Mangileva T.A.[1], Kazantseva O.A.[2], Karaseva I.V.[2]

[1] Medical Academy named after S.I. Georgievsky, CFU named after V.I. Vernadsky, Simferopol, Russia

[2] Republican Clinical Hospital named after N.A. Semashko, structural unit "Car-diological dispensary", Simferopol, Russia

Peculiarity of clinical course of postpericardiotomy syndrome in different methods of surgery and postoperative antithrombotic therapy

Aim	To compare features of the disease course and the effectiveness of nonsteroidal anti-inflammatory drug (NSAID) treatment of postpericardiotomy syndrome (PPS) in patients after coronary bypass (CB) surgery who were treated with antiplatelet drugs and in patients after surgical correction of heart valve disease (CHVD) who received the anticoagulant warfarin for prevention of thrombotic complications.
Material and methods	This study included 89 patients of whom 53 patients had underwent CB and 36 patients had underwent CHVD. At 15 [13; 15] days after surgery, the severity of inflammatory response, the state of coagulation hemostasis, and hematocrit were studied. At 5 days after the first test, blood count and measurement of C-reactive protein were repeated. Echocardiography was used to determine the presence and volume of pleural effusion. For prevention of thrombotic complications, antiplatelet drugs were administered after CB and warfarin was administered after CHVD. PPS was detected in 35 (66%) patients after CB and 18 (50%) patients after CHVD. The ibuprofen treatment (600 mg twice a day) was administered to all patients with PPS. If positive changes in inflammatory markers were absent during the NSAID treatment, ibuprofen was replaced with prednisolone 0.5 mg/kg body weight with subsequent laboratory and instrumental monitoring.
Results	Patients after CHVD treated with warfarin had higher values of international normalized ratio (INR) and activated partial thromboplastin time (aPPT) and lower values of prothrombin index (PTI), fibrinogen ($p<0.001$ for all), hemoglobin ($p=0.0016$), and hematocrit ($p=0,0032$) than patients after CB treated with antiplatelet drugs. 21 (40%) patients with PPS required changing the anti-inflammatory therapy from ibuprofen to prednisolone. These patients displayed hypocoagulation, which was evident as reduced PTI ($p=0.0023$) and fibrinogen ($p=0.0209$), increased INR ($p=0.0291$) and aPPT ($p=0.0416$), and a higher incidence of pericardial effusion ($p=0.0080$). The insufficient effectivity of NSAIDs that required administration of prednisolone was more frequently observed in patients after CHVD (61% vs. 29%, $p=0.037$).
Conclusion	Hypocoagulation observed in patients after CHVD due to the anticoagulant treatment with warfarin was associated with more severe course of PPS and lower effectiveness of the NSAID treatment compared to patients after CB. This results in more frequent replacement of NSAIDs with glucocorticoids in the treatment of patients after CHVD.

Keywords	Postpericardiotomy syndrome; coronary bypass; surgical repair of heart valve disease; antithrombotic therapy; hypocoagulation; nonsteroidal anti-inflammatory drugs; prednisolone
For citation	Mangileva T.A., Kazantseva O.A., Karaseva I.V. Peculiarity of clinical course of postpericardiotomy syndrome in different methods of surgery and postoperative antithrombotic therapy. Kardiologiia. 2021;61(4):53–59. [Russian: Мангилева Т.А., Казанцева О.А., Карасева И.В. Особенности течения постперикардиотомического синдрома при различных способах оперативного лечения и послеоперационной антитромботической терапии. Кардиология. 2021;61(4):53–59]

Corresponding author	Mangileva T.A. E-mail: elnego@mail.ru

Introduction

Postpericardiotomy syndrome (PPS) is a clinical syndrome most commonly developed after surgical interventions involving pericardiotomy. This complication is usually diagnosed in 1–6 weeks after surgery in 10–40% of patients [1]. Despite numerous studies the mechanism of PPS development and the most significant predictors of its course are still not completely understood [2–4]. However, some experts associate its development with blood trapped in the pericardial and pleural cavity [5].

The postoperative prevention of thrombotic complications varies significantly in different surgical techniques. For example, after preventive coronary artery bypass grafting (CABG), antiplatelet drugs are administered [6], and after valve reconstruction surgeries and mechanical and bioprosthetic valve replacements, anticoagulant warfarin is essential [7],

which is accompanied by pronounced drug-induced hypocoagulation.

The frequency of cardiac surgical interventions, such as open-heart surgeries with pericardiotomy, in on the increase. On the other hand, the prevention of postoperative thrombotic complications is becoming very important. It is, therefore, relvant that a study of the effects of antithrombotic therapy on the course of the postoperative inflammatory process be carried out.

Objective

To perform a comparative analysis of the peculiarities of the PPS course and the efficacy of its treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) in patients subjected to CABG who received antiplatelet drugs, and patients after surgical correction of valve disease who took warfarin to prevent thrombotic complications.

Material and methods

The study included 89 patients who were transferred from the cardiac surgery department to the cardiology department in N.A. Semashko Republican Clinical Hospital 8–28 days after the surgical intervention. The inclusion criterion was cardiac surgery with pericardiotomy 1 to 4 weeks before admission. Exclusion criteria were: active infection requiring antibacterial therapy, postoperative wound complications; less than three months after myocardial infarction; heart, lung, pleura, or pericardium malignancies or traumas; glomerular filtration rate less than 30 mL/min/1.73 m^2; chronic heart failure functional class IV. Aortocoronary/mammary coronary bypass grafting was performed in 53 patients. 34 patients underwent prosthetic aortic (n=22) or mitral (n=12) valve replacement, and 2 patients had valvuloplasty. PPS was diagnosed on the basis of two or more of the following criteria: fever of unknown origin; pleural or pericardial chest pain; pericardial and/or pleural rub; first-time or progressing pericardial and/or pleural effusion with elevated levels of C-reactive protein (CRP) [8, 9]. PPS was diagnosed in 53 subjects: 35 patients after CABG; and 18 patients after surgical correction of valve disease. The signs of PPS were absent in 36 patients (18 patients after CABG and 18 patients after surgical correction of valve disease). Among patients with PPS, pericardial effusion was detected in 34 (64%) patients, pleural effusion with elevated CRP in 27 (51%) patients, fever without alternative cause in 7 (13%) patients, chest pain in 52 (98%) patients, while pericardial or pleural rub was not detected in the subjects. The criteria of PPS severity were persistent fever above 37°C, persistent high or increasing erythrocyte sedimentation rate (ESR) and CRP levels, increased pericardial or pleural effusion for five or more days during the use of NSAIDs, and ultrasonographic evidence of simultaneous pericardial and pleural effusion. The above parameters were evaluated on admission and 5 days after the beginning of anti-inflammatory therapy. The criteria for evaluating the efficacy of anti-inflammatory treatment were: absence of fever; regression of the amount of pleural or pericardial effusion; normalization of laboratory indicators of inflammation. They were re-evaluated 5 days after the beginning of anti-inflammatory therapy.

Upon admission to the cardiology department, a complete blood count was made using a Swelab Alfa Hematology Analyzer (Boule Medical A.B., Sweden) including hemoglobin (Hb), hematocrit (Ht), WBC, and ESR, in a median of 15 [13; 15] after surgery. Prothrombin ratio (PR), international normalized ratio (INR), activated partial thromboplastin time (aPTT), fibrinogen levels were also established. CRP levels were investigated by means of immunoturbidimetry using a Cobas e 411 Roche biochemical analyzer (Roche Diagnostics GmbH, Germany). The complete blood count and CRP were repeated 5 days after the first test, in a median of 20 [20; 22] days after surgery.

All patients underwent color Doppler echocardiography using a Philips EnVisor C system (Philips, Netherlands) 20 days after surgery. Echocardiography was used to determine left ventricular ejection fraction (LVEF) and the presence of pericardial and pleural effusion. In order to detect rhythm and conduction disorders, 24-hour ECG monitoring was performed using a Cardiotechnics-04–8 (M) device (Incarta, Russia) which detected atrial fibrillation in 21% of subjects. All patients were subjected to chest X-ray, and a series of examinations, if necessary. The retrospective analysis of cardiosurgery department discharge summaries estimated the duration of bed rest after surgery, donated blood transfusion, number of postoperative drainages, and LVEF before surgery. Body mass index (BMI) was calculated at admission.

All patients received antiplatelet drugs after CABG: acetylsalicylic acid 75–100 mg/day as monotherapy or in combination with clopidogrel 75 mg/day [6]. After surgical correction of valve disease, patients received warfarin 2–5 mg/day irrespective of the type of valve prosthesis implanted [7]. Drug treatment started after adequate hemostasis was achieved after surgery (days 2–3 after surgery) in the cardiosurgery department and continued at the time of examination.

Initial treatment with NSAID ibuprofen 600 mg/bid was ordered for all patients with PPS. If anti-inflam-

ISSN 0022-9040. Kardiologiia. 2021;61(4). DOI: 10.18087/cardio.2021.4.n1374

matory therapy failed, and pericardial and/or pleural effusion persisted or progressed, and there were no positive trends in the levels of CRP and ESR, and body temperature persisted over 37.0°C, ibuprofen was replaced with prednisolone 0.5 mg/kg of body weight. Prednisolone was administered in 21 patients (10 patients after CABG and 11 patients after surgical correction of valve disease).

The study was performed in accordance with the Declaration of Helsinki. The study was approved by the ethics committee of the Crimean Federal University. All patients signed the informed consent to participate in the study.

The data obtained was analyzed using Statistica 12.0. The normality of distribution was evaluated using the Shapiro-Wilk test. Variables with normal distribution were compared using the unpaired (independent samples) or paired (dependent samples) Student t-test. The results are expressed as mean and standard deviation (M (SD)). If at least one of the distributions was asymmetric, the non-parametric Mann-Whitney or Wilcoxon tests were applied (related samples). Then, the data was represented as the median and the 25th and 75th quartiles (Me [P25; P75]). Qualitative data is represented as percentages (%). Nominal variables were compared using the Pearson chi-squared test. If the number of observations in at least one cell of the 2×2 table was less than 5, the Fisher exact test was applied. The value α=0.05 was used as a critical level of bilateral significance, i.e., differences at $p<0.05$ were considered statistically significant.

Results

In order to analyze the data received, patients were divided into the following groups: patients after CABG (n=53) and patients after surgical correction of valve disease (n=36). PPS was diagnosed in 35 (66%) of 53 patients after CABG and 18 (50%) of 36 patients after surgical correction of valve disease. Comparative analysis of the results of the examination of patients with and without PPS included 18 patients after CABG and 18 patients after surgical correction of valve disease. Patients with PPS had higher ESR and CRP levels and lower hematocrit at admission to the cardiology department than subjects without PPS (Table 1). Diabetes mellitus was established in 31 patients with PPS and only 8 patients without PPS. Both groups showed reduced inflammation within a week: ESR ($p<0.0001$ for patients with PPS and $p=0.00036$ for patients without PPS) and CRP ($p<0.0001$ for patients with PPS and $p=0.0431$ for patients without PPS) significantly decreased. Significant growth of hemoglobin ($p=0.0133$) and hematocrit ($p=0.0018$) was observed only in patients with PPS.

In orer to study the effects of the surgical technique on the late postoperative period, the characteristics of patients after CABG and surgical correction of valve disease were compared (Table 2). The study included 53 patients after CABG (35 patients had PPS) and 36 patients after surgical correction of valve disease (18 patients with PPS). The groups did not differ in terms of demographics, BMI, glycemia, frequency of smoking, LVEF, duration of bed rest, and number of drainages. There were no inter-group differences in the frequency of PPS ($p=0.187$). In the CABG group, patients smoked more often ($p=0.021$) and had concomitant diabetes mellitus ($p=0.030$). In the reconstructive surgery group, high-density lipoprotein cholesterol ($p=0.041$) was higher. Surgical correction of valve disease was associated with more frequent ($p=0.23$) arrhythmias (mainly atrial fibrillation) and longer bed rest after surgery ($p=0.00093$). There was a decrease in markers of inflammation during treatment in both groups: ESR ($p=0.000046$ after surgical correction of valve disease and $p<0.0001$ after CABG) and CRP ($p=0.000196$ after surgical correction of valve disease and $p<0.0001$ after CABG).

After surgical correction of valve disease, patients had hypocoagulation during the administration of warfarin, which was manifested by increased INR and aPTT and decreased PR and fibrinogen. Although the rate of blood transfusions did not differ after CABG and surgical correction of valve disease, patients after valve reconstructions had lower Hb and Ht in 20 [20; 22] days after surgical correction of valve disease. A statistically significant increase in hemoglobin during treatment ($p=0.00044$) was observed only in patients who had undergone CABG. Hematocrit increased in both groups, but more significantly after CABG ($p=0.0462$ after surgical correction of valve disease and $p=0.00005$ after CABG).

Patients with PPS who had no positive changes in the clinical picture, laboratory tests, and clinical investigations during the use of ibuprofen were transferred to prednisolone. There were 10 (18%) such patients in the CABG group and 11 (30.5%) patients after surgical correction of valve disease. Drug treatment compliance did not differ in patients with the different treatment options, thus excluding its effect on the outcomes. Signs of inflammation decreased and hemoglobin and hematocrit increased over time in both groups. If ibuprofen failed (Table 3), patients were more likely to have hypocoagulation, which manifested as a decrease in PR and fibrinogen levels and increased INR and aPTT. Patients who required a change of

Table 1. Examination results of patients with and without postpericardiotomy syndrome

Parameter	With PPS (36)	Without PPS (36)	p
Hb at admission, g/L	118 (14.9)	124 (13.6)	0.086
Hb in 5 days, g/L	123 (12.4)	128 (15.7)	0.170
Ht at admission, %	32 (4.5)	35 (4.03)	0.013
Ht in 5 days, %	35 (3.8)	36 (5.2)	0.140
ESR at admission, mm/h	35.0 [34.0; 46.0]	15.0 [12.0; 24.5]	<0.0001
ESR in 5 days, mm/h	25.0 [12.0; 33.5]	11.5 [10.0; 15.5]	<0.0001
CRP at admission, g/L	12.0 [6.0; 12.0]	0.0 [0.0; 0.0]	<0.0001
CRP in 5 days, g/L	0.0 [0.0; 4.0]	0.0 [0.0; 0.0]	0.026
INR	1.775 [1.0; 2.385]	1.4 [1.0; 2.25]	0.469
Fibrinogen, g/L	4.2 [3.60; 4.50]	4.1 [3.65; 4.50]	0.804
PR, %	66.0 [59.0; 99.0]	66.5 [49.0; 100.0]	0.478
aPPT, sec	30.0 [27.0; 40.0]	34.0 [27.0; 40.0]	0.353
Blood transfusion, number of patients (%)	7 (19)	1 (3)	0.055

Hb, hemoglobin; Ht, hematocrit; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; INR, international normalized ratio; PR, prothrombin ratio; aPTT, activated partial thromboplastin time.

Table 2. Examination results of patients after coronary bypass grafting and surgical correction of valve disease

Parameter	CABG (53)	Surgical correction of valve disease (36)	p
Hb at admission, g/L	123 (13.8)	117 (14.5)	0.061
Hb in 5 days, g/L	129 (14.1)	120 (11.1)	0.0016
Ht at admission, %	35 [31.3; 37.9]	34 [30.15; 35.65]	0.11
Ht in 5 days, %	37 (4.7)	34 (3.1)	0.0032
ESR at admission, mm/h	35.0 [17.0; 41.0]	34.0 [15.0; 38.0]	0.51
ESR in 5 days, mm/h	19.0 [10.0; 27.0]	15.5 [10.0; 26.5]	0.78
CRP at admission, g/L	6.0 [0.0; 12.0]	4.0 [0.0; 12.0]	0.38
CRP in 5 days, g/L	0.0 [0.0; 0.0]	0.0 [0.0; 0.0]	0.60
INR	1.0 [0.90; 1.06]	2.3 [2.1; 2.8]	<0.0001
Fibrinogen, g/L	4.5 [4.2; 4.5]	3.6 [3.33; 3.90]	<0.0001
PR, %	100.0 [88.0; 107.0]	52.5 [45.0; 60.0]	<0.0001
aPPT, sec	27.0 [27.0; 28.0]	40.0 [37.5; 41.0]	<0.0001
Pericardial effusion, number of patients (%)	14 (26%)	20 (56%)	0.055
Effusion volume, mL	0.0 [0.0; 80.0]	0.0 [0.0; 70.0]	0.23
Pleural effusion, number of patients (%)	17 (32%)	10 (28%)	0.665
Effusion volume, cm	0.0 [0.0; 1.40]	0.0 [0.0; 0.85]	0.64
Blood transfusion, number of patients (%)	8 (15%)	4 (11%)	0.755

Hb, hemoglobin; Ht, hematocrit; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; INR, international normalized ratio; PR, prothrombin ratio; aPTT, activated partial thromboplastin time.

drugs were more likely to have pericardial effusion and a higher volume than those with effective NSAID treatment. It should be emphasized that NSAID failure was significantly more common in patients after surgical correction of valve disease who used warfarin in the post-operative period than in patients after CABG who received only antiplatelet drugs (61% vs. 29%, p=0.037).

Discussion

Analysis of the various risk factors of PPS has been undertaken by many researchers [2–4, 10]. The results are ambiguous, and often contradictory. One study [11] found an inverse relationship between diabetes mellitus and the incidence of PPS in patients after CABG, which was not confirmed by other researchers [10, 12]. In our work, the incidence of diabetes mellitus was, by contrast, higher in patients with PPS than in patients without PPS.

Prior to 2002, markers of inflammation (ESR, CRP, WBCs) were included in the diagnostic criteria of PPS, but later they were abandoned due to lack of specificity [12]. According to our data, the inflammatory response

ISSN 0022-9040. Kardiologiia. 2021;61(4). DOI: 10.18087/cardio.2021.4.n1374

in patients with PPS in most cases is more pronounced than in patients without PPS in 15 and 20 days after surgery. The timing of tests can explain the apparent contradictions between our data and the results of Ilmar Choler et al., who evaluated the inflammation markers on days 3, 7–10, and 30 after surgery.They found no intergroup differences of the inflammation markers in patients with and without PPS [13]. Non-specific markers of inflammation are likely to reach their maximum in PPS (a mean of 14–16 days after open-heart surgery [10, 14]), which determines the administration of anti-inflammatory therapy, and then regress due to the treatment.

Differences in the determination of PPS development factors and the severity of its course can be associated with both the transformation of PPS definition and a wider variety of heart surgeries, which can be complicated by the development of PPS. In a study including 28,761 patients with open-heart surgery, a higher frequency of PPS was detected after aortic or mitral valve replacement than after CABG [4]. In order to clarify the effects of surgical techniques on the development of PPS, we divided patients into two groups: patients after CABG and patients after surgical correction for valve disease. Since smoking and diabetes mellitus are the established risk factors for coronary artery disease [15], it is reasonable that they are detected more often in patients who have undergone CABG. Even with statin treatment, high-density lipoprotein cholesterol levels were lower in CAD patients than in patients with valve disease. Arrhythmias (mainly short paroxysms of atrial fibrillation) were observed more often in patients with valve diseases, which can be explained by more severe atrial remodeling [16]. Surgical correction of valve disease required longer bed rest after intubation, probably due to more extensive damage of the pleural cavity and myocardium during surgery.

The most significant inter-group differences concerned coagulation hemostasis. The effects of post-operative antithrombotic therapy in patients after CABG and surgical correction of valve disease on PPS severity and treatment efficacy remain neglected. At the same time, antiplatelet therapy is indicated for all patients after CABG [6], and warfarin is by necessity administered after surgical correction of valve disease anticoagulant [7]. The use of warfarin in our study was accompanied by severe hypocoagulation with the involvement of intrinsic, extrinsic, and common coagulation pathways. These disorders are likely to cause microbleeding into the pericardial and pleural cavity. This can contribute to more frequent and severe PPS and is confirmed by lower hemoglobin and hematocrit levels and slow recovery of RBC count in patients after surgical correction of valve disease.

Under the current guidelines [8, 9], aspirin and ibuprofen are recommended as first-line therapy in PPS, and if they fail, glucocorticoids are recommended. The

Table 3. Examination results of patients with the postpericardiotomy syndrome with good and unsatisfactory effect of ibuprofen therapy

Parameter	Therapy failed (n=21)	Effective therapy (n=32)	p
Hb at admission, g/L	115 (13.3)	121 (14.8)	0.103
Hb in 5 days, g/L	120 (11.5)	126 (11.8)	0.082
Ht at admission, %	31 (3.8)	33 (4.6)	0.174
Ht in 5 days, %	34 (3.3)	36 (3.8)	0.108
ESR at admission, mm/h	38.0 [35.0;41.0]	35.0 [33.0; 46.0]	0.643
ESR in 5 days, mm/h	25.0 [12.0; 30.0]	25.0 [12.0; 31.0]	0.870
CRP at admission, g/L	12.0 [6.0; 12.0]	6.0 [6.0; 12.0]	0.368
CRP in 5 days, g/L	0.0 [0.0; 6.0]	0.0 [0.0; 2.0]	0.317
INR	2.04 [1.0; 2.79]	1.0 [1.0; 1.425]	0.0291
Fibrinogen, g/L	4.1 [3.3; 4.5]	4.5 [4.1; 4.5]	0.0209
PR, %	64.0 [56.0; 88.0]	100.0 [73.7; 105.0]	0.0023
aPPT, sec	31.0 [28.0; 40.0]	27.0 [27.0; 31.0]	0.0416
Pericardial effusion, number of patients (%)	18 (86)	16 (50)	0.008
Effusion volume, mL	80.0 [60.0; 140.0]	30.0 [0.0; 90.0]	0.0141
Pleural effusion, number of patients (%)	9 (43)	18 (56)	0.340
Effusion volume, cm	0.0 [0.0; 2.2]	1.15 [0.0; 1.9]	0.617
Blood transfusion, number of patients (%)	5 (24)	6 (19)	0.736

Hb, hemoglobin; Ht, hematocrit; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; INR, international normalized ratio; PR, prothrombin ratio; aPTT, activated partial thromboplastin time.

change from ibuprofen to prednisolone was required much more often in patients after surgical correction of valve disease than in patients after CABG. This can be associated with the mandatory administration of warfarin after valve reconstruction surgeries. The use of oral anticoagulants in the post-operative period was considered as a risk factor for PPS by other researchers, too [17]. Blood trapped in the pericardial and pleural cavities may cause inflammatory reactions occurring in the early and late post-operative periods [5, 12].

Permanent oral anticoagulant therapy with vitamin K antagonists is recommended for all patients with mechanical valve prostheses and is administered to prevent thrombotic complications within the first three months after the bioprosthesis implantation or valve reconstruction [7]. Although according to Eikelboom et al. [18], direct oral anticoagulants with a significantly lower risk of bleeding than that of vitamin K antagonists showed worse results in the treatment of patients with mechanical prosthetic heart valves, and are contraindicated with safety and efficacy subject to further study [7].

Thus, the study established that the course of PPS was more severe in patients after surgical correction of valve disease, which was confirmed by the changes in the inflammatory markers (ESR, CRP) and the incidence of pericardial effusion on ultrasonography. Patients who had undergone valve reconstruction and who were taking warfarin were more likely to require the replacement of ibuprofen with prednisolone due to the inefficacy of NSAIDs than patients after CABG who were treated with antiplatelet drugs after surgery. The administration of anticoagulants is one factors potentially affecting the course of PPS. Glucocorticoids are not recommended in patients with PPS as the first-line anti-inflammatory therapy [8]. For this reason, patients with PPS and signs of hypocoagulation should particularly carefully monitor the efficacy of NSAIDs, in order to replace NSAIDs with prednisolone in due time, in order to reduce the duration of treatment and prevent the development of complications.

Since the study was limited by a relatively small number of patients, these findings are preliminary. Large-scale research is required to implement these findings in real-world clinical practice.

No conflict of interest is reported.

The article was received on 21/09/2020

REFERENCES

1. Imazio M, Brucato A, Ferrazzi P, Spodick DH, Adler Y. Postpericardiotomy syndrome: a proposal for diagnostic criteria. Journal of Cardiovascular Medicine. 2013;14(5):351–3. DOI: 10.2459/JCM.0b013e328353807d
2. van Osch D, Dieleman JM, Bunge JJ, van Dijk D, Doevendans PA, Suyker WJ et al. Risk factors and prognosis of postpericardiotomy syndrome in patients undergoing valve surgery. The Journal of Thoracic and Cardiovascular Surgery. 2017;153(4):878-885.e1. DOI: 10.1016/j.jtcvs.2016.10.075
3. Imazio M, Brucato A, Rovere ME, Gandino A, Cemin R, Ferrua S et al. Contemporary Features, Risk Factors, and Prognosis of the Post-Pericardiotomy Syndrome. The American Journal of Cardiology. 2011;108(8):1183–7. DOI: 10.1016/j.amjcard.2011.06.025
4. Lehto J, Kiviniemi T, Gunn J, Airaksinen J, Rautava P, Kytö V. Occurrence of Postpericardiotomy Syndrome: Association With Operation Type and Postoperative Mortality After Open-Heart Operations. Journal of the American Heart Association. 2018;7(22):e010269. DOI: 10.1161/JAHA.118.010269
5. Boyle EM, Gillinov AM, Cohn WE, Ley SJ, Fischlein T, Perrault LP. Retained Blood Syndrome after Cardiac Surgery: A New Look at an Old Problem. Innovations: Technology and Techniques in Cardiothoracic and Vascular Surgery. 2015;10(5):296–303. DOI: 10.1097/imi.0000000000000200
6. Valgimigli M, Bueno H, Byrne R, Collet J, Costa F, Jeppsson A et al. 2017 ESC focused update on dual antiplatelet therapy in coronary artery disease developed in collaboration with EACTS. Russian Journal of Cardiology. 2018;23(8):113–63. [Russian: Valgimigli M., Bueno H., Byrne R., Collet J., Costa F., Jeppsson A. и др. Двойная антитромбоцитарная терапия при ишемической болезни сердца, обновленная версия 2017 года. Российский кардиологический журнал. 2018;23(8):113–63]. DOI: 10.15829/1560-4071-2018-8-113-163
7. Baumgartner H, Falk V, Bax J, De Bonis M, Hamm C, Holm PJ et al. 2017 ESC/EACTS guidelines for the management of valvular heart disease. Russian Journal of Cardiology. 2018;23(7):103–55. [Russian: Baumgartner H., Falk V., Bax J., De Bonis M., Hamm C., Holm P.J. и др. Рекомендации ESC/EACTS 2017 по лечению клапанной болезни сердца. Российский кардиологический журнал. 2018;23(7):103-55]. DOI: 10.15829/1560-4071-2018-7-103-155
8. Ministry of Health of Russian Federation. Clinical guidelines. Postcardiotomy syndrome. I97.0. Av. at: https://racvs.ru/clinic/files/2020/postcardiotomy_syndrome.pdf. 2020. [Russian: Министерство здравоохранения Российской Федерации. Посткардиотомический синдром. Клинические рекомендации. I97.0. 2020. Доступно на: https://racvs.ru/clinic/files/2020/postcardiotomy_syndrome.pdf]
9. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases. Russian Journal of Cardiology. 2016;21(5):117–62. [Russian: Adler Y., Charron P., Imazio M., Badano L., Barón-Esquivias G., Bogaert J. и др. Рекомендации ESC по диагностике и ведению пациентов с заболеваниями перикарда 2015. Российский кардиологический журнал. 2016;21(5):117-62]. DOI: 10.15829/1560-4071-2016-5-117-162
10. Lehto J, Gunn J, Björn R, Malmberg M, Airaksinen KEJ, Kytö V et al. Adverse events and survival with postpericardiotomy syndrome after surgical aortic valve replacement. The Journal of Thoracic and Cardiovascular Surgery. 2020;160(6):1446–56. DOI: 10.1016/j.jtcvs.2019.12.114
11. Lehto J, Gunn J, Karjalainen P, Airaksinen J, Kiviniemi T. Incidence and risk factors of postpericardiotomy syndrome requiring medical attention: The Finland postpericardiotomy syndrome study. The Journal of Thoracic and Cardiovascular Surgery. 2015;149(5):1324–9. DOI: 10.1016/j.jtcvs.2015.01.031
12. Van Osch D, Nathoe HM, Jacob KA, Doevendans PA, van Dijk D, Suyker WJ et al. Determinants of the postpericardiotomy syn-

ISSN 0022-9040. Kardiologiia. 2021;61(4). DOI: 10.18087/cardio.2021.4.n1374

drome: a systematic review. European Journal of Clinical Investigation. 2017;47(6):456–67. DOI: 10.1111/eci.12764
13. Köhler I, Saraiva PJ, Wender OB, Zago AJ. Behavior of inflammatory markers of myocardial injury in cardiac surgery: laboratory correlation with the clinical picture of postpericardiotomy syndrome. Arquivos Brasileiros de Cardiologia. 2003;81(3):285–90. DOI: 10.1590/S0066-782X2003001100008
14. Nishimura M, Goda N, Hatazawa K, Sakaguchi K. Delayed diagnosis of postcardiac injury syndrome. BMJ Case Reports. 2019;12(2):e228877. DOI: 10.1136/bcr-2018-228877
15. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice. Russian Journal of Cardiology. 2017;22(6):7–85. [Russian: Piepoli M.F., Hoes A.W., Agewall S., Albus C., Brotons C., Catapano A.L. и др. Европейские рекомендации по профилактике сердечно-сосудистых заболеваний в клинической практике (пересмотр 2016). Российский кардиологический журнал. 2017;22(6):7-85]. DOI: 10.15829/1560-4071-2017-6-7-85
16. Kirchhof P, Benussi S, Kotecha D, Ahlsson A, Atar D, Casadei B et al. 2016 ESC Guidelines on management of atrial fibrillation developedin collaboration with EACTS. Russian Journal of Cardiology. 2017;22(7):7–86. [Russian: Kirchhof P., Benussi S., Kotecha D., Ahlsson A., Atar D., Casadei B. et al. Рекомендации ESC по лечению пациентов с фибрилляцией предсердий, разработанные совместно с EACTS. Российский кардиологический журнал. 2017;22(7):7-86]. DOI: 10.15829/1560-4071-2017-7-7-86
17. Nakatseva E.V., Moiseeva O.M., Titarenko O.T., Androsova M.V., Nikulina V.P. Postpericardiotomy syndrome after open heart surgery: modern diagnostic methods. Arterial Hypertension. 2008; 14(S1-2):23–8. [Russian: Накацева Е.В., Моисеева О.М., Титаренко О.Т., Андросова М.В., Никулина В.П. Постперикардиотомный синдром после операции на открытом сердце: современные методы диагностики. Артериальная гипертензия. 2008;14(S1-2):23-8]
18. Eikelboom JW, Connolly SJ, Brueckmann M, Granger CB, Kappetein AP, Mack MJ et al. Dabigatran versus Warfarin in Patients with Mechanical Heart Valves. New England Journal of Medicine. 2013;369(13):1206–14. DOI: 10.1056/NEJMoa1300615