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POSTOPERATIVE PERICARDIAL EFFUSION: PECULIARITIES OF THE DEVELOPMENT AND COURSE

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Heart transplantation continues to be the gold standard treatment for end-stage chronic heart failure. As with any cardiac surgery, heart transplantation is associated with postoperative complications. One of the most common complications is postoperative pericardial effusion. Heart recipients have a greater risk of developing pericardial effusion than patients after cardiac surgery on their own heart, due to surgical and immunological features. Severe pericardial effusions negatively affect the postoperative period and may be the cause of life-threatening conditions. Identification of risk factors, prevention, early diagnosis and treatment of this disease can significantly reduce the risks of adverse events in this group of patients. The purpose of this literature review is to analyze the development and course of pericardial effusion in heart recipients in world practice.

Keywords: heart transplant, pericardial effusion.

INTRODUCTION

Heart transplantation (HT) remains the only definitive treatment for end-stage chronic heart failure. The International Society for Heart and Lung Transplantation estimates that about 5000 heart transplants are performed annually in the world, and the number of these operations is steadily growing [1]. Russia has also witnessed a significant increase in the number of HT surgeries due to an emerging new donor and recipient selection approach, and improvements in patient management techniques [2]. In recent years, about 300 heart transplants are performed in Russia every year. The Shumakov National Medical Research Centre of Transplantology and Artificial Organs occupies a leading position among transplant centres in the world in terms of number of surgeries performed. Along with increased number of interventions, the number of perioperative complications is also growing. The most severe of them are graft rejection, graft coronary artery disease, heart rhythm disturbances, renal dysfunction, malignant tumors and infectious complications [3]. The attention of clinicians is primarily focused on these problems because they lead to significant deterioration in prognosis after surgery and in the long-term period. However, apart from the main group of complications, there are conditions that also have a high incidence, can lead to life-threatening consequences, and worsen long-term prognosis. One of such complications is pericardial effusion. This complication is typical both for patients after cardiac surgery on their own heart and for HT recipients. In the latter, the incidence of effusion is significantly higher due to different immunological and surgical components [4]. Unfortunately, to date it is impossible to unequivocally identify the causes and mechanisms of the development of this condition due to the multifactorial etiology of the process. Further study of risk factors, identification of possibilities of prevention, early diagnosis and treatment options are all necessary for prevention of adverse events in this cohort of patients.

PERICARDIAL EFFUSION AFTER CARDIAC SURGERY

Pericardial effusion is a common early postoperative complication in patients after heart surgery [4–8]. This complication is the buildup of significant amount of fluid in the space around the heart, which can affect the patient's hemodynamic indicators. The effusion can be idiopathic or result from local or systemic inflammatory reactions [6]. This complication typically manifests itself in the early postoperative period and regresses after 7–10 days. In some cases, it can persist, leading to tamponade [9]. According to most sources, the incidence of clinically significant effusion varies from 1.5 to 25%, depending on the study design and focus [4–8]. The most common causes of this condition are postcardiotomy syndrome, increased bleeding amidst anticoagulant and/or antiplatelet therapy, and lysis of pre-formed clots. Risk factors include prolonged cardiopulmonary bypass time, hypertension, renal failure, increased body surface area, young age, immunosuppression, surgery type and urgency

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[4, 8]. Pericardial effusion is usually classified according to the rate of increase in size, distribution, influence on hemodynamics and composition. The main characteristic of postoperative effusion remains its volume and distance between the parietal and visceral layers during diastole. There are mild (<10 mm), moderate (10–20 mm) and large (>20 mm) pericardial effusions [10]. The classic clinical manifestations of large effusion include the Beck's triad, described as early as 1935 [11]. It involves hypotension, increased pressure in the jugular veins, and muffled heart sounds. However, the manifestation of this triad is typical for acute, "surgical" tamponade, associated with a sharp increase in pressure in the pericardial cavity and cardiac chamber compression, which is often due to surgical complications. Tamponade appearing in the first hours after surgery is usually associated with pericardial hemorrhage, which requires repeated surgical intervention [9]. Acute symptoms also include tachycardia, severe general weakness, shortness of breath on exertion, and chest pain. Local compression symptoms such as nausea, dysphagia, hoarseness, and hiccups may appear. Nonspecific symptoms include cough, lack of appetite, and palpitations [12–14]. Fever is a nonspecific symptom that may be associated with local or systemic inflammation [15]. Pericardial friction murmur is mainly found in patients with concomitant pericarditis [16]. In most cases, the symptoms of pericardial effusion are nonspecific and there are not always classical clinical manifestations in the early stages of the process due to no cardiac chamber compression and compensation of pericardial pressure resulting from pericardial distension. For example, according to E.A. Ashikhmina et al. [4], only 42% of postoperative pericardial effusions are accompanied by hemodynamic changes, and according to P. Meurin et al. [17], in 22%, this complication is asymptomatic within 2 weeks after intervention. In this regard, early instrumental diagnostics is extremely important. Echocardiography is used as the primary diagnostic imaging, volume assessment, and hemodynamic impact. Semi-quantitative assessment of pericardial effusion consists of measuring the rim of fluid between the parietal and visceral pericardial layers by 2D echocardiography [18]. More often, effusion is defined as an echo-negative space, less often there can be adhesions, fibrin threads, or echo-positive clots, which are a sign of active or completed bleeding [19]. An important assessment criterion is to determine the localization of the effusion, including for selecting further surgical tactics. When hemodynamically significant effusion develops, EchoCG may show symptoms such as collapse of various parts of the heart, due to increased pericardial pressure, and inferior vena cava dilation, due to increased venous pressure. Collapse usually occurs at the end of diastole, primarily affecting the right heart, and is a highly sensitive and specific sign of tamponade. Inferior vena cava plethora is manifested by a <50% decrease in its diameter during full inspiration and is a highly sensitive, but nonspecific sign of tamponade [20, 21]. Although EchoCG remains the primary diagnostic tool for detecting or confirming pericardial effusion, CT and MRI should be used when echocardiographic findings are difficult to interpret or there is suspicion of localized or hemorrhagic effusion in the pericardium or thickening. CT and MRI are also used to qualitatively characterize pericardial masses detected by echocardiography [22].

One of the main objectives of modern research is to identify risk factors and predictors of pericardial effusion. According to M. Pepi et al. [5], pericardial effusion is a common complication after cardiac surgery, its frequency and nature depend on the type of intervention; oral anticoagulants are an additional risk factor for cardiac tamponade. The study was conducted on 803 patients. Most of them underwent coronary artery bypass grafting (CABG) surgery or valve replacement. Pericardial effusion was detected in 498 (64%) patients. Moderate or large effusion was found in 30 patients (3.84%). Effusion led to cardiac tamponade in 15 of them (12 took oral anticoagulants). Effusion was more often associated with CABG (75%) than with valve replacement (52%).

Unlike M. Pepi et al., researchers from the Mayo Clinic, Rochester, Minnesota [4] in their retrospective study found that all surgical interventions had a greater risk of pericardial effusion than CABG, and that heart transplantation was considered a separate risk factor for effusions. It has also been reliably proven that previous cardiac operations were associated with lower risk of effusion. The study included 21,416 patients who underwent cardiac surgery. Of these patients, 327 (1.5%) showed signs of moderate or large pericardial effusion. Classic clinical manifestations were detected only in 136 of them, and 280 had nonspecific symptoms. Independent risk factors for effusion were larger body surface area, pulmonary thromboembolism, hypertension, immunosuppression, renal failure, urgency of operation, and prolonged cardiopulmonary bypass.

M. Khassawneh et al. [6] in their study detected postoperative pericardial effusion in 235 (85%) of 335 patients. It was classified as moderate in 70 patients, and large in 15. The researchers also found that small pericardial effusions were more frequent after CABG, while moderate and large effusions were typical for patients after valve replacement. L.B. Weitzman et al. [7] studied 122 consecutive patients after cardiac surgery. One hundred and three (84%) patients had pericardial effusions after surgery. Both studies have similar conclusions that pericardial effusion is a common complication of cardiac surgery; however, most of them regress and do not cause associated complications. The researchers are convinced that patients with effusion do not require prolonged in-hospital follow-up. However,

all patients with previously identified signs of pericardial effusion require outpatient follow-up.

N.K. Khan et al. [8] analyzed the data of 1308 patients within 6 months after surgery for the presence of clinically significant pericardial effusion. The study found that 81 (6.2%) patients had clinically significant pericardial effusion, which required surgical intervention in the late postoperative period (8–87 postoperative days). Haemodynamic instability was present in 34.6% and signs of cardiac chamber compression in 54.3%. The independent risk factors in multivariable analysis were correction of valvular defects, young age and high hemoglobin levels were independent risk factors. Age 60–69 years was associated with lower risk of complications. Results from the above studies confirm the urgency of the problem of pericardial effusion after cardiac surgery and the effect of this complication in the postoperative period.

POSTPERICARDIOTOMY SYNDROME AFTER CARDIAC SURGERY

Touching upon such a topic as postoperative pericardial effusion, one cannot but mention such complication as postpericardiotomy syndrome (PPS). This complication is one of the most common in cardiac surgery [23]. PPS is the development of a systemic inflammatory response, manifested by increased body temperature, chest pain, pleural and pericardial effusions, pericardial thickening, increased C-reactive protein (CRP), pleural and pericardial friction rub. The most dangerous complications of this syndrome are tamponade and constrictive pericarditis [25]. M. Imazio et al. [26] conducted a study involving 360 patients after cardiac surgery. It was found that PPS occurs in 15% of patients during the first 3 months after the operation, 89% had pericardial effusion during the syndrome. Younger patients are more likely to develop the syndrome. J. Lehto et al. [27] found that PPS occurs more often after valve replacement than in CABG. Patients with PPS have a higher mortality rate within the first year of surgery. The primary cause of PPS is thought to be an autoimmune inflammatory response to pericardiotomy and intraoperative mechanical exposure. Unfortunately, there are yet no studies comparing the incidence of the syndrome in patients after interventions on their own heart and in heart transplant recipients. However, it is believed that heart recipients are less susceptible to this syndrome due to suppression of autoimmune factors [28]. U. Sevuk et al. [29] found that intraoperative use of methylprednisolone at a 1 mg/kg dose leads to a lower number of PPS and pericardial effusions, but the severity of effusions was greater in the group receiving methylprednisolone. The study included 200 patients after CABG, 100 of whom received methylprednisolone. A.K. Cabalka et al. [28] who studied 15 patients after heart transplantation at the age of 1 to 17 years, found that PPS was a frequent complication in this group of patients, despite ongoing immunosuppressive therapy. This complication was partially associated with cell-mediated mechanisms, as evidenced by changes in the expression of lymphocyte activation markers. Therefore, the issue of PPS development and incidence in heart transplant recipients requires further study.

PERICARDIAL EFFUSION IN HEART TRANSPLANT RECIPIENTS

The first mention of post HT-transplant pericardial effusion was described back in 1968 by Christian Barnard [30]. Unfortunately, there is still no clear understanding of the pathogenetic mechanisms of this condition due to the multifactorial nature of the process. Most pericardial effusions are known to develop in the first 3 months after HT [31–33], and their incidence in this group of patients is significantly higher than in patients after cardiac surgery on their own heart [4]. According to most sources, incidence of clinically significant effusion in patients after HT varies from 6 to 35% [31–37]. Hemodynamically significant effusions are typically characterized by moderate to large volume and are exudative contents. A peculiarity in the development of this condition in transplant recipients is the influence of several additional factors that are not encountered in patients who undergo interventions on their own heart. Various reports have shown that the occurrence and course of pericardial effusion is influenced by immunosuppressive therapy, anthropometric data of donor-recipient pair, previous cardiac surgery, use of aminocaproic acid during surgery, graft ischemia time, and graft rejection. However, data on these factors vary and there is currently no consensus regarding the main causes of pericardial effusion in patients after HT [31–37].

H.A. Valantine et al. [32] were among the first to conduct a retrospective study with a large sample, addressing the issue of pericardial effusion in patients after H.T. During 1 year, 12 of their transplant population (total, 189) developed moderate or large pericardial effusions. These effusions occurred within 1 month of transplantation in 10 patients and at 3 months and 4.5 years in the other two. Pericardiocentesis was performed because of clinical evidence of increasing effusions in 8 patients. One of the main objectives of the study was to identify the correlation between the occurrence of acute cellular rejection and development of pericardial effusion. Endomyocardial biopsy revealed moderate or severe cellular rejection in 11 out of the 12 patients as the pericardial effusion progressed. Moreover, before the manifestation of pericardial effusion, only 2 out of 12 patients had episodes of moderate rejection. These studies suggest a relationship between the development of moderate to large pericardial effusion and cardiac

transplant rejection. The clinical course and autopsy results in heart transplant recipients indicate a difference in the etiology and prognosis of pericardial effusions in this group of patients relative to patients after cardiac surgery on their own heart. G.R. Ciliberto et al. [35] also found a significant correlation between the severity of acute rejection episodes and pericardial effusion. Pericardial effusions were significantly more frequent in the group of patients with the highest frequency, duration and severity of acute rejection episodes. The study included data from 150 post-HT patients with a 1-year follow-up.

B.F. Vandenberg et al. [31] could not find a correlation between pericardial effusions after transplantation and rejection. In their study, which included 38 patients, the presence of pericardial effusion in patients after their transplantation did not demonstrate independent correlation with chest tube output after operation, cyclosporine therapy, level of blood urea nitrogen, infection, or preoperative diagnosis of idiopathic dilated cardiomyopathy. However, a combination of three factors, namely, cyclosporine therapy, acute rejection, and a preoperative diagnosis of idiopathic dilated cardiomyopathy, yielded an 86% probability of having pericardial effusion. Pericardial effusion was documented in 15 of 38 patients. Moreover, effusion volume was moderate or large in 8 patients. In 60% of patients, there was no evidence of effusion. As described by the authors, the reason for the differing data on correlation between pericardial effusion and acute rejection may be down to different research methodology.

An important factor in the study of pathology is to identify the predictors influencing further development or progression of complications. J.A. Quin et al. [33] studied the influence of 90 different perioperative factors on the development of pericardial effusions. The study included 241 HT recipients. Forty-two patients had moderate or large pericardial effusion develop, and 19 of these patients required drainage. When drainage was required, it was achieved by placement of a subxiphoid pericardiostomy tube. Pericardial effusions were significantly less likely to occur in recipients with a history of previous cardiac surgery. Patients with idiopathic dilated cardiomyopathy, younger patients with lower BMI and high central venous pressure, had a greater risk of complications. The use of hearts from female donors was associated with significant effusion in the postoperative period. Intraoperative administration of aminocaproic acid increased the likelihood of effusion approximately 6-fold. No correlation was found between acute rejection and development of pericardial effusion. Pericardial effusion was detected in 11 (26%) of 42 patients with rejection and 34 (21%) of 161 patients without graft rejection. No graft rejection was detected within 5 years after surgery in 73 ± 7% and 77 ± 3% patients with and without pericardial effusions, respectively.

P.J. Hauptman et al. [34] studied the experience of 203 heart transplants for the presence of pericardial effusion. According to the study, 18 (8.9%) of the 203 transplant recipients developed moderate to large pericardial effusions. Eight patients required pericardiocentesis, and 5 of them subsequently required pericardiectomy in connection with recurrent effusion. None of the 18 patients with significant effusions had a history of previous cardiac surgery. No postoperative pericardial effusion was revealed in 67 patients with previous intervention. In addition to the above factors, the ratio of the recipient's weight to the donor's weight was considered. It was found that in the group of patients with developed effusions, the recipient's weight on average exceeded the donor's weight by 11.9 ± 4.1 kg, while in the group of patients without this complication, the recipient's average weight exceeded the donor's weight by 2.2 ± 1.1 kg. The combination of a significantly greater recipient weight and the absence of previous cardiac surgery predisposed to the development of effusions in 83% of cases. There was no significant difference in the incidence of rejection in patients with and without pericardial effusion. Signs of rejection were found in 6 out of 18 patients with effusion. Factors such as graft ischemic time, cardiopulmonary bypass time, recipient heart size, preoperative use of mechanical circulatory support, postoperative use of anticoagulants, age, sex, and status (according to the United Network for Organ Sharing classification) of patients were not statistically significant factors in the development of pericardial effusion.

One of the most recent retrospective studies on pericardial effusions in heart transplant patients is a work by A.S. Al-Dadah et al. [37]. The study included 91 consecutive patients who underwent orthotopic heart transplantation. A total of 31 (35%) patients developed moderate to large effusions. Only 3 patients with large effusions required drainage; in all other cases the process regressed within 3 months. The only significant factor correlating with effusion was the longer graft ischemic time, which was 180 ± 59 min in the group of patients with significant effusion. According to the authors, a possible mechanism that would implicate the cause of effusion would most likely involve ischemia-reperfusion injury of the graft that would ultimately affect or involve the recipient pericardium. The authors also believe that whatever the etiology of these effusions, they tend to regress within 3 months of surgery. Between 2008 and 2012, Z. Yu et al. [36] evaluated 292 patients within the first 6 months post HT for the development of effusion. In this study, 33 (11.3%) patients developed moderate pericardial effusion. The average time to detection of pericardial effusion was 22.4 ± 18.4 postoperative days. In follow up, 78.8% had resolution of the pericardial effusion, 9.1% had no change in terms of volume and nature of the effusion at 1 year follow up, and 12.1%

had worsening of pericardial effusion requiring surgical intervention. All patients were given a trial of diuretics to reduce the effect of the pericardial effusion prior to intervention and were initiated with tacrolimus and mycophenolate as immunosuppression.

S.F. Stämpfli et al. [38] conducted a study of pericardial effusions in the long-term period after surgery. Hemodynamically irrelevant pericardial effusion unrelated to surgery was found to be a predictor of adverse outcome. Effusions detected during the first year were not included in the study; median follow-up period was 11.9 years. Of 152 patients, 25 developed pericardial effusion. The risk of death and re-hospitalization was 2.5 times higher in the group of patients with effusion than in the group without it.

Prevention of complications is certainly an important factor. With regard to pericardial effusions after transplantation, one way could be the use of prolonged drainage of the postoperative wound using a soft drain. Yun Seok Kim et al. [40] enrolled 250 patients who underwent heart transplantation between July 1999 and April 2012. They received two conventional tubes (n = 96) or two tubes with a soft drain (n = 154). At 1 month after transplantation, 69 patients (27.6%) developed significant pericardial effusion. Among these, 13 patients required surgical intervention. On postoperative day 77, only one patient with the use of a soft drain had pericardial effusion, which required pericardial drainage. According to multivariate analysis, history of previous cardiac surgery and placement of a soft drain were significant factors that prevented pericardial effusion in the postoperative period. However, the average time of prolonged drainage of the postoperative wound with the help of soft drain was 15.6 ± 6.2 days, which may affect the patient's stay in the hospital and development of postoperative wound infection, although there was no increase in these factors in this study.

Some researchers have identified an increased risk of pericardial effusion in the presence of cyclosporin A in an immunosuppressive regimen [39]. To date, this risk factor has no prognostic value since the vast majority of cases no longer use cyclosporine as a basic immunosuppressant. At present, there are no large studies examining the separate effect of modern immunosuppressive drugs on the development of pericardial effusion after heart transplantation. However, some authors point out that the use of immunosuppressants is a risk factor for postoperative effusion [4].

KEY ASPECTS OF THE TREATMENT OF POSTOPERATIVE PERICARDIAL EFFUSION

Pericardial effusions after cardiac surgery often do not manifest clinically; they are detected only on control EchoCG. Therefore, early diagnosis is extremely important and can be of key importance in the further course of this complication. In patients at risk of this complication, such as cardiac transplant recipients, a protocol for routine instrumental examination methods should be established for early and subsequent diagnosis. The treatment strategy is based on the clinical course and EchoCG picture. Moderate effusion is not an indication for surgical intervention; it requires further careful monitoring [36]. Anticoagulant and antiplatelet therapy should be adjusted, diuretic, anti-inflammatory therapy should be prescribed, if there are signs of an inflammatory process. According to the POPE study [41], non-steroidal anti-inflammatory drugs are ineffective in the treatment of moderate to severe pericardial effusions, and should not be prescribed if there are no signs of active inflammatory process due to possible side effects. Colchicine has long been included in the treatment regimen for pericardial effusion, but the POPE 2 [42] demonstrated no effect from the drug. In some cases, the clinical picture develops with the manifestation of classic signs, such as cervical vein distension, tachycardia, weakened heart tones on auscultation, and increased central venous pressure. Manifestation of these signs indicates a fulminant course of the complication and requires immediate action. For hemodynamically significant effusions leading to tamponade, surgical intervention remains the only possible solution. Ultrasound-guided pericardiocentesis is the preferred method, but it cannot always be performed due to inaccessible anatomical location of the fluid (posterior, lateral surface of the heart) or if there is insufficient distance between the pericardial layers due to increased risk of myocardial injury. The undeniable advantage of pericardiocentesis is the minimal invasiveness of the method. In the early postoperative period, the simplest method is to drain the pericardial cavity from the subxiphoid access by separating the previously applied sutures. This manipulation is easy to perform and allows evacuating pericardial effusion of any location in most cases, although it is a more traumatic procedure than pericardiocentesis [43]. To date, there are no clear criteria for choosing a particular surgical tactics for fluid evacuation in pericardial effusions. However, in most of the studies cited, the minimally invasive approach was used more often.

CONCLUSION

Pericardial effusion is one of the most common early postoperative complications in patients after cardiac surgery. The incidence of this complication is significantly higher in heart transplant recipients, although its etiology remains unclear. In the early stages of effusion development, there may be no obvious clinical signs of complication, so timely diagnosis is important. Large pericardial effusions can lead to tamponade and the only treatment for such conditions is emergency surgery. According to various sources, the risk factors

for pericardial effusion in heart transplant recipients are immunosuppressive therapy, initial diagnosis of dilated cardiomyopathy, large anthropometric parameters of the recipient, no previous cardiac surgery, acute heart transplant rejection, and longer graft ischemia time. Most authors agree that patients with identified pericardial effusion require close monitoring. Even hemodynamically insignificant effusion can be a predictor of adverse outcome. It is obvious that identification of risk factors, prevention, early diagnosis, and treatment of this condition can significantly improve the postoperative period and reduce the risks of adverse events in this patient cohort. Further study of pericardial effusions in heart recipients and the development of a clinical diagnostic protocol is a crucial task, which, if addressed, would improve outcomes in modern cardiac transplantation.

The authors declare no conflict of interest.

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