniczego

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Streszczenie

Wrodzonym wadom serca często towarzyszą wady wielkich naczyń, takie jak koarktacja aorty z różnymi formami hipoplazji łuku aorty, pierścienie naczyniowe i anomalie pozycyjne. Leczenie operacyjne i wybór optymalnej techniki korekcji wady u dziecka mogą wpływać na odtworzenie lub utrzymanie, lub wyłączenie odaortalnego dopływu krwi do naczyń tętniczych odchodzących od łuku aorty. Prawidłowe, najbliższe fizjologicznemu unaczynienie poszczególnych części ciała i narządów jest niezbędne do właściwego rozwoju i utrzymania funkcji. Konsekwencją zastosowania różnych strategii naprawy wad wrodzonych aorty i wykorzystania dostępnych technik operacyjnych mogą być zaburzenia perfuzji, także w zakresie naczyń zaopatrujących kończyny górne, najczęściej lewą kończynę górną.

Słowa kluczowe: wrodzone wady serca, wady naczyń łuku aorty, kardiochirurgia dziecięca, koarktacja aorty, hipoplazja łuku aorty, korekcja kardiochirurgiczna, ocena kończyn górnych, siła mięśniowa, EMG, AHA

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Wstęp

W niniejszej pracy podjęto próbę opracowania uniwersalnej strategii oceny rozwoju i funkcji kończyn górnych u dzieci. Składają się na nią kardiologiczne metody oceny układu krążenia oraz fizjoterapeutyczne testy czynnościowe kończyn górnych dostosowanych do wieku pacjentów. Potwierdzenie długofalowego znaczenia zachowania lub odtworzenia odaortalnego dopływu tętniczego na podstawie obiektywnych testów czynnościowych kończyn górnych może być dodatkowym argumentem przy wyborze optymalnej metody operacyjnej u najmłodszych dzieci.

Wrodzone wady serca i naczyń dotyczą także nieprawidłowości strukturalnych naczyń odchodzących od łuku aorty. Może to być powodem zaburzenia przepływu krwi i dawać objawy kliniczne w obszarze ich nietypowego przebiegu i unaczynienia, obejmując narządy śródpiersia, kończyn górnych i głowy. Leczenie operacyjne wrodzonych wad serca ma na celu skorygowanie wszystkich korygowalnych anomalii. Pozwala to na uzyskanie krążenia krwi w sposób najbardziej zbliżony do normy fizjologicznej, niezależnie od zakresu pierwotnego, wrodzonego uszkodzenia. W zakres korekcji wad wrodzonych wchodzą także zabiegi naprawcze tętnicy głównej i naczyń odchodzących od jej łuku, tak zwanych naczyń dogłowowych. Interwencja naczyniowa zazwyczaj zmienia ich orientację przestrzenną, położenie i zaopatrzenie w krew znajdujących się w obszarze naprawy naczyń. Może to mieć znaczenie dla dalszego funkcjonowania

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zarówno samych tętnic, jak też struktur i narządów przez nie unaczynianych.

Do najczęstszych wrodzonych wad z udziałem naczyń łuku aorty należą koarktacja aorty z różnymi formami hipoplazji łuku aorty, pierścienie naczyniowe oraz anomalie pozycyjne — położenia aorty i tętnicy płucnej w śródpiersiu. Wybór techniki operacyjnej w leczeniu tych wad może wpływać na utrzymanie lub odtworzenie odaortalnego dopływu krwi do naczyń tętniczych, co możliwe jest dzięki zmianie pozycji, przeznaczenia czynnościowego i orientacji przestrzennej. Może to wymagać wykonania dodatkowego zabiegu naprawczego.

Tradycyjne strategie operacyjne w korekcji wrodzonych anomalii serca i naczyń śródpiersia opracowywano w celu osiągnięcia zasadniczego, głównego celu, jakim jest poprawa tętniczego krążenia krwi w zakresie podstawowych struktur naczyniowych. Wymagające modyfikacji techniki operacyjnej metody uzupełniającej rekonstrukcji wszystkich naczyń z zachowaniem ich źródłowego dopływu oznaczają rozszerzenie zakresu zabiegu, z przedłużeniem czasu trwania operacji oraz rozleglejszą interwencją chirurgiczną w klatce piersiowej. Podjęcie decyzji o zmianie strategii operacyjnej jest dodatkowo utrudnione wczesnym — noworodkowym i niemowlęcym wiekiem pacjenta. Niska masa ciała i małe rozmiary dziecka mogą stanowić utrudnienie techniczne. Stoi to w sprzeczności z racjonalną przesłanką, że jednoczesna naprawa wszystkich możliwych do korekcji wad może mieć pozytywne znaczenie dla dalszego prawidłowego rozwoju dziecka.

Założenia

Prawidłowe unaczynienie tętnicze tkanki i narządu leży u podstaw prawidłowego rozwoju i utrzymania fizjologicznej funkcji. Zachowanie fizjologicznego unaczynienia obręczy barkowej i kończyn górnych u pacjentów po zabiegach korekcji wad serca i naczyń powinno zatem gwarantować prawidłowy rozwój tych obszarów z zachowaniem właściwej czynności.

Anatomia wady oraz sposób jej naprawy wywołują skutki hemodynamiczne, zarówno dla całkowitego krążenia krwi w ustroju, jak też przepływu krwi w naczyniach dogłowowych, bezpośrednio sąsiadujących z obszarem zwężonej cieśni i hipoplastycznego łuku aorty. Konsekwencją zastosowania strategii naprawy zwężenia cieśni aorty i wykorzystania konkretnej techniki operacyjnej, często z koniecznym wyłączeniem przepływu krwi, mogą być zaburzenia perfuzji, także w zakresie naczyń zaopatrujących kończyny górne, najczęściej lewą kończynę górną.

W efekcie operowanej w wieku dziecięcym, a często u noworodka, wady łuku aorty z ograniczeniem odaortalnego dopływu do lewej tętnicy podobojczykowej, odległym następstwem zabiegu może być ograniczenie sprawności funkcjonalnej lewej kończyny górnej.

Cel pracy

W celu oceny późnych następstw zachowania odaortalnego unaczynienia podjęto próbę opracowania uniwersalnej strategii oceny rozwoju i funkcji kończyn górnych u dzieci po operacjach wrodzonych wad serca i naczyń, z wykorzystaniem metod kardiologicznej oceny układu krążenia oraz fizjoterapeutycznych testów oceny czynnościowej kończyn górnych, dostosowanych do wieku i rozwoju badanych pacjentów.

Potwierdzenie długofalowego znaczenia zachowania lub odtworzenia odaortalnego dopływu tętniczego na podstawie obiektywnych, małoinwazyjnych testów czynnościowych kończyn górnych może być dodatkowym, istotnym argumentem w dyskusji o właściwym wyborze optymalnej strategii operacyjnej u najmłodszych dzieci.

Najczęstsze wady wrodzone łuku aorty i wielkich tętnic odchodzących od łuku aorty — z uwzględnieniem wyjściowej anatomii i patofizjologii

Koarktacja aorty z hipoplazją łuku aorty

Koarktacja aorty jest wrodzoną wadą serca definiowaną jako zwężenie aorty w okolicy cieśni [1]. Wada ta stanowi około 0,4% urodzonych noworodków i od 6 do 8% dzieci z wrodzonymi wadami serca [2]. Może występować jako anomalia izolowana lub bywa składową innych wad, co powoduje różnorodność objawów klinicznych i zagrożeń dla całego organizmu. U noworodka z krytycznym zwężeniem cieśni aorty tylko wczesne rozpoczęcie intensywnego leczenia zapewnia możliwość przeżycia. Wada zaliczana jest do tak zwanych przewodozależnych wad krytycznych.

W literaturze podstawowej opisane są dwa typy budowy anatomicznej aorty i łuku aorty, traktowane jako formy fizjologiczne. Typ I, najczęściej występujący wariant (74,0–89,4% populacji) składa się z trzech wielkich naczyń wychodzących z łuku aorty. Pierwszym naczyniem jest pień ramienno-głowowy, który dzieli się na tętnicę podobojczykową prawą i tętnicę szyjną wspólną prawą. Drugie naczynie to tętnica wspólna szyjna lewa, a ostatnią odgałęziającą się od łuku aorty strukturą jest tętnica podobojczykowa lewa. Wariantem II jest tak zwany *bovinetrunk* (sylwetka byka) i występuje u 7,2–21,1% populacji [3]. Niektórzy autorzy wyróżniają również dwa podtypy wariantu II: *longbovinetrunk*, czyli typ II-A, oraz *shortbovinetrunk*, czyli typ II-B [4]. Należy podkreślić, że oba warianty stanowią formy fizjologiczne z zachowanym prawidłowym przepływem i wszystkimi elementami prawidłowej anatomii naczyń.

We wrodzonej wadzie serca i naczyń nazwanej *bovinetrunk* pień ramienno-głowowy i lewa tętnica szyjna odchodzą jako wspólne naczynie w kontynuacji aorty wstępującej. Proksymalny łuk aorty nie wykształca się w tej anomalii.

Kontynuacją jest dystalny łuk aorty o relatywnie długim przebiegu sięgającym do okolicy tętnicy podobojczykowej lewej i cieśni aorty. W postaci noworodkowej wady przepływ obwodowy do narządów położonych poniżej łuku aorty zapewnia drożny przewód tętniczy.

Pierścienie naczyniowe z anomalią przebiegu tętnic podobojczykowych

Prawidłowe unaczynienie tętnicze kończyny górnej zapewnia tętnica podobojczykowa, która po odejściu od pnia ramienno-głowowego po stronie prawej lub dystalnego łuku aorty (po stronie lewej) rozgałęziając się, przechodzi w silną tętnicę pachową odpowiedzialną za zaopatrzenie mięśni kończyny górnej wraz z obręczą barkową [5]. Tętnica pachowa rozpoczyna się na brzegu zewnętrznym pierwszego żebra, a kończy się na wysokości brzegu dolnego mięśnia piersiowego większego. Tętnica pachowa wspierana jest przez pomniejsze liczne odnogi łączące się z gałęziami szyjnymi tętnicy podobojczykowej i tętnicami międzyżebrowymi.

Wada serca zwana pierścieniem naczyniowym zawiera wiele form anatomicznych. Z punktu widzenia unaczynienia kończyn górnych istotne są anomalie odejścia i przebiegu tętnic podobojczykowych, które odchodzą nieprawidłowo od łuku aorty po przeciwnej stronie od zaopatrywanej kończyny. Taka pozycja naczyń tętniczych powoduje objawy uciskowe narządów śródpiersia, w tym ważnych życiowo: tchawicy, oskrzeli i przełyku. W międzynarodowej nomenklaturze przyjęto pochodzące z łaciny określenie: *arteria lusoria*, odpowiednio prawa lub lewa, co oznacza: tętnica błądząca.

Impresja (ucisk) struktur naczyniowych na narządy śródpiersia

Pozycja łuku aorty z jego zwrotem określanym jako lewo- lub prawostronny, albo w pozycji pośredniej — określanej klinicznie jako pozycja „zero" — może powodować objawy uciskowe, dodatkowo nasilone przez podstawowe parametry hemodynamiczne aorty: wysokie ciśnienie krwi, wysoką falę tętna z wysoką objętością przepływu i sztywność jej ściany.

U pacjentów z niekorzystną pozycją i orientacją przestrzenną aorty w jej początkowym, śródpiersiowym odcinku występują objawy impresji podobne do typowych dla obecności pierścienia naczyniowego.

Mięśnie czynnościowe kończyn górnych odpowiedzialne za fizjologiczną funkcję kończyn

Kluczowymi mięśniami w funkcji kończyny górnej są: mięśnie zaliczane do stożka rotatorów, mięsień naramienny, mięśnie dwugłowy i trójgłowy ramienia, mięśnie grup zginaczy i prostowników nadgarstka i ręki [6]. Wyżej wspomniane mięśnie do prawidłowej pracy potrzebują też naczyń zaopatrywanych źródłowo od aorty o odpowiedniej średnicy i przepustowości. Adekwatne unaczynienie tętnicze wydaje się także naturalnym warunkiem fizjologicznego rozwoju kończyn, zgodnego ze wzrostem całego organizmu w okresie dorastania dziecka.

Kardiochirurgiczne metody naprawcze w leczeniu wad naczyń łuku aorty

Klasyczne metody naprawy zwężenia cieśni aorty z hipoplazją łuku aorty

Preferowaną obecnie strategią leczenia jest wczesna naprawa aorty w granicach własnych, zdrowych tkanek. Koarktacji aorty często towarzyszą różne formy niedorozwoju (hipoplazji) łuku aorty [7]. Klasyczne leczenie operacyjne polega na resekcji okolicy zwężenia z usunięciem nieprawidłowych tkanek, w niektórych technikach z odcięciem tętnic dogłowowych, głównie tętnicy podobojczykowej lewej. Wykonanie najbardziej popularnej operacji naprawczej noworodków i niemowląt z niską masą ciała utrudnia lub uniemożliwia zachowanie tętnicy podobojczykowej lewej, w której przepływ krwi zapewnia obecność krążenia obocznego. Źródłem krążenia obocznego dla odciętej tętnicy podobojczykowej są: tętnica piersiowa wewnętrzna, tętnica kręgowa — za pośrednictwem koła Willisa oraz naczynia okolicy obręczy barkowej. Pozbawienie odaortalnego unaczynienia dla tak istotnego naczynia, jakim jest zaopatrująca duże partie mięśniowe kończyny górnej tętnica podobojczykowa, usposabia do powstania objawów niedokrwienia oraz tak zwanych objawów podkradania, w związku z wstecznym przepływem krwi w naczyniach tętniczych. Najtrudniej kompensowanymi objawami podkradania są powikłania neurologiczne wynikające z hipoperfuzji OUN w zakresie koła tętniczego Willisa. Najczęstszymi wczesnymi objawami u pacjentów są bóle i zawroty głowy oraz niebezpieczne dla zdrowia i życia omdlenia.

Zmodyfikowana metoda resekcji koarktacji wraz ze zwężeniem cieśni i proksymalnego zespolenia metodą: koniec-do-boku, z odtworzeniem dopływu do tętnicy podobojczykowej lewej z wykorzystaniem hipoplastycznego dystalnego odcinka łuku aorty

Naprawa anomalii typu *bovinetrunk* u noworodka jest możliwa z typowego dostępu bocznego. W początkowym etapie operacji rekonstruuje się dopływ do lewej tętnicy podobojczykowej z wykorzystaniem dystalnego odcinka hipoplastycznego łuku aorty, który płynnie przechodzi w tętnicę podobojczykową lewą. Dzięki utrzymaniu wlewu prostaglandyny i utrzymaniu drożności przewodu tętniczego, mózg i obie kończyny górne zaopatrywane są odsercowo, podczas gdy dolna część ciała perfundowana jest odprzewodowo. Następnie po podwiązaniu przewodu tętniczego zamyka

się klemem naczyniowym aortę zstępującą i rozlegle resekuje zwężoną cieśń wraz z tkanką okołoprzewodową, po czym — po bocznym nacięciu proksymalnej części łuku aorty u nasady wspólnego pnia ramienno-głowowego i wysokim przemieszczeniu aorty zstępującej — wykonuje się zespolenie metodą koniec-do-boku. Dodatkowym utrudnieniem może być wariant anatomiczny z odejściem tętnicy podobojczykowej w okolicy zwężonej cieśni lub wręcz poniżej, od tkanki okołoprzewodowej, co wymaga niezależnego przeszczepienia tętnicy podobojczykowej lewej do pnia lewej tętnicy szyjnej wspólnej, z zespoleniem metodą koniec-do-boku.

Po wykonaniu tej operacji z zespoleniem koniec-do-boku przepływ do aorty zstępującej odbywa się z pominięciem odcinka łuku, którego hipoplastyczną strukturę wykorzystuje się jedynie do zachowania ciągłości i zapewnienia odaortalnego dopływu do tętnicy podobojczykowej lewej. Podstawową zaletą tej techniki jest możliwość jej wykonania bez zastosowania krążenia pozaustrojowego, z dostępu bocznego, w granicach i z wykorzystaniem zdrowych, własnych tkanek pacjenta, z zachowaniem odaortalnego dopływu do wszystkich tętnic.

Ocena kliniczna pacjenta po operacji naczyń łuku aorty

Diagnostyka i metody obrazowania

Najważniejszym zagadnieniem diagnostycznym jest wyjściowa anatomia wad naczyniowych. U pacjentów z koarktacją ocenia się nasilenie zwężenia cieśni aorty, hipoplazji łuku aorty i rozwój oraz orientację naczyń odchodzących od łuku aorty. Dzieci z rozpoznaniem pierścienia naczyniowego wymagają identyfikacji łuku aorty oraz szczegółowej oceny zarówno punktu początkowego odejścia, jak też dalszego przebiegu naczyń dogłowowych. U pacjentów z objawami impresji wielkich naczyń szczegółowego obrazowania wymaga pozycja łuku aorty z jego zwrotem, określanym jako lewo-, prawostronny lub w pozycji pośredniej, określanej klinicznie jako pozycja „zero". Dodatkowo ocenia się stan narządów w obszarze ucisku, z analizą stopnia i trwałości lub nieodwracalności ich uszkodzenia. Badania obrazowe powtarzane są wielokrotnie w okresie pooperacyjnym, stanowią one także podstawę oceny klinicznej pacjenta w odległej obserwacji.

Echokardiografia

Dwuwymiarowa echokardiografia (ECHO) zapewnia doskonałe obrazowanie u najmłodszych dzieci, noworodków i niemowląt. Jest obecnie standardowym badaniem wykonywanym u noworodków z wadami serca i naczyń. Bardzo często wystarcza do właściwej kwalifikacji do leczenia operacyjnego. Jest też podstawowym badaniem kontrolnym pooperacyjnym.

Angiografia

Tradycyjna angiografia (ANGIO) jest wykorzystywana w przypadku wątpliwości echokardiograficznych lub u pacjentów z niejasną anatomią łuku aorty, podejrzeniem wad dodatkowych i starszych pacjentów. Badanie angiograficzne pozwala na precyzyjną ocenę większości naczyń tętniczych i przepływów krwi, wymaga jednak podawania środków kontrastowych.

Rezonans magnetyczny

Łuk aorty i proksymalny odcinek aorty zstępującej są dobrze obrazowanymi strukturami w rezonansie magnetycznym (MRI). Metoda ta zapewnia lepsze obrazy dwuwymiarowe i rekonstrukcje przestrzenne niż jakakolwiek inna technologia nieinwazyjna. MRI umożliwia także szczegółowe obrazowanie układu naczyń w stosunku do innych struktur śródpiersia. W badaniu tym możliwa jest także ocena stanu tkanek i narządów sąsiadujących z naczyniami.

Tomografia komputerowa

Zasadniczą zaletą tomografii komputerowej (angio-CT) jest precyzyjne obrazowanie u pacjentów po implantacji metalowych stentów z wysokim ryzykiem wytwarzania tętniaków w okresie pooperacyjnym i przeciwwskazaniami do wykorzystywania metody MRI [8].

Metody oceny funkcjonalnej kończyn górnych u pacjenta w odległym okresie po operacji naczyń łuku aorty

Przed przystąpieniem do oceny antropometrycznej, biomechanicznej i funkcjonalnej kończyn górnych powinno się określić dominację czynnościową ręki. Informacja o prawo- lub leworęczności jest niezbędna do przeprowadzenia analizy porównawczej kończyn pod względem ich sprawności czy siły, zwłaszcza u dzieci starszych. Rozpatrywanie uzyskanych wyników badań wyłącznie przez pryzmat prawej i lewej strony ciała może zaburzać proces wnioskowania oraz być nieużyteczne dla klinicystów i pacjentów, zwłaszcza że odsetek osób leworęcznych wynosi około 10% populacji [9]. Jednym z narzędzi pozwalających określić dominację czynnościową kończyny górnej u dziecka jest *Peg Moving Task* (PMT-5). Badanie polega na jak najszybszym umieszczeniu pięciu patyczków w otworach drewnianej deseczki. Ręka, która wykonała zadanie w krótszym czasie, uznawana jest za dominującą [10]. U dzieci starszych za kończynę dominującą uznaje się tą, której ręką dziecko pisze, używa łyżki, szczoteczki do zębów czy nożyczek [11].

Badanie długości i obwodów kończyny górnej

Zakłócenie głównego przepływu tętniczego do kończyny górnej u dorosłych zwykle skutkuje ustanowieniem krążenia obocznego zdolnego do utrzymania życia i funkcji kończyny.

U dzieci podobna dysfunkcja może negatywnie wpływać na odżywienie tkanek kończyny, a przez to zaburzać ich wzrost i funkcję [12]. Poza wykonaniem pomiarów długości względnej i bezwzględnej kończyn górnych, użyteczne jest uwzględnienie pomiarów odcinkowych ramienia i przedramienia, w celu dokładnego określenia lokalizacji zaburzenia wzrostu kości długich. Przeprowadzenie pomiarów obwodów zwłaszcza ramiennego pierwszego (R1) krótkiego i długiego, ramiennego drugiego (R2) oraz przedramiennego pierwszego (P1) mogą dostarczyć informacji o występującej hipotrofii tkanek miękkich.

Badanie siły chwytu ręki

Elektroniczno-hydrauliczny dynamometr ręczny (HGS, *Hand Grip Strenght*) jest uznawany przez American Society of Hand Therapist za obiektywne narzędzie monitorujące siłę chwytu ręki u pacjentów po zabiegach kardiochirurgicznych [13]. Klasyczny protokół zakłada pomiar siły izometrycznej w pozycji stojącej [14], natomiast ze względu na specyfikę testu oraz możliwość nagłego wzrostu ciśnienia krwi, u pacjentów kardiochirurgicznych zaleca się pozycję siedzącą z ramieniem lekko odwiedzionym od tułowia i kończyną zgiętą w stawie łokciowym do 90° (wg protokołu JAMAR) [15]. Maksymalny ścisk ręki musi zostać utrzymany przez minimum 3 sekundy [16]. Urządzenie jest ustandaryzowane i może być stosowane u pacjentów powyżej 4. roku życia [17].

Badanie siły mięśniowej

Dynamometr Biodex System PRO4 umożliwia precyzyjny pomiar momentów sił mięśni w warunkach izometrycznych (statycznych), izokinetycznych (dynamicznych) i izotonicznych u osób zdrowych, a także w procesie rehabilitacji. Urządzenie może zostać wykorzystane do obiektywnego pomiaru siły dla poszczególnych ruchów kończyny w stawie ramiennym [18] oraz w stawie łokciowym [19] w pełnych zakresach ruchu pacjenta [20]. Ponadto istnieje możliwość określenia relacji między siłą mięśni antagonistycznych. Ma to na celu określenie proporcji siły mięśni w obrębie danego stawu [21, 22]. Pomiar momentów sił mięśniowych obu kończyn górnych pozwala na ich bezpośrednie porównanie i określenie symetrii siły [21].

U pacjentów po zabiegach kardiochirurgicznych nierekomendowane jest przeprowadzanie pomiarów w warunkach izometrycznych, ze względu na duże obciążenie organizmu pracą statyczną. W procesie rehabilitacji często prowadzi się pomiary w warunkach izokinetycznych [22, 23], w których ruchy wykonywane są ze stałą, zaprogramowaną przez badacza, wartością prędkości kątowej w stawie, a generowany opór dopasowuje się indywidualnie do możliwości pacjenta.

Dane wykorzystywane do analizy to maksymalny moment siły osiągany przy danej prędkości kątowej (wartości absolutne i względne, tj. odniesione do masy ciała badanego), praca całkowita, maksymalna i średnia moc i wskaźnik zmęczenia. Na wartość momentu siły mogą mieć wpływ czynniki, takie jak wiek, płeć, BMI (*Body Mass Index*, wskaźnik masy ciała) czy dominacja kończyny [24]. Istnieje możliwość adaptacji ramion dynamometru dla dzieci od 6. roku życia [25].

Badanie aktywności elektrycznej mięśni

Elektromiografia zajmuje się badaniem zmian aktywności elektrycznej mięśni w poszczególnych okresach [26]. Elektromiografia z użyciem elektrod powierzchniowych (sEMG) jest użyteczna w uzupełnianiu diagnostyki chorób nerwowo-mięśniowych u dzieci [27]. Ze względu na mogące się pojawiać pooperacyjne powikłania neurologiczne u pacjentów kardiochirurgicznych, przeprowadzenie nieinwazyjnego badania z wykorzystaniem sEMG (*surface electromyography*) podczas wykonywania wybranych zadań ruchowych, może dostarczyć informacji o występującym zaburzeniu i różnicach we wzorcach aktywacji mięśni [26]. Powszechnie stosowanymi zaleceniami do umieszczania elektrod na skórze są wytyczne SENIAM-u (*surface EMG for non-invasive assessment of muscles*) [28].

W celu porównania ze sobą osób badanych oraz ewentualnego monitowania procesu rehabilitacji, niezbędna jest normalizacja amplitudy sygnału EMG, czyli porównania wielkości zmierzonej z wartością referencyjną, którą pozyskuje się w zależności od wybranej metody. Technika MVIC (*maximum voluntary isometric contraction*) zakłada wykonanie maksymalnego skurczu izometrycznego [26]. Należałoby rozważyć zasadność stosowania tej techniki normalizacji w przypadku pacjentów kardiochirurgicznych, u których mogłoby to doprowadzić do pogorszenia stanu zdrowia. Innymi sposobami normalizacji jest zarówno pozyskanie wartości referencyjnej z badania, biorąc maksymalną wartość amplitudy jako poziom 100% lub wartość średnią, jak i wykonanie pomiaru wartości referencyjnej dla warunków stałego znanego, niemaksymalnego, obciążenia grupy mięśniowej w warunkach statycznych [26].

Dane poddawane analizie to najczęściej średnia amplituda sygnału, pole powierzchni pod obwiednią sygnału, częstotliwość średnia i środkowa widma sygnału EMG oraz analiza charakterystyki czasowej pracy mięśnia.

Ze względu na dość dużą łatwość przeprowadzenia badań, elektromiografia stała się powszechnym narzędziem do badania potencjału czynnościowego mięśni, nie powinna być ona jednak utożsamiana z pomiarem siły czy mocy mięśniowej [26].

Badanie termowizyjne

Prawidłowe funkcjonowanie układu krążenia jest warunkiem do odpowiedniego rozwoju kończyny i zachowania jej pełnej funkcjonalności. Układ krwionośny odpowiedzialny jest między innymi za utrzymanie odpowiedniej ciepłoty tkanek, dlatego termowizja może być pomocna

w lokalizowaniu zaburzeń ukrwienia kończyn górnych. Mogą się one objawiać obniżeniem temperatury struktur obwodowych. Badania termowizyjne ze względu na małą inwazyjność i łatwość wykonania są stosowane do oceny temperatury ciała zarówno dorosłych, jak i dzieci [29]. Mogą być one wyłącznie uzupełnieniem badań podstawowych, oceniających przepływ krwi w naczyniach kończyn górnych. W dostępnym piśmiennictwie nie odnaleziono jednoznacznych wskazań do stosowania termowizji i protokołów postępowania diagnostycznego.

Badanie motoryki małej

Ocena stanu funkcjonalnego kończyny górnej powinna opierać się na badaniu motoryki małej. Powszechnie stosowanym narzędziem jest ocena ręki wspomagającej (AHA, *Assisting Hand Assessment*). Za pomocą AHA mierzy się i opisuje, w jaki sposób dzieci z jednostronną niepełnosprawnością wykorzystują rękę objętą zaburzeniem do wykonywania zadań/zabaw wymagających użycia obu rąk. Składa się ona z 22 części podlegających obserwacji, które w zależności od jakości wykonania są oceniane od 1 do 4 punktów [30]. AHA została stworzona dla dzieci w wieku od 18 miesięcy do 12 lat i umożliwia monitorowanie procesu rehabilitacji [31]. Dla dzieci w wieku od 18 miesięcy do 5 lat badanie polega na obserwacji spontanicznej zabawy, zabawkami wymagającymi użycia dwóch rąk (*Small Kids AHA*). Dla dzieci w wieku 6–12 lat opracowano grę planszową (*School Kids AHA*), która zapewnia odpowiedni dla wieku kontekst do obsługi tych samych zabawek, co w przypadku AHA dla małych dzieci [32]. Jest ona uznawana za użyteczne narzędzie kliniczne i badawcze [33]. W 2017 roku opracowano pierwsze narzędzie oceniające funkcję ręki dzieci w wieku 3–12 miesięcy (HAI, *Hand Assessment for Infants*). Narzędzie to składa się z 31 części ocenianych w skali od 0 do 2 punktów w zależności od jakości prezentowanych umiejętności ruchowych niemowlęcia [34]. Zarówno AHA, jak i HAI są stosowane podczas prowadzenia diagnostyki dzieci z zaburzenia neurologicznymi, szczególnie z mózgowym porażeniem dziecięcym [30, 34].

Ocena rozwoju psychomotorycznego dzieci po operacjach naczyń łuku aorty

Poza powyższymi instrumentami, które są ściśle dedykowane do oceny funkcji kończyn górnych, istnieją różne narzędzia służące do kompleksowej ewaluacji stanu rozwoju psychomotorycznego dziecka, których poszczególne moduły obejmują również obszary motoryki małej. American Heart Association opublikowało w roku 2012 oświadczenie, w którym rekomenduje stałe monitorowanie rozwoju dzieci i młodzieży z wrodzonymi wadami serca za pomocą wystandaryzowanych narzędzi oceny. W celu określenia poziomu rozwoju motoryki małej oraz funkcji ręki stowarzyszenie to zaleca wykorzystanie następujących metod i testów: *The Bayley Scales of Infant and Toddler Development-Third Edition, Bruininks-Oseretsky Test of Motor Proficiency — Second Edition* (BOT-2), *Grooved Peg Board, NEPSY-II, Peabody Developmental Gross Motor Scale* (PDMS-II), *Scales of Independent Behavior — Revised* (SIB-R), *Vineland Adaptive Behavior Scales — third Edition* (Vineland-3), *Test of Visual-Motor Integration, Supplemental-Motor Coordination II* (VMI) [35].

Mianem „złotego standardu" w ocenie niemowląt i dzieci z zaburzeniami rozwoju, a także w monitorowaniu skuteczności podjętych działań terapeutycznych określa się skalę *Bayley Scales of Infant and Toddler Development* (Bayley-III). Skala Bayley-III jest to wystandaryzowane narzędzie służące do oceny rozwoju dziecka od 1. do 42. miesiąca życia. Bayley-III zawiera podskalę oceniającą rozwój motoryki małej, czyli ruchów precyzyjnych dłoni. Podskala precyzyjnej motoryki wchodzi w skład całościowej oceny rozwoju dziecka, ale może być traktowana również jako niezależna część testu. Ocena motoryki małej polega na badaniu 66 cech i mierzy takie zdolności, jak jakość chwytu, sprawność manualna, manipulowanie przedmiotami, koordynacja wzrokowo-ruchowa oraz dynamika ruchu. Uzyskiwane w badaniu rezultaty punktowe porównywane są z przyjętymi normami [36].

Powyższe narzędzia ewaluacyjne są również przydatne w celu kompleksowej oceny deficytów ruchowych kończyn górnych, które mogą występować po zabiegach kardiochirurgicznych u najmłodszych pacjentów.

Wnioski

Ocena antropometryczna, biomechaniczna i funkcjonalna kończyn górnych u dzieci w odległej obserwacji po operacjach wad naczyń łuku aorty pozwala na dodatkową ewaluację skuteczności i ewentualnych skutków ubocznych poszczególnych metod operacyjnych.

Monitorowanie rozwoju i sprawności kończyn górnych u dzieci leczonych z powodu najczęstszych wrodzonych wad serca i naczyń, z konsekwencjami w postaci zaburzeń ukrwienia powinny opierać się na podstawowej ocenie hemodynamicznej, a także powtarzalnych, nieinwazyjnych narzędziach diagnostycznych i testach sprawdzających zmiany strukturalne w zakresie kończyn górnych, jak i badaniach funkcjonalnych, w tym przede wszystkim sprawdzających motorykę małą.

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The essence and character of an expert opinion as evidence in Polish civil trial

Istota oraz charakter dowodu z opinii biegłego w polskich procesach cywilnych

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Abstract

In the Polish civil litigation procedure, the cases concerning medical errors always require an expert opinion for the court to assess doctor's due diligence. The court does not have specialist knowledge, thus their decision, if not taken based on a specialist opinion, is procedurally incorrect. Expert's opinion can be effectively questioned by each of the parties of the dispute, which may lead to either complementary or further opinion. Every specialist opinion of an expert in civil law disputes concerning medical errors has to meet the requirements of integrity, logic and be comprehensive enough.

Key words: medical error, civil trial, expert's opinion

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Following Article 278 [1] of the Polish Code of Civil Procedure (hereinafter the 'CCP'), in cases requiring specialist knowledge, the court — having heard the parties' motions as to the number and selection of expert witnesses — can appoint one or more experts to seek their opinion. If the case cannot be resolved without specialist knowledge, expert opinion evidence is a necessity [1]. Hence, if the case requires an expert opinion, such evidence cannot be substituted with any other evidence [2].

An expert opinion, just as any other evidence, shall be subject to free evaluation by the court, both concerning formal requirements and the evidentiary value. If an expert opinion fails to provide a professionally grounded rationale in support of its conclusions, this will prevent the appropriate evaluation of its evidentiary value, with the result being that any judgment based on such an opinion would exceed the limits of free evaluation of evidence [3]. The subject-matter of an expert opinion is not the presentation of the facts but their evaluation based on expert knowledge (specialist knowledge) and, therefore, an expert opinion is not subject to the same true-or-false verification as a piece of evidence used to establish the facts [4].

In a civil case, unlike a criminal trial, the lawmakers have not specified the constituent elements of an opinion allowing it to be accepted as correct; hence, the only statutory requirement is for the opinion to contain a rationale [5]. In principle, however, the court, when analysing such evidence in the case, cannot base their conviction concerning the existence or non-existence of circumstances the examination of which require specialist knowledge solely on the basis of the conclusions of the expert's opinion.

The body of court decisions points out that the court ought to verify that the various elements contributing to the accuracy of the conclusions are correct, emphasizing the established criteria for the evaluation of such type of evidence in civil proceedings — the principles of logic and the level of the expert's knowledge, uniformity and universality of the method used, the certainty of scientific research results, professionalism, reliability, sound logic, exhaustiveness and completeness of the opinion, and its firmness [6].

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In matters concerning medical errors, it is not the court's role to evaluate the expert's opinion for consistency with medical records and treatment provided, but to evaluate the opinion based on the specified criteria [7]. The opinion should also be exhaustive, for it serves as evidence to evaluate factual circumstances from the perspective of the expert's specialist knowledge [8]. On the other hand, the role of an expert is not to make independent findings of fact relevant to the application of a specific legal norm but only to cast light on the circumstances being explained from the perspective of specialist knowledge, having regard to the material gathered at trial [9].

The purpose of the expert's opinion as a piece of evidence is to enable the court to make a proper evaluation of the evidence material gathered in the case, without, however, being in itself capable of serving as a source of the factual material in the case, let alone as a basis for determining the existence of the facts being the subject matter of the evaluation itself [10]. In the evaluation of an expert opinion in the context of its utility in arriving at the judgment, it is of significant importance whether the opinion gives a reliable and unambiguous answer to the questions posed by the court [11]. For it is of the essence that the conclusions of the expert's opinion be firm and unequivocal [12]. The expert's task is not to determine the facts of the case [13]. The expert's duties and powers do not include resolving points of law. The application and interpretation of legal provisions belong to the court, not to the expert [14].

The expert's opinion is subject to evaluation by the adjudicating court both as to its exhaustiveness and consistency with formal requirements and as to its persuasive value [15]; the court, however, is not under a duty to strive to reach the situation where the parties to the proceedings are persuaded by the expert's opinion — it is enough that the court has been persuaded by the expert opinion [16]. The methodology of accepting evidence derived from an expert opinion is such that it should not be limited to filing the opinion with the case record; the expert should be summoned to the hearing so that the parties could pose their questions [17]. If a party raises objections as to the expert's written opinion and consequently moves to summon the expert to a hearing so that they give oral explanations as to the objections raised, then failure to grant such a motion is a procedural error justifying an appeal [18].

If needed, the court is obliged to admit evidence from additional experts or the opinion of an institute, which happens when the opinion initially admitted contains significant gaps, is incomplete so that it does not respond to the challenges posed by the evidence, or when it is unclear, i.e. not properly reasoned, or unverifiable [19]. If the expert has responded to the objections to the opinion, then there is no need to admit evidence from an opinion of further experts, since the need to appoint an additional expert should arise from the circumstances of the case and not from the party's dissatisfaction alone [20].

The existence of a procedural need in the specific case determines the necessity of requesting an additional opinion from the same or other experts [21]. From Article 286 CCP it follows that the court may demand an oral explanation of an opinion submitted in writing, and it may demand an additional opinion from the same or other experts if needed [22].

The Court must admit evidence stemming from an additional expert opinion if it has been found that the expert opinions already available to the court are not sufficient to explain the case [23]. The Court, however, does not enjoy arbitrary freedom to appoint additional experts, and it is up to the parties to demonstrate circumstances justifying the appointment of another expert [24]. A party to the trial, when demanding the opinion of a different expert, must show that the prior opinion is incomplete, unclear or contains an inconsistency. As noted, a party dissatisfied with the expert's opinion cannot insist on demanding additional experts until one of them submits an opinion 'demonstrating' what the party 'intends to prove' [25].

If two opinions are issued in the case by experts in the same speciality field, then the need to appoint another expert arises when the existing expert opinions come with equally persuasive reasoning but differ only in the expert conclusions arising from such opinions [26]. Hence, the courts note that, as a consequence of the principle of adversarial trial, the party should show the necessary activity and demonstrate such errors, inconsistencies or other defects in expert opinions as may disqualify such an opinion or justify the appointment of additional experts; however, the decision whether to appoint new experts, belongs to the court and depends on the merits of the evidentiary motions [27].

The opinion of a scientific or research institute is not a separate type of evidentiary measure, but a type of evidence-based on an expert opinion, used in complicated cases requiring broader consultation [28]. The institute's opinion is not 'super evidence' binding on the court adjudicating the case; it is subject to the court's evaluation the same as all other evidence [29]. However, such an opinion, being the collective work of a scientific institute, enjoys that institute's scientific authority and therefore a higher rank than the evidence derived from an individual expert opinion [30]. The institute's opinion extends only to views uniformly or predominately represented in it; hence, the personal views of one of the co-authors cannot form the basis for the court's findings [31]. Admitting evidence from the opinion of a scientific or research institute will be expedient and justified when the problem the court has to evaluate requires, due to its complexity, explanation by

specialists with a particularly high degree of theoretical preparation and when it is necessary to include the findings of the most recent research, and when contradictions in opinions already available cannot be eliminated otherwise [32].

The institute's opinion given at the court's request should be adopted collectively, after jointly conducting the research, and should reflect the position not of any individual persons but the institute as a body [33]. The opinion should list not only the full names of those who conducted the research and issued the opinion but also their academic degrees and professional positions, specifying the field in which they specialize [34]. Irrespective of the knowledge and experience of those selected by the institute to issue the opinion, they can consult within a broader circle of specialists at the relevant unit, which guarantees a more thorough examination of the case [35]. In principle, the private opinion of a court expert or institute does not constitute the evidence of special knowledge. In a civil trial, such type of evidence is regarded as evidence stemming from a private document and is subject to evaluation within the perception framework of the party relying on it. Evidence from an expert opinion admitted by the court and meeting the criteria developed based on the body of court decisions and output of legal scholars, or evaluation of such type of evidence from the perspective of its utility for resolving the case is of significantly greater evidentiary value.

Unquestionably, in civil proceedings evidence from an expert opinion cannot be substituted either by witness testimony or documentary evidence, for such types of evidence refer primarily to factual circumstances, while their evaluation from the perspective of specialist knowledge is possible only based on the evidence stemming from the opinion of an expert, whose role in the civil trial thus remains invariably significant [36].

Streszczenie

W polskim postępowaniu cywilnym sprawy dotyczące błędów medycznych zawsze wymagają opinii biegłego, aby sąd mógł ocenić należytą staranność lekarzy. Sąd nie posiada specjalistycznej wiedzy, dlatego jego orzeczenie, o ile nie zostało podjęte na podstawie opinii specjalisty, o tyle jest błędne proceduralnie. Opinia biegłego może być skutecznie zakwestionowana przez każdą ze stron sporu, co może prowadzić do opinii uzupełniającej lub dalszej. Każda specjalistyczna opinia eksperta w sporach cywilnoprawnych dotyczących błędów medycznych musi spełniać wymogi rzetelności, logiki i być dostatecznie wyczerpująca.

Słowa kluczowa: błąd medyczny, postępowanie cywilne, opinia biegłego

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