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Association between selected risk factors and the incidence of venous obstruction after pacemaker implantation: demographic and clinical factors

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Abstract

Background: Venous obstruction and subsequent pulmonary embolism belong to the most common and dangerous complications of pacemaker implantation. Thus, identification of patients at risk of venous obstruction seems to be of critical importance.

Aim: To determine risk factors of venous obstruction following pacemaker implantation.

Methods: Eighty one patients with permanent cardiac pacing (31 F, 50 M; mean age 71.1 ± 7.6 years) were included. Prior to pacemaker implantation, the following factors were evaluated in each patient: indications for pacemaker implantation, heart failure severity assessed using the NYHA classification, coexisting diseases, a history of tobacco smoking, medications used before the procedure (antiplatelet drugs, anticoagulants, antibiotics), a history of thrombotic or infectious complications, and previous temporary cardiac pacing. Type of venous access and procedure time were also assessed. Venous ultrasound examination to evaluate veins in both upper extremities, shoulder areas and the neck was performed before pacemaker implantation and 6 and 12 months following the procedure. Computed tomography and conventional digital subtraction angiography were performed to confirm the diagnosis of venous obstruction.

Results: The patients were divided into two groups based on the occurrence of venous obstruction after pacemaker implantation. Group I (n = 71, 29 F, 42 M; mean age 71.0 ± 7.7 years) included patients without venous obstruction, and group II (n = 10, 2 F, 8 M; mean age 71.6 ± 7.0 years) included patients diagnosed with venous obstruction. Each patient was followed for 19 months. In group II (12.3% of the study population), venous obstruction developed mean 13 months after pacemaker implantation. In this group, symptomatic venous obstruction was observed in 3 patients (3.7% of the study population), mean 15 months after pacemaker implantation. Risk factors for venous obstruction included a history of myocardial infarction, temporary cardiac pacing, arrhythmia, venous anomalies, NYHA class III and IV heart failure, a history of infection, and tobacco smoking. Depending on the number of risk factors, the probability of development of venous obstruction was described by the following equation: $e^{-14.6 + 3.19x}/1 + e^{-14.6 + 3.19x}$, where x is the number of risk factors. In patients who had more than 6 risk factors, almost a 100% probability of the occurrence of venous obstruction was observed.

Conclusions: 1. Risk factors for venous obstruction include a history of myocardial infarction, temporary cardiac pacing, arrhythmia, venous anomalies, infections, NYHA class III and IV heart failure, and tobacco smoking. 2. In patients who had more than 6 risk factors, almost a 100% risk of venous obstruction was observed.

Key words: permanent cardiac pacing, venous obstruction, risk factors for venous obstruction

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INTRODUCTION

One of the most common and most dangerous postoperative complications of cardiac pacemaker implantation is venous obstruction associated with the inserted lead. Venous obstruction may result from all conditions that might interrupt blood flow in a vessel, mainly thrombosis, and the latter is associated with pulmonary embolism [1]. The pathogenesis of venous obstruction is related to the presence of a foreign body, i.e., the pacemaker lead in the blood vessel which may result in endothelial damage and perturbations in laminar blood flow, leading to activations of factors included in the Virchow's triad and subsequent thrombosis and/or fibrosis [2, 3].

The incidence of upper limb venous thromboembolic disease in patients with cardiac pacemaker was reported at 5.5% to 44% [4–18]. Due to the route used for transvenous lead placement, the thrombus is most frequently located in the left subclavian vein, usually in its proximal portion. Thrombotic complications were also reported, however, in other veins of the upper body, including axillary, jugular, brachiocephalic veins, and also in the superior vena cava [6, 8, 10, 11, 16, 18]. Of note, most episodes of venous thrombosis are asymptomatic [1, 2, 4–6, 9, 14, 15, 17, 18].

It was found that early thrombosis often propagates due to local relative hypercoagulability related to the presence of the implanted lead [17]. These patients are at risk of massive venous thrombosis, including superior vena cava syndrome, which may result in life-threatening pulmonary embolism, and subsequently in the development of postthrombotic syndrome [1]. Studies evaluating venous obstruction as a complication of pacemaker implantation gave inconclusive results. Thus, identification of patients at risk of venous obstruction seems to be important and warrants further research regarding this complication.

In the present study, we attempted to identify risk factors for venous obstruction and evaluate their effects on the the development of this complication in patients undergoing pacemaker implantation.

METHODS

From January to April 2009, we studied 81 consecutive patients (31 women, 50 men, mean age 71.1 ± 7.6 years) scheduled for cardiac pacemaker implantation (including single- and dual-chamber devices). During this period, overall 224 patients underwent pacemaker implantation in our centre. The study population did not include patients who did not give consent for the study (n = 15), were found to have stenoses in the veins of upper extremities, shoulder areas and neck before pacemaker implantation (n = 4), or could not undergo appropriate imaging studies (n = 25), patients with cardiac valvular prosthesis (n = 15) or with a history of coronary artery bypass grafting (n = 10) or coronary stenting (n = 12), and patients scheduled for pacemaker replacement or change of the pacing mode (n = 9). We have also excluded patients with incomplete data (n = 40) and those in whom the results of imaging studies were difficult to interpret (n = 13).

All patients had leads implanted by a venesection of the left cephalic vein or a branch of the left subclavian vein, or by the left subclavian vein puncture. The type and design of intracardiac leads did not differ between patients. The study had a prospective design. Prior to pacemaker implantation, the following factors were evaluated in each patient: indications for pacemaker implantation (type of arrhythmia or conduction disturbances), patient clinical status based on the New York Heart Association (NYHA) functional classification to evaluate heart failure (HF) severity, coexisting diseases, a history of tobacco smoking, medications used before the procedure (antiplatelet drugs, anticoagulants, antibiotics), a history of thrombotic or infectious complications, and previous temporary cardiac pacing. During pacemaker implantation procedure, venous access (use of one or two veins, presence of venous anomalies) and procedure time (including implantation of both the generator and the leads) were assessed.

Venous ultrasound examination (using the Vivid 7 Expert system with a 5–13 MHz linear probe) to evaluate veins within the shoulder areas and the neck was performed in the supine position, and the examination to evaluate veins of the upper extremities also in the sitting position. Venous morphology and blood flow were evaluated in real-time by two-dimensional and duplex Doppler (using pulsed wave imaging) ultrasonography.

In addition, colour Doppler was used to evaluate vessel patency and blood flow. Vessel walls and the lumen of the jugular veins and upper extremity veins were assessed in transverse and longitudinal views. Accessible parts of subclavian and brachiocephalic veins in longitudinal views were also imaged. Jugular or upper extremity vein obstruction was examined by compression ultrasonography, as well as by the presence of abnormal intraluminal echoes, increased vessel diameter, and the absence of blood flow in spectral and colour Doppler [17, 19].

Venous ultrasonography was performed before and after pacemaker implantation, at 6 and 12 months following the procedure, and also with the development of symptoms and signs of venous obstruction. Computed tomography angiography (CTA; Siemens Definition, Erlangen, Germany) using three-dimensional volume reconstruction and conventional digital subtraction angiography (DSA) was performed to confirm or detect venous obstruction.

Symptomatic venous obstruction was defined as the presence of symptoms and signs (limb pain, oedema worsened by limb movement, cyanosis, and the appearance of superficial venous collateral circulation network) associated with the evidence of venous obstruction (vessel lumen diameter reduction by at least 50%) in imaging studies (ultrasonography, CTA, DSA). Asymptomatic venous obstruction was diagno-

sed when vessel lumen diameter reduction by at least 50% in imaging studies was not accompanied by the presence of symptoms and signs of venous obstruction [15, 20].

Statistical analysis

The analysed risk factors are reported as distributions (in absolute numbers) and percentages (in %) in the two study groups using appropriate codes (yes/no for the presence/absence of a given risk factor). Differences in risk factors between patients with or without venous obstruction were compared using the χ^2 test or the exact Fisher test with the alpha value of 0.05. Selected risk factors were additionally evaluated with the percentages test by comparing the percentage contribution of these factors in the study groups. In addition, the power of the test (1-β) was calculated for the alpha value of 0.05. Significance was set at $\alpha = 0.05$ and 1-$\beta = 0.80$ [21].

All risk factors present in a given patient were attributed a weight of 1, with weights of all factors added in individual patients to give the overall number of risk factors in a given patients, ranging from 0 (no risk factors present) to 7 (all risk factors present). Logistic regression analysis was used to describe the effects of the number of risk factors on the incidence of complication. We calculated β_0 and β values, the odds ratio (OR) and the respective 95% confidence interval. Using the receiver-operating characteristic (ROC) curves, we determined the critical duration of the procedure, including implantation of both the generator and the lead(s).

Calculations were performed using the STATISTICA version 9.0 software (StatSoft Poland 2010). All patients gave written informed consent and the study was accepted by the Bioethics Committee at the Jagiellonian University (approval No. KBET/63/B/2009).

RESULTS

The patients were divided into two groups based on the occurrence of venous obstruction after pacemaker implantation. Group I (n = 71, 29 women, 42 men; mean age 71.0 ± ± 7.7 years) included patients without venous obstruction, and group II (n = 10, 2 women, 8 men; mean age 71.6 ± 7.0 years) included patients diagnosed with venous obstruction. Each patient was followed for 19 months. No significant differences in age or gender were found between groups I and II.

Venous obstruction developed mean 13 months after pacemaker implantation in 10 patients in group II (12.3% of the overall study population). In 7 cases, asymptomatic venous obstruction was identified using in ultrasonography scheduled at 12 months after pacemaker implantation, and symptomatic venous obstruction developed in 3 patients (3.7% of the overall study population) mean 15 months after pacemaker implantation (at 8.8, 18.9, and 18.7 months after implantation) and involved the left subclavian and axillary vein (on the side of pacemaker implantation). In the latter cases, nearly total vessel occlusion was noted, with a 90% lumen stenosis (Figs. 1–3).

The selected risk factors for venous obstruction and the significance of their occurrence (yes/no codes) are presented in Table 1.

According to the ROC curve analysis, the mean cut-off value of the procedure duration was 90 min (sensitivity 100%, specificity 87.3%; $p < 0.001$) (Fig. 4). With longer procedure duration, the risk of venous thrombosis increased markedly, but the procedure duration was excluded from the analysis, as this factor overlapped with the presence of venous anomalies (19 patients had both venous anomalies [code 1] and the procedure duration of > 90 min [code 1], and the remaining patients had code 0 for both these variables). At $\alpha = = 0.05$, the following variables had no effect on the incident thrombosis: sinus node disease, heart block, the number of veins used, diabetes, and antiplatelet treatment. Antibiotic use was also excluded from the analysis, as this factor overlapped with infection.

Thus, the following factors were selected for further analysis: a history of myocardial infarction (MI), arterial hypertension, temporary cardiac pacing, arrhythmia, venous anomalies, the NYHA class, a history of infection, tobacco smoking, established atherosclerosis, and no anticoagulant treatment. The results of this analysis are shown in Table 2.

The criteria we set ($\alpha = 0.05$ and 1-$\beta = 0.80$) were not fulfilled by three factors: arterial hypertension, established atherosclerosis, and no anticoagulant treatment (low statistical power of the test at $p < 0.05$). Ultimately, seven significant risk factors for venous obstruction were identified, i.e., a history of MI, temporary cardiac pacing, arrhythmia, venous anomalies, NYHA class, a history of infection, and tobacco smoking.

Analysis of parameters of the model describing the relationship between the probability of venous obstruction development and the number of identified risk factors yielded the following values: regression coefficient $\beta = 3.19$ (in the Wald test $\beta = 0.013$), and OR = 24.4 (95% CI 1.9–314). The coefficient of the constant term was 14.6.

These data were entered into logistic regression analysis, using the following equation: Probability of incident venous obstruction $= e^{-14.6 + 3.19x}/1 + e^{-14.6 + 3.19x}$, where x is the number of risk factors (1). Figure 5 shows the plot of the function (1), and Table 3 shows the probability of incident venous obstruction depending on the number of risk factors. Data presented in Table 3 indicate that the risk of venous obstruction was 14% with 4 risk factors, 21% with 5 risk factors, and with 6 or 7 risk factors nearly all patients developed venous obstruction (risk > 99%).

We did not observe adverse sequelae of venous thrombosis, i.e., pulmonary embolism, systemic embolism, or superior vena cava syndrome. Partial vessel recanalisation occurred upon instituting anticoagulant treatment in all 10 patients, along with resolution of clinical symptoms and signs.

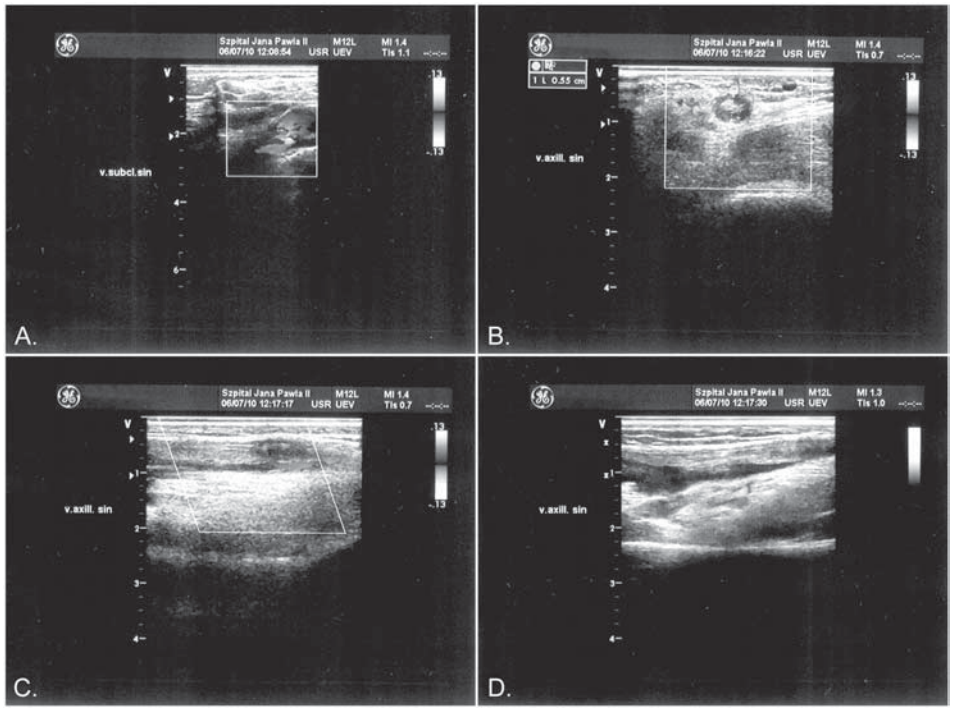

Figure 1. Venous ultrasound examination. **A.** Left subclavian vein with the pacemaker lead. Note old recanalised thrombi visible in the distal part of the vein, with turbulent flow and partially patent vessel lumen (50% stenosis); **B.** Left axillary vein dilated to 0.55 cm in diameter and obstructed with a large thrombus, with blood flow limited to a narrow lateral stream (90% stenosis); **C, D.** A thrombus extends to the distal part of a brachial vein, up to the point where brachial veins join, where it occludes only 30% of the vessel lumen

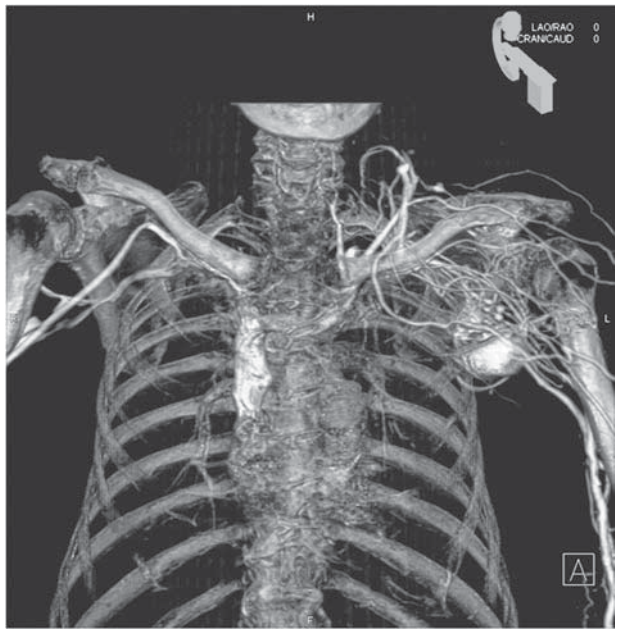

Figure 2. Three-dimensional reconstruction of the computed tomography angiography images of the veins of the upper part of the chest in a patient after pacemaker implantation. Note occlusion of the left subclavian vein and axillary veins. Superficial collateral veins are also seen in the left shoulder area

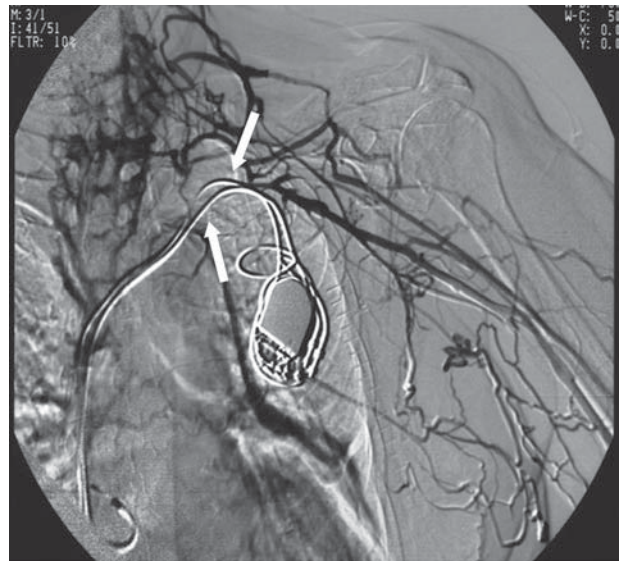

Figure 3. Digital subtraction angiography in a patient with dual-chamber pacemaker. Note left subclavian and axillary venous obstruction (arrows). Superficial collateral veins are also seen in the left shoulder area

Table 1. The analysed risk factors for venous obstruction — a preliminary analysis

Risk factor	Codes	P
History of myocardial infarction	0: no; 1: yes	0.00005
Hypertension	0: no; 1: yes	0.041
Sinus node disease	0: no; 1: yes	0.468
Heart block	II°: 2; III°: 3; 0: absence	0.182
Temporary cardiac pacing	0: no; 1: yes	0.0000
Atrial arrhythmia	0: no; 1: yes	0.00045
Number of veins used	1: 1 vein, 2: ≥ 2 veins	0.582
Venous anomalies	0: no; 1: yes	0.00000
Procedure duration [min]	0: ≤ 90; 1: > 90	0.00000
NYHA class	0 = 0 + I + II; 1 = III + IV	0.00056
Diabetes	0: no; 1: yes	0.473
Infection	0: no; 1: yes	0.00002
No antibiotic use	1: no; 0: yes	0.00002
Smoking	0: no; 1: yes	0.0007
Atherosclerosis	0: no; 1: yes	0.033
Antiplatelet treatment	0: no; 1: yes	0.426
No anticoagulation	1: no; 0: yes	0.012

DISCUSSION

Venous obstruction may result from all conditions that might interrupt blood flow in a vessel, including thrombosis. In most cases, thrombosis develops days to months after lead insertion and is usually asymptomatic. Intracardiac leads modify blood flow from laminar to turbulent, and the site of lead entry into the vein is characterised by accumulation of platelet and fibrin, associated with activation of the coagulation system. Thrombosis is later replaced by fibrosis and does not necessarily lead to venous lumen obstruction by the thrombus. Signs and symptoms of venous obstruction only occur

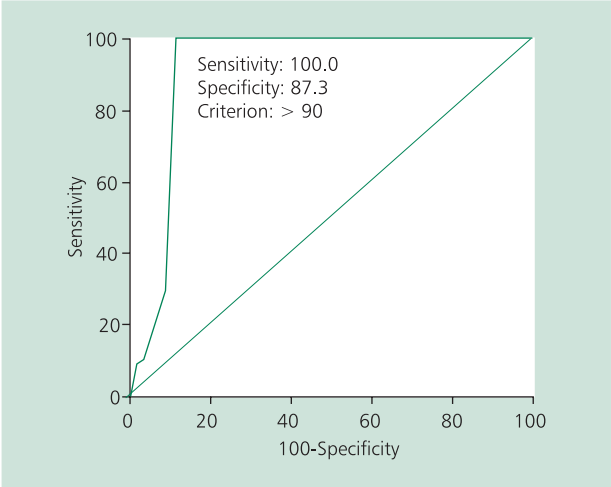

Figure 4. The ROC curve and the cut-off value for the duration of the pacemaker implantation procedure

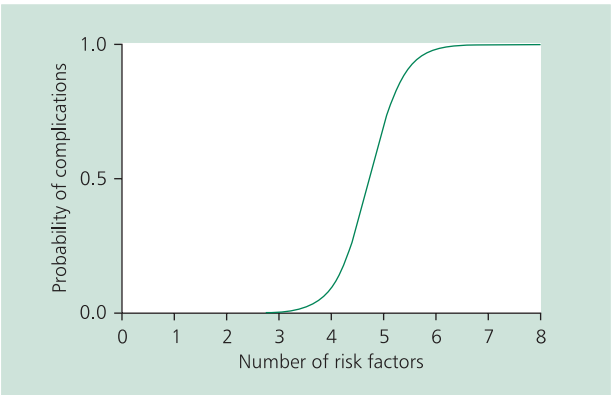

Figure 5. Probability of venous obstruction depending on the number of risk factors based on the model (1)

Table 2. The analysed risk factors for venous obstruction — differences between group I (without venous obstruction) and group II (with venous obstruction)

Variable	Group I (n = 71)	Group II (n = 10)	P	Power of the test for p = 0.05
History of myocardial infarction	18 (25.4%)	9 (90.0%)	< 0.001	0.99
Hypertension	40 (56.3%)	9 (90.0%)	0.041	0.35
Temporary cardiac pacing	6 (8.5%)	7 (70.0%)	< 0.001	0.98
Atrial arrhythmia	30 (42.3%)	10 (100.0%)	0.001	0.99
Venous anomalies	9 (12.7%)	10 (100.0%)	< 0.001	0.99
NYHA class III + IV	22 (30.9%)	9 (90.0%)	< 0.001	0.97
Infection	2 (2.8%)	6 (60.0%)	< 0.001	0.98
Smoking	11 (15.5%)	7 (70.0%)	< 0.001	0.92
Atherosclerosis	23 (32.4%)	7 (70.0%)	0.021	0.50
No anticoagulation	42 (59.1%)	10 (100.0%)	0.012	0.72

Table 3. Probability of venous obstruction depending on the number of risk factors based on the model (1)

Number of risk factors	Probability of venous obstruction
0	0.00
1	0.00
2	0.00
3	0.01
4	0.14
5	0.79
6	0.99
7	1.00

with axillary vein occlusion. Subclavian vein occlusion usually remains asymptomatic, as it is rapidly followed by the development of effective collateral circulation through the neck veins [15, 18, 22, 23].

Attempts to analyse the results of studies on venous obstruction are challenging due to differences in diagnostic methods and the duration of follow-up. Symptomatic venous obstruction is vary rare. It was reported to develop in 1–3% of patients undergoing pacemaker implantation (3.7% of patients in our study) [1, 2, 3–5, 12, 18, 22, 23]. Identification of prodromal symptoms that would allow early institution of appropriate preventive or therapeutic measures is thus difficult.

Until now, no clear factors predisposing to venous obstruction have been identified. Specifically, the incidence of this complication has not been related to age, gender, cardio-thoracic index, left ventricular ejection fraction, concomitant cardiovascular disease, indications for pacemaker implantation, the route of transvenous approach, and lead size [1, 4, 6, 14, 15].

In our study, we also did not find any relationship between indications for pacemaker implantation (sinus node disease, atrioventricular block) or the route of transvenous approach and the incidence of venous obstruction. In contrast, a history of MI, NYHA class III or IV HF, and atrial arrhythmias were found to be predictors of venous obstruction. An association between venous obstruction and atrial fibrillation was already reported by Korkeila et al. [15]. This may be explained by the fact that these patients have increased levels of procoagulant and proinflammatory markers, show elevated central venous pressure, and are more prone to activation of the coagulation cascade, initiated by vascular endothelial damage by the inserted leads, ultimately leading to vessel obstruction [15].

Likely risk factors also include previous insertion of a temporary pacing lead, systemic infection, venous anomalies, previous venous thrombosis, and smoking [1, 4–6, 9, 14, 20]. Similar findings were also reported by us. Temporary pacing leads are stiff, harder, and of different design in comparison to endocavitary leads used for permanent pacing. They may result in more extensive damage to venous endothelium and activate the coagulation cascade, induce inflammation (endothelial cell response to cytokines and inflammatory mediators results in increased expression and activity of tissue thromboplastin), and stimulate fibrosis. All these mechanisms may lead to venous obstruction. A history of systemic infection is associated with a twofold increased risk of venous obstruction. Venous anomalies resulted in difficult lead insertions, with procedure duration significantly exceeding 90 min. It is likely that more extensive endothelial damage ensued in these cases, leading to thrombophlebitis and/or endothelial response resulting in connective tissue activation. Nicotine accelerates atherogenesis, results in endothelial damage (by tissue hypoxia), and is a strong procoagulant factor.

Another factor predisposing to venous obstruction may be the presence of multiple intracardiac risk, although some authors reported that this does not affect the incidence of obstruction [1, 4, 6, 9, 14, 15, 22]. This risk is likely much smaller (8%) with the presence of a single lead and increases up to 26% when leads are introduced to two cardiac chambers [1, 4, 6, 9, 14, 15]. In contrast, further increase in the number of leads (to 3–4) does not result in any additional risk [1]. In our study, similarly to other authors, we found no association between the occurrence of venous obstruction and the number of inserted cardiac leads.

We did not find any significant differences in antiplatelet treatment (used due to a history of MI) between groups I and II in our study population, while anticoagulant treatment differed significantly (patients who developed symptomatic venous obstruction did not receive anticoagulants in addition to aspirin), but the statistical power of the test was weak and below the set threshold for significance. This was caused by a low number of patients in our study group. The same factor was likely responsible for the observed lack of association between venous obstruction and hypertension, atherosclerosis, and diabetes. Thus, based on our findings it cannot be concluded that absence of anticoagulant treatment predisposes to symptomatic venous obstruction.

Limitations of the study

Our analysis and conclusions are limited by a low number of cases of venous obstruction (n = 10). For this reason, and also due to a relatively short follow-up duration, we were not able to perform a multivariate analysis of the effects of interactions between the studied risk factors for venous obstruction.

CONCLUSIONS

1. Risk factors for venous obstruction include a history of MI, temporary cardiac pacing, arrhythmia, venous ano-

malies, infections, NYHA class III and IV HF, and tobacco smoking.

2. In patients who had more than 6 risk factors, almost a 100% probability of incident venous obstruction was observed.

Conflict of interest: *none declared*

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Związek między wybranymi czynnikami ryzyka a wystąpieniem niedrożności żylnej po wszczepieniu rozrusznika serca. Czynniki demograficzne i kliniczne

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Streszczenie

Wstęp: Do najczęstszych powikłań pooperacyjnych i niosących największe zagrożenie można zaliczyć niedrożność naczyń żylnych w miejscu wprowadzenia elektrod. Niedrożność żylna obejmuje wszystkie przyczyny prowadzące do braku przepływu krwi przez naczynie, w tym zakrzepicę i związaną z nią zatorowość płucną. Wyniki badań nad niedrożnością żylną, jako powikłaniem po wdrożeniu przezżylnego układu stymulującego, są rozbieżne. Ważne jest zidentyfikowanie chorych podatnych na rozwój niedrożności żylnej.

Cel: Celem pracy było określenie prawdopodobieństwa wystąpienia niedrożności żylnej w zależności od liczby czynników ryzyka.

Metody: Analizowano dane 81 (31 kobiet, 50 mężczyzn; średnia wieku 71,1 ± 7,6 roku) chorych z implantowanym układem stymulującym serce. U każdego pacjenta przed wszczepieniem rozrusznika serca oceniano wskazania do zabiegu, klasę niewydolności serca wg NYHA, choroby współistniejące, nikotynizm, stosowane leki przeciwpłytkowe i przeciwzakrzepowe, antybiotyki, wywiad w kierunku przebytych powikłań zakrzepowych, infekcyjnych oraz wcześniejszej czasowej stymulacji serca. W trakcie zabiegu wszczepienia rozrusznika serca oceniano rodzaj dostępu żylnego i czas trwania implantacji. Przeprowadzono badanie ultrasonograficzne żył kończyn górnych i obręczy barkowej oraz szyi (USG), przed zabiegiem i po wszczepieniu rozrusznika serca w 6. i 12. miesiącu. W celu potwierdzenia rozpoznania niedrożności wykonano tomografię komputerową i angiografię klasyczną substrakcyjną.

Wyniki: Uwzględniając wystąpienie powikłania niedrożności żylnej po wszczepieniu rozrusznika serca, chorych podzielono na 2 grupy. Grupę I (29 kobiet, 42 mężczyzn; śr. wieku 71,0 ± 7,7 roku) stanowili pacjenci, u których nie stwierdzono niedrożności. Do grupy II (2 kobiety, 8 mężczyzn; śr. wieku 71,6 ± 7,0 roku) włączono chorych z niedrożnością żylną. Okres obserwacji każdego badanego wynosił 19 miesięcy. U 10 chorych z grupy II rozwinęła się niedrożność żylna w czasie średnio 13 miesięcy po zabiegu wszczepienia rozrusznika serca, co stanowiło 12,3% całej populacji. W tej grupie tylko u 3 (3,7% całej populacji) chorych pojawiła się objawowa niedrożność żylna w czasie średnio 15 miesięcy po zabiegu (odpowiednio: 8,8; 18,9; 18,7 miesięcy), natomiast w pozostałych 7 przypadkach bezobjawową niedrożność żylną wykryto w czasie 12 miesięcy, kiedy przypadał termin planowego badania USG. Czynnikami zwiększającymi ryzyko niedrożności były: przebyty zawał serca, stymulacja czasowa, arytmia, anomalie żylne, III i IV klasy NYHA, przebyte infekcje oraz palenie tytoniu. Prawdopodobieństwo wystąpienia niedrożności w zależności od liczby wyodrębnionych czynników ryzyka opisano modelem: $e^{-14.6 + 3.19x}/1 + e^{-14.6 + 3.19x}$, gdzie x oznaczała liczbę czynników. U chorych, u których stwierdzono powyżej 6 czynników ryzyka niedrożności żylnej, obserwuje się prawie 100-procentowe prawdopodobieństwo rozwinięcia się tego powikłania.

Wnioski: 1. Czynnikami ryzyka niedrożności żylnej były: przebyty zawał serca, czasowa stymulacja zewnętrzna, arytmia, anomalie żylne, infekcja, klasa wg NYHA większa niż II stopień i palenie tytoniu. 2. U chorych, u których stwierdzono powyżej 6 czynników ryzyka niedrożności żylnej, obserwuje się prawie 100-procentowe prawdopodobieństwo rozwoju tego powikłania.

Słowa kluczowe: układ stymulujący serce, niedrożność żylna, czynniki ryzyka niedrożności

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