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ABSTRACT

of the dissertation for a degree Doctor of Sciences

ALGORTHMS OF LIFE SUPPORT AND SURGICAL TREATMENT OF POST-INFARCTION RUPTURES OF THE INTERVENTRICULAR SEPTUM

Specialty: 3236.01. – cardio-vascular surgery

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GENERAL CHARACTERISTICS OF THE DISSERTATION

The relevance of research. Post-infarction ventricular septal rupture (PVSD) is a life-threatening complication of acute myocardial infarction (AMI), developing within a week after the onset of AMI and accounting for 0.2-3% in the population of the latter¹. The severity of disturbances in intracardiac hemodynamics in PVSD is determined by: the size of the rupture, the volume of the shunt, the size of the infarction zone, the presence of a post-infarction aneurysm of the left ventricle, necrosis of the papillary muscles with the development of mitral valve dysfunction and an increase in pressure in the pulmonary artery to 50 -70 mm Hg. The mass of the affected myocardium in 35–40% of the total mass of the myocardium leads to the development of cardiogenic shock. The natural course of PVSD is complicated by progressive heart failure (HF) with mortality reaching 25 % in the first day after PVSD formation. by 2 weeks - 65%, by 2 months - 70-92%, by the end of the year - 93- $95\%^2$. High mortality during intervention in the early stages of the onset of AMI is due to: 1. incomplete post-infarction "heart remodeling"; 2.unstable hemodynamics / cardiogenic shock; 3.posterior localization of the PVSD; 4.early terms of intervention after the development of PVSD; 5. insufficient mechanical strength of the myocardium along the perimeter of the ventricular rupture, causing the cutting of sutures³; 6.volume of intervention, including both correction of the PVSD, coronary artery bypass grafting (CABG), and concomitant mechanical complications of AMI.

To date, controversial positions in the correction of PVSD remain:

1) Tactics of life support for the patient before correction of

¹ Pang, PY Outcome and survival analysis of surgical repair of post-infarction ventricular septal rupture / PY Pang, YK Sin, CH Lim, [et al.] // Journal of Cardiothoracic Surgery, – 2013. 8, – p. 44.

 $^{^2}$ Jones, BM Ventricular septal rupture complicating acute myocardial infarction: a contemporary review / BM Jones, SR Kapadia, NG Smedira [et al.] // European Heart Journal, -2014.~35~(31), -p.~2060–2068.

 $^{^3}$ Deja, MA Post infarction ventricular septal defect - can we do better? /MA Deja, J. Szostek, K. Widenka [et al.] // European Journal of Cardio-Thoracic Surgery, -2000. 18 (2), -p. 194–201.

the rupture. To date, there is no consensus on the advisability of using mechanical hemodynamic support and endovascular occlusion of the ventricular ruprure⁴. In 65-100% of patients with PVSD, the use of IABP is indicated. The latter helps to stabilize hemodynamics, allowing you to choose the optimal time for surgical intervention. However, in some cases, IABP is not able to stabilize the patient's condition, requiring the usage of extracorporeal membrane oxygenation (ECMO). From 2006 to 2011 ECMO utilization rate in the US increased by 433%⁵. Endovascular correction of rupture can be used as an independent treatment method; as temporary hemodynamic support followed by surgery; when correcting residual IVS defects after surgical correction, accounting for 13-40% of cases⁶. According to the 3 largest series, which included 12; 12. and 29 observations of endovascular occlusion of the PVSD, the effectiveness of interventions was 83-86% with hospital mortality 42-65%; Recanalization of patches was 8.3-13.8%^{6,7}. Analysis of long-term outcomes of endovascular occlusion of the PVSD and comparing them with the results of surgical correction requires further research⁸.

2) Justification for surgical correction in case of unstable hemodynamics and timing of surgery in case of stable hemodynamics.

Determining the optimal timing of surgical intervention and the advisability of performing it in the early stages after the development of

 $^{^4}$ Poulsen, SH Ventricular septal rupture complicating acute myocardial infarction: clinical characteristics and contemporary outcome / SH Poulsen, M. Praestholm, K. Munk [et al.] // The Annals of Thoracic Surgery, $-2008.\,85$ (5).

⁵ Sauer CM, Yuh DD, Bonde P. Extracorporeal membrane oxygenation use has increased by 433% in adults in the United States from 2006 to 2011. ASAIO J. 2015 Jan-Feb;61(1):31-6.

 $^{^6}$ Thiele, H. Immediate primary transcatheter closure of postinfarction ventricular septal defects / H. Thiele, C. Kaulfersch, I. Daehnert [et al.] // European Heart Journal, $-2009.\ 30\ (1), -p.\ 81-88.$

 $^{^7}$ Maltais, S. Postinfarction ventricular septal defects: towards a new treatment algorithm? / S. Maltais, R. Ibrahim, AJ Basmadjian [et al.] // The Annals of Thoracic Surgery, $-2009.\ 87\ (3), -p.\ 687-692.$

⁸ Kopitsa, N.P. Mechanical complications of acute myocardial infarction / N.P. Kopitsa, A.N. Abolmasov, E.I. Litvin [and others] // – Kharkov: Ukrainian therapeutic journal, -2013. No. 1, -p. 108–113.

PVSD is fundamental. In those operated on during hospitalization \leq 24 hours, the mortality rate was 87.5%⁹; in those operated on at gestational age \leq 7 days - 54.1%; in those operated on for periods >7 days - 18.4%¹⁰. Indications for the timing of surgery have undergone significant changes in 2014-2018. If the recommendations of the American Heart Association (ACC) from 2012. pointed out the need for early surgical intervention, then in the ACC recommendations of 2013. better inhospital outcomes have been reported with delayed interventions¹¹. The advisability of early or delayed correction of PVSD remains a discrete option.

3) Optimal surgical tactics include: choice of access to the PVSD; scope of surgery, including CABG and correction of concomitant mechanical complications of AMI¹². To this day, the role of simultaneous myocardial revascularization in the correction of PVSD remains unclear. Several studies have suggested that concomitant CABG confers improved long-term survival, but this has not been reliably reported in current guidelines¹³.

Takahashi et al. (2015)¹⁴ identified the inferiority of myocardial

⁹ Papalexopoulou, N., Young, CP, Attia, RQ What is the best timing of surgery in patients with post-infarct ventricular septal rupture? // Interactive Cardiovascular and Thoracic Surgery, -2013. 16 (2), -p. 193–196.

¹⁰ Arnaoutakis, GJ Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database / GJ Arnaoutakis, Y. Zhao, TJ George [et al.] // The Annals of Thoracic Surgery, – 2012. 94 (2), – p. 436–444.

¹¹ Nair R, Subbaiyan K, Rm K, Mani R, Kathamuthu B. Protocol-based Surgical Intervention to Manage Ventricular Septal Rupture from a Tier Two City. Braz J Cardiovasc Surg. 2023 May 4;38(3):331-337.

¹² Lundblad, R. Surgical repair of postinfarction ventricular septal rupture: risk factors of early and late death / R. Lundblad, M. Abdelnoor, OR Geiran [et al.] // The Journal of Thoracic and Cardiovascular Surgery, – 2009. 137 (4), – p. 862–868.

¹³ Malhotra A, Patel K, Sharma P, Wadhawa V, Madan T, Khandeparkar J, Shah K, Patel S. Techniques, Timing & Prognosis of Post Infarct Ventricular Septal Repair: a Re-look at Old Dogmas. Braz J Cardiovasc Surg. 2017 May-Jun;32(3):147-155.

¹⁴ Takahashi, H. Long-term results after surgical treatment of postinfarction ventricular septal rupture / H. Takahashi, R. Arif, A. Almashhoor, [et al.] // European Journal of Cardio-Thoracic Surgery, – 2015. 47 (4), – p. 720–724.

revascularization as the leading predictor of 30-day mortality. Huang at al¹⁵ noted the positive impact of complete revascularization on long-term survival rates in patients. The same uncertainty concerns the choice of access to PVSD. Despite the widespread use method "exclusion of infarction" when correcting PVSD, residual shunts remain the main and complications, accounting for 10-25%. Labrousse et al. did not reveal any residual defects in a series of 37 patients operated on using double patches layered with glue, whereas when using one patch, in 11% of observations residual shunts were noted¹⁶.

5) The influence of PVSD localization on intervention outcomes. Preoperative determination of the localization of the PVSD predetermines tactics for correcting anterior and posterior defects. An analysis of the localization of MI in groups of patients with PVSD showed that, despite the larger area of MI in anterior ruptures, posterior PVSD are more likely to be a predictor of hospital complications and mortality¹⁷. Thus, data regarding the localization of PVSD, the characteristics of the morphology of the defect, its spatial geometry, and damage to adjacent structures of the heart are of great practical importance in predicting and determining the scope and tactics of surgical treatment.

Goal of the work. Development of an algorithm for the tactics of preoperative management of patients with PVSD, depending on the clinical status that determines the timing of intervention.

Research objectives:

1. Systematize options for life support tactics for patients with

¹⁵ Huang SM, Huang SC, Wang CH, Wu IH, Chi NH, Yu HY, Hsu RB, Chang CI, Wang SS, Chen YS. Risk factors and outcome analysis after surgical management of ventricular septal rupture complicating acute myocardial infarction: a retrospective analysis. J Cardiothorac Surg. 2015 May 4;10:66. doi:10.1186/s13019-015-0265-2. PMID: 25935413; PMCID: PMC4426168.

 $^{^{16}}$ Labrousse, L. Surgery for post infarction ventricular septal defect (VSD): risk factors for hospital death and long term results / L. Labrousse, E. Choukroun, J. M. Chevalier [et al.] // European Journal of Cardio-Thoracic Surgery, -2002. 21 (4), -p. 725–731.

¹⁷ Cummings, RG Correlates of survival in patients with postinfarction ventricular septal defect / RG Cummings, R. Califf, RN [et al.] // The Annals of Thoracic Surgery. 1989. 47 (6), – p. 824–830.

PVSD depending on the clinical status of the patient;

2. To study the outcomes of endovascular correction of PVSD and the optimal time for the operation after stabilization of hemodynamics;

3. Compare hospital outcomes of operations depending on the timing of the intervention, its volume and localization of the PVSD;

4. Identify the leading predictors of the risk of in-hospital and 30day mortality after surgical correction of PVSD;

5. To evaluate the effectiveness of connecting an IABP on hospital outcomes of operations in patients with PVSD.

Basic provisions for defense:

1. To what extent does PVSD correction delayed up to 2 weeks after AMI, provided that patients are hemodynamically stable, help reduce in-hospital mortality and complications;

2. Are the presence of chronic renal failure (CRF), diabetes and cardiogenic shock independent predictors of 30-day mortality in patients after correction of PVSD;

3. Is it right to consider the localization of the PVSD as an independent predictor of the outcome of surgical intervention;

4. Is it advisable to consider endovascular closure of the PVSD an independent alternative to surgical correction, or a stage of hybrid intervention.

Scientific novelty of the research

For the first time, an algorithm for the tactics of preoperative management of patients with PVSD has been devloped, taking into account the clinical status that determines the timing of intervention.

Determined the feasibility of wait-and-see surgical treatment of patients was depending on the time period after the development of AMI and the location of the IVS rupture.

The feasibility of endovascular occlusion of the PVSD as a stage of hybrid intervention has been proven.

Based on a study of the postoperative prognosis, the optimal timing of surgical correction of PVSD after AMI was substantiated.

Determined a group of independent predictors of 30-day mortality in patients with PVSD.

The localization of PVSD is not a reliable predictor of lethal outcomes of cardiac origin.

Practical significance of the research.

1. The feasibility of endovascular correction of PVSD in patients with unstable hemodynamics has been systematized;

2. The earliest possible connection of an IABP in case of unstable hemodynamics/shock, refractory to inotropic support, allows early intervention against the background of unstable hemodynamics, or its delay after stabilization of the latter;

3. Hospital outcomes of PVSD correction are determined, firstly, by the clinical status of patients, and secondly, by the timing, volume of operations and localization of ruptures;

4. Isolated correction of anterior VSD with resection of infarcted myocardium and preservation of LV geometry reduces the risk of postoperative formation of LV aneurysm;

5. Long-term outcomes of operations depend on the completeness of myocardial revascularization;

Reliability of conclusions and recommendations. Compliance with the principles of good clinical practice when carrying out this dissertation research, use in the research of the results obtained on modern clinical and laboratory equipment, as well as a comprehensive approach to scientific analysis (sufficient research power and sample size (90 patients), use of modern methods of statistical data processing) and advanced software necessary to carry out this research are evidence of the reliability of the results obtained, correctly formed conclusions and practical recommendations of this dissertation o research.

Implementation of research results into practice. The results of the dissertation research were introduced into the clinical practice of the Central Clinical Hospital, the Central Hospital of Oil Workers, the Scientific Center of Surgery named after. M.A. Topchibashev Ministry of Health of the Republic of Azerbaijan and can be used in other cardiac surgery centers. Based on the implementation of the research results in the above centers, implementation certificates were obtained.

Statistical processing.

The research results were processed by statistical methods after creating a database in Microsoft Excel 2016. Statistical calculations were carried out using the SPSS 17.0 for Windows. Univariate and multiple logistic regression analyzes were performed using GraphPad

PRISM 9.0 software.

Based on the dissertation materials, 25 works were published, of which 7 thesis reports at international conferences and 18 journal articles, of which 12 articles in leading scientific publications recommended by the Higher Attestation Commission of the Republic of Azerbaijan (including 12 in journals indexed in international scientometric databases (11 in the SCOPUS database, 3 in the Web database of Science)).

The main provisions of the dissertation work were reported and discussed:

- International scientific and practical conference, Lublin, Poland. 2021) DOI https://doi.org/10.30525/978-9934-26-038-4-2.

- 70th International Congress of the European Society of CardioVascular Surgeons (ESCVS), June 20-23, 2022. Liège, Belgium. //Aorta J. 2022; v. 10 (Suppl.01).

- 17th Congress of the Turkish Society of Cardiovascular Surgery (TKDCD), Nov. 17-20, 2022. Antalya, Turkey;

- 18th International Congress of Update in Cardiology and Cardiovascular Surgery (UCCVS), Dec.01-04,2022. Antalya, Turkey;

- International scientific and practical conference. "Svitova medicine: current trends and development factors." January 29-30, 2021 Lviv. Ukraine

- international conference "7th BAKU HEART DAYS" June 2019 Baku. Azerbaijan;

- at a joint scientific conference of employees of the Department of Heart Surgery, the Center for Pediatric Cardiac Surgery, the Department of Anesthesiology and Reanimation of the Scientific Center for Surgery named after. M.A. Topchibasheva; Department of Heart Surgery of the Central Clinical Hospital; Department of Cardiac Surgery of the Russian Children's Center; Department of Heart and Vascular Diseases of the Central Hospital of Oil Workers; Department of Heart Surgery of the Central Hospital of the Ministry of National Security of the Republic of Azerbaijan in 2023.

- at a joint meeting of departments and laboratory diagnostic units of the Scientific Center for Surgery named after. M.A.Topchibasheva 06/05/2023.

Structure and volume of the dissertation. The dissertation work is presented in the form of a prepared manuscript. The dissertation is presented on 312 pages (412 662 characters) of typewritten text. Consists of an introduction, literature review, materials and research methods, 3 chapters of own research, discussion of results, conclusions, practical recommendations, list of references. The list of references includes 177 modern sources: 19 in Cyrillic, 158 in Latin. The work is illustrated with 43 figures and 66 tables. The work passed the stages of ethical and scientific examination and was approved at a meeting of the Scientific Council of the Scientific Center for Surgery named after M.A. Topchibashev (Baku, Azerbaijan) (protocol No. 1, June 5, 2023). Approbation of the dissertation was carried out at a scientific seminar operating under the dissertation council BED 1.12 (protocol No. 1, March 1, 2024).

MATERIAL AND METHODS OF RESEARCH

The paper presents a retrospective analysis of data from 90 patients with ischemic heart disease complicated by PVSD, hospitalized for surgery for the period 2002-2019. 12 patients with PVSD were hospitalized at the Central Clinical Hospital Baku, 78 patients - at the State Institution "National Institute of Cardiovascular Surgery named after N. M. Amosov National Academy of Medical Sciences of Ukraine". The work was carried out in accordance with the agreement on joint scientific cooperation of the Central Clinical Hospital Baku and State Institution "National Institute of Cardiovascular Surgery named after N. M. Amosov National Academy of Medical Sciences of Ukraine". The ages of hospitalized patients ranged from 29 to 81 years.

The majority of hospitalized patients with PVSD were over 60 years of age (Table 1). At the same time, among hospitalized patients with PVSD who were scheduled for surgical intervention, 17.8% (n = 16) were over 70 years of age (11 men and 5 women). The data presented in Table 1 indicate the prevalence of male patients (72.3% men and 27.7% women). P age distribution corresponded to the normal distribution law (60.0 ± 9.6 years; Z = 0.729, p = 0.663).

Table 1

	Number patients Men		Women
Age (years)			N=25
18-29	1	1 (1.1%)	0
30-44	4	4(4.4%)	0
45-59	35	28(31.2%)	7(7.7%)
60-74	45	30(33.4%)	15(16.7%)
75-89	5	2(2.2%)	3(3.3%)
≥90	0	0	0
Total	90	65	25

Distribution of patients with PVSD, by age and gender, n /%

Designations: PVSD – post-infarction rupture of the interventricular septum.

The exact timing of the development of PVSD was diagnosed in 23 patients by comparing anamnestic data with ECHO-CG. PVSD within the first 24 hours after the onset of AMI was observed in 10 patients; within a period of 2 to 3 days – in 6; from 4 to 7 days – in 3; from 8 to 29 days – in 4 patients. The time period from the development of AMI to the occurrence of IVS rupture varied from 1 to 29 days (n = 23; Mo = 1; Me = 3 ($Q_1 = 1$; $Q_3 = 6$)). The duration of hospitalization of patients with PVSD from the development of AMI varied from 1 to 462 days. The distribution of patients by length of hospitalization after AMI did not correspond to the law of normal distribution (Z =1.967, p =0.001), and the descriptive characteristics of the patient sample were as follows: n=90; Mo = 1; Me = 35.0 ($Q_1 =$ =15.5; $Q_3 = 72.0$). (Fig. 1.)

The data presented in Figure 1 illustrates that the largest number of hospitalizations of patients with PVSD (39 people) occurred 1 month after the development of AMI. All patients with PVSD were divided into three groups depending on the timing of the performed or expected surgical intervention after AMI:

Group I consisted of 28 patients with PVSD who underwent surgery within 1 to 28 days (14.0 \pm 8.6) after AMI (Z =0.614, p =0.846).

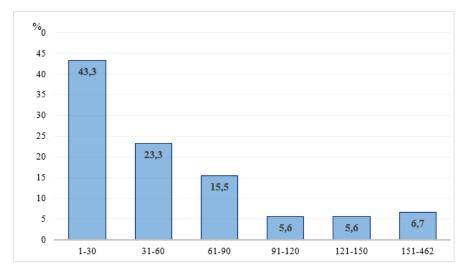


Figure 1. Distribution of patients with PVSD depending on the duration of hospitalization after acute myocardial infarction (days)

II - 26 patients with PVSD who underwent surgical intervention within 29 to 56 days (42.1 \pm 7.5) after AMI (Z =0.760, p =0.610).

Group III – 36 patients who were operated on at \geq 57 days (120.6 ± 80.1) after AMI (Z = 1.312, p = 0.064).

The first group (n = 28) consisted of 21 male patients and 7 female patients; second (n = 26) - 19 men and 7 women; third group (n = 36) 25 men and 11 women. The distribution of patients with PVSD in the study groups by gender is presented in Figure 2.

Intergroup analysis did not reveal statistically significant gender differences between them χ^2 (2, n=90)= 0.256; p= 0.880 showing that all groups are quite homogeneous in terms of this characteristic, and the differences obtained later are not associated with the peculiarities of the percentage representation of female and male patients in each of the groups (Fig. 2; Table 2).

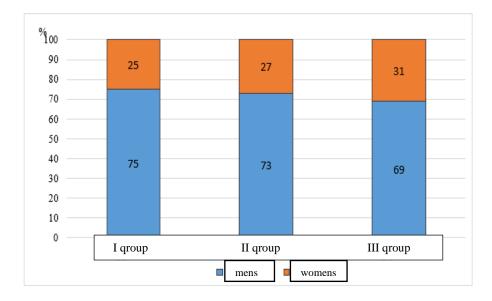


Figure 2. Distribution of patients with PVSD in the study groups by gender (in percentage)

Table 2

Distribution of patients with PVSD in the study groups by gender

Floor	Group 1	Group 2	Group 3
Men	21	19	25
Women	7	7	eleven
Total	28	26	36

The age of patients with PVSD in group 1 ranged from 44 to 81 years. (61.4 ± 9.4) (Z = 0.452, p =0.987); Group 2 - 29 - 79 years old (58.9 ± 12.5) (Z = 0.764, p =0.604); Group 3 - 45 - 76 years (59.7 ± 7.3) (Z = 0.779, p =0.579) (H = 0.896 (2, n =90), p =0.639). The distribution of patients with PVSD in the study groups by age is presented in Table 3.

Table 3

by age, n/%.						
	Group 1		Group 2		Group 3	
Age (years)	n	%	n	%	n	%
18-29	0	0	1	3.9	0	0
30-44	1	3.6	3	11.5	0	0
45-59	9	32.1	6	23.1	20	55.6
60-74	17	60.7	14	53.8	14	38.8
75-89	1	3.6	2	7.7	2	5.6
90 and above	0	0	0	0	0	0
Total	28	100	26	100	36	100

Distribution of patients with PVSD in the study groups by aga n^{10}

Designations: PVM - post-infarction rupture of the interventricular septum.

Despite the fact that in groups I and II patients aged 60-74 years prevailed, and in group 3 the largest number of patients were operated on at the age of 45-59 years (Table 3), no statistically significant age differences were found between the groups (χ^2 (2, n=90)=3.041; p=0.219). In order to normalize the studied groups of patients with PVSD, the influence of individual risk factors for the development of coronary artery disease was determined, which made it possible to more correctly compare them (Table 4). O burdened heredity was revealed only in 2 patients (7.7%) of group 2 and 2 patients (5.6%) of group 3. When analyzing risk factors for the development of coronary artery disease in patients of the study groups, no statistically significant differences were revealed in body mass index (more than 24.9) χ^2 (2, n=90)=1.803; p=0.406, blood cholesterol level $\chi^2(2, n=90) = 4.402$; p=0.111, blood glucose $\chi^2(2, n=90) = 4.402$; p=0.1111, blood glucose $\chi^2($ n=90 = 3.137; p=0.208 and the presence of arterial hypertension χ^2 (2, n=90)=0.480; p=0.786. At the same time, statistically significant differences in the level of blood triglycerides were revealed in patients of the study groups $\chi^2(2, n=90) = 6.800$; p=0.033, which may be due to individual characteristics of taking statins. The latter is supported by the higher level of triglycerides in group 1, due to the shorter duration of statin therapy after AMI (Table 4). Concomitant pathology was represented by: endocrine disorders in 2(7.1%) patients of group 1 and 1(3.8%) patient of group 2; history of acute cerebrovascular accident - in 2(5.6%) patients of group 3; the presence of encephalopathy was diagnosed in 1(3.8%) patient of group 2 and 1 (2.8%) patient of group 3. And insulin therapy to correct hyperglycemia was carried out in 2(7.1%) patients of group 1; 3(11.5%) patients of group 2 and 1 (2.8%) patient of group 3.

Table 4

Risk factors	I (n=28)	II (n=26)	III (n=36)
Smoking	8/28.6	3/11.5	4/11.1
BMI(Kg/m2 ⁾ (>24.9)	20/71.4	14/53.8	23/63.9
Cholesterol, mmol/l (>5)	19/67.9	13/50.0	15/41.7
Glucose, mmol/l (>6.1)	8/28.6	5/19.2	4/11.1
Triglycerides, mmol/l (>1.82)	19/67.9	11/42.3	13/36.1
AH	23/82.1	20/76.9	27/75.0

Demographics of 90 patients with PVSD

Designations: BMI - body mass index; AH-arterial hypertension.

When analyzing concomitant pathology in patients of the study groups, no statistically significant differences were revealed in the presence of pathology of the genitourinary system χ^2 (1, n=64)=2.102; p=0.147, SD χ^2 (2, n=90)=3.289; p=0.193, gastrointestinal pathologies χ^2 (2, n=90)=0.797; p=0.671. At the same time, statistically significant differences in the presence of stage IIIb - I V chronic renal failure were revealed in patients of the study groups. and multifocal ATS, which was most common in patients of group 3. There were no statistically significant differences between the groups in the presence of pathology of the respiratory system (Table 5).

Table 5

0.005

0.007

0.677

4/11.1

12/33.3

6/16.7

in patients with PVSD, n / %.						
Concomitant pathology	Group 1 (n=28)	Group 2 (n=26)	Group 3 (n=36)	FFHt, p		

1/3.8

4/15.4

2/7.7

10/35.7

1/3.6

4/14.3

Chronic renal failure

Pathology respiratory

IIIb - I V stage.

Multifocal ATS

system

Frequency of detection of concomitant pathology

Designations: PVSD – post-infarction rupture of the interventricular septum; ATS - atherosclerosis.

The high incidence of stage IIIb-IV chronic renal failure was noted. in patients of group 1, acting as a predictor of the complicated clinical course of PVSD and hospital mortality (Table 5). Intergroup analysis of the frequency and number of AMI in the anamnesis in patients with PVSD (Fig. 3) showed that the development of PVSD was more often noted after a previous first AMI in the medical history, amounting to 27.8% (n = 25), 27.8% (n=25) and 38.8% (n=35) observations in patients of the first, second and third groups, respectively. A history of several AMIs was observed in 3 patients of group 1, 1 patient of group 2, and 1 patient of group 3. Analysis of the frequency and number of AMI in the history of patients of the first, second and third groups did not reveal statistically significant differences between them (p=0.509). The latter gives reason to believe that these groups are quite homogeneous in terms of this characteristic, and discrepancies in the percentage of patients with different numbers of suffered AMI are not significant (Fig. 3).

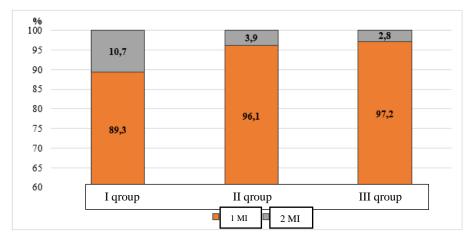


Figure 3. Frequency and number of myocardial infarctions in patients in the study groups in percentage (MI – myocardial infarction)

Table 6

Distribution of patients with PVSD according to the history of myocardial reperfusion tactics, n / %.

Myocardial reperfusion option	I (n=28)	II group(n=26)	III (n=36)
PCI	8/ 28.6%	-	3/ 8.3%
Isolated thrombolysis	-	2/ 7.7%	1/ 2.8%

Designations: PVSD – post-infarction rupture of the interventricular septum; PCI - percutaneous coronary intervention.

Table 6 shows that in each of the groups, patients who did not undergo myocardial reperfusion predominated. The analysis using the Fisher test suggests that there are no statistically significant differences between the groups of patients with PVSD who underwent myocardial reperfusion (p=0.103). At the same time, in group 1, 28.6% of patients with a history of myocardial reperfusion were noted, which may indicate a severe course of coronary artery disease with the development of cardiogenic shock in 87.5% of patients and the need for emergency myocardial revascularization.

Among patients who underwent myocardial reperfusion using PCI (n=11), blood flow was restored in 55% of patients with RCA lesions (55%). Among the 8 prosthetized patients of group 1, 5 patients had RCA lesions, 2 had LAD lesions, 1 patient had LAD and RCA lesions requiring emergency PCI when AMI developed. In patients of group 3 who had a history of emergency PCI, the cause of AMI in 1 case was damage to the RCA; in one lesion of the LAD; in one - LAD and RCA (Fig. 4). Pay attention predominance of anterior localization of PVSD (n=51) than posterior ones (n=39) (Table 7).

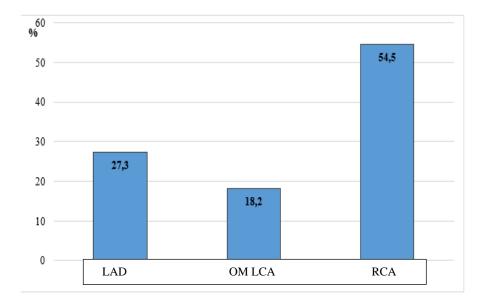


Figure 4. Areas of coronary lesions in patients with PVSD requiring PCI. Designations: (LAD – Left Anterior Descending artery; OM LCA – Obtuse Margin branch of Left Coronary Artery; RCA - right coronary artery)

Table 7

Distribution of patients with PVSD by rupture location n/%.

Localization of PVSD	Group I (n=28)	Group II (n=26)	III group (n=36)	χ^2 , p
Anterior	13/46.4%	17/ 65.4%	21/ 58.3%	χ^2 (2, n=90)
Posterior	15/ 53.6%	9/ 34.6%	15/ 41.7%	= 2.041; p=0.361

Designations: PVSD – post-infarction rupture of the interventricular septum.

Among 90 patients with PVSD, intervention was performed in 87 patients (96.7%); one patient did not survive to the scheduled operation; two patients refused surgery. Surgical treatment of PVSD (rupture repair) was performed in 84 patients, including 4 patients after occluder implantation. Endovascular Occlusion of the PVSD was used in 7 patients (7.8%), three of whom underwent isolated EVO; four – EVO followed by surgery correction. 79 patients (94.0%) out of 84 patients who underwent PVSD surgery required CABG, 1 (1.2%) required mitral valve replacement; 73 (86.9 %) patients underwent aneurysmectomy. 36 (42.9%) patients had a posterior aneurysm, and 48 (57.1%) had an anterior aneurysm. In 46 (54.8%) patients, aneurysm correction was performed according to W. M. Dagget, 35 (41.7%) according to T. E. David, 7 (8.3%) in V. Dor.

48 (57.1%) patients were operated on for anterior PVSD; 36(42.9%) - regarding posterior PVSD. 9 (10%) patients were operated on with multiple PVSD; alone sick - with with a clear rupture of the free wall of the left ventricle and the interventricular septum. Access to the PVSD was carried out through the infarction zone of the LV or right atrium in 97.6% (n = 82) and 2.4% of patients (n = 2), respectively. Depending on the location of the MI, the incision was made at a distance of 2-3 cm from the left anterior descending coronary artery or the posterior descending coronary artery. Plastic surgery of the prostate esophagus was performed using the concept (*defect exclusion technique*) Correction of the IVS defect was carried out by suturing an autopericardial flap, a xenograft (bovine pericardium) or an artificial prosthesis (Dacron or Gore-Te x). Plastic surgery of LV aneurysm according to W. M. Dagget was performed in 54.8% of patients (n = 46), according to T. E. David – in 36.9% of patients (n = 31). In 8.3% of patients (n = 7) with apical VSD, closure of the aneurysm and IVS defect was performed according to V. Dor.

Research methods.

All information about patients was entered into a computer database developed and adapted for this study by the dissertation student in Microsoft Excel. The database contains the following information: passport and anthropometric data, medical history, data from clinical, laboratory and instrumental research methods, features of drug and surgical treatment. All patients with PVSD were examined in accordance with the existing standard protocol, including the collection of anamnestic data, generally accepted clinical and laboratory methods (general clinical blood count, biochemical blood tests), ECG, ECHO-CG, chest radiography, spirometry (pulmonary function), CT and MRI. When collecting anamnesis, the presence of pain syndrome, hypertension, the number of AMIs in the anamnesis, the time of formation of PVSD from the development of AMI, and other concomitant pathologies were recorded. The patients' condition was assessed by exercise tolerance. When determining individual risk factors for coronary artery disease, the presence of a burdened family history was assessed - the fact of confirmed pathology of the cardiovascular system in a first-degree relative. FC of chronic HF was determined according to the classification (Killip).

In order to prevent postoperative complications from the gastrointestinal tract, including in the absence of complaints, 59 (65.5%) patients with PVSD underwent routine fibrogastroduodenoscopy before surgery.

When conducting ECHO-CG, special attention was paid to the presence of signs of volume overload of the LV, pulmonary hypertension and analysis of the morphostructures of the LVAD: the number of defects (single or multiple), localization (anterior or

posterior) and size of the defect.

All patients underwent radiography of the chest organs in a direct projection using the Neodiagnosticum apparatus (Hungary) under teleradiography conditions at a distance of 165 cm from the tube to the patient.

Before surgery, ultrasound was performed dopplerography of the arteries of the lower extremities and duplex scanning of the brachiocephalic vessels in patients over 60 years of age.

The presence of multifocal ATS was diagnosed when a lesion was detected with a decrease in the diameter of the vessel lumen by 50% or more in at least one peripheral artery (brachiocephalic vessels and/or arteries of the lower extremities).

At the planning stage of surgical correction, if multiple IVS defects were suspected, as well as for the purpose of their visualization, 4.4% of patients (n = 4) underwent MRI.

MRİ was performed using a "GE Lightspeed" machine. Using this method, the sizes of all cavities of the heart and great vessels, the thickness of the walls of the LV and IVS, the presence and number of IVS defects, their sizes and localization were determined. LVEF was calculated separately. When examining coronary arteries, their diameter, the presence of atherosclerotic lesions and calcification were described.

To confirm the diagnosis of IHD, as well as to determine the localization and severity of coronary ATS, the number of affected arteries, and assess the functional state of the LV myocardium, all patients underwent coronary artery angography (CAG).

Ventriculography was performed in the right oblique projection. In addition, in order to assess the contractility of the IVS, blood discharge into the RV and the size of the anterior apical aneurysms of the LV, ventriculography was performed in the left lateral projection. In the case of visual assessment of disorders of general contractility of the LV, the classification of MV Herman et was used. al (1974).

Selective coronary angiography was performed by isolated contrasting of the right and left coronary arteries. At the same time, the type of coronary circulation, the number of affected arteries, the localization and severity of ATS were assessed. Determination of the type of coronary blood circulation of the heart was based on identifying the coronary artery, which primarily supplies blood to the posterior LV walls.

Based on the results of coronary angiography, indications for CABG surgery and myocardial revascularization plan.

The endovascular method of PVSD correction (occluder implantation) was used in 7 (7.8%) patients hospitalized for surgery. Transcatheter closure of the VSD using an occluder was performed using a standard technique using access through the femoral vein. Implantation of an IABP, with the aim of temporarily stabilizing systemic hemodynamics, allowing to delay surgical intervention in patients with signs of cardiogenic shock, was carried out according to a standard technique using a transfemoral approach.

The level of serum enzymes (creatine phosphokinase and its MB fraction), troponin I was determined on the 2nd day of the postoperative period and subsequently according to indications (in case of ischemic changes on the ECG). The diagnosis of perioperative MI was established according to clinical and laboratory data:

• an increase in the level of troponin I, creatine phosphokinase and its MB fraction by 10 or more times from the initial preoperative value;

• the appearance on the ECG of a new pathological Q wave or a sharp decrease in the R wave in at least two adjacent chest or main leads, not caused by postoperative rotation of the heart;

• emergence of new disorders of LV contractility according to ECHO-CG data. The severity of the postoperative period was assessed taking into account the following characteristics: length of stay in the ICU, duration of artificial ventilation, the need for the use of temporary mechanical hemodynamic support devices (IABP, ECMO) and their support (sympathomimetics), the presence duration, drug of postoperative complications, structure frequency, their and postoperative survival period.

In the postoperative period, the levels of plasma protein, glucose, creatinine, functional indicators of liver failure, bilirubin and urea levels, and blood ion composition (potassium, sodium, magnesium) were monitored throughout the patient's stay in the ICU.

Analysis of data.

As a result of the research, data was obtained reflecting the qualitative and quantitative characteristics of individual indicators of the analyzed samples of patients. In order to identify differences between the studied samples and the level of their reliability, data from both groups (both qualitative and quantitative) were subjected to statistical processing.

Data related to qualitative indicators were analyzed using various nonparametric criteria. Pearson's goodness-of-fit test (χ^2 test) was used to analyze four-field or multi-field tables. *Yates* Amendment *correction*) was used to assess the statistical significance of differences between groups in the absence of a 20% excess of cells with a value ≤ 5 . Otherwise, Fisher's Exact Test (Ft) for four-field tables and Fisher's extended test were used to check the homogeneity of the contingency table (*Fisher – Freeman – Halton test (FFHt*)) for multifield tables. When using the Pearson goodness-of-fit test, the results of statistical calculations were presented in the format χ^2 (*df* - degrees of freedom, total sample size (n)), χ^2 value, statistical significance (p). When using Fisher's exact test (*Ft*) or extended Fisher's test (*FFHt*), only the statistical significance of the results obtained (p) was presented in the work.

In order to determine the possibility of using parametric and nonparametric methods of statistical analysis to identify the reliability of differences in groups of quantitative data, the nature of the distribution of numerical values of individual indicators was determined using graphical analysis and the Kolmogorov-Smirnov goodness-of-fit test (Z) with the Lilliefors correction (Kolmogorov-Smirnov (Lillefors) test). If the number of variants in the variable series did not exceed 50, then the Shapiro-Wilk Test was used to assess the distribution. W). When using the Kolmogorov-Smirnov goodness-of-fit test with the Lilliefors correction, the results of statistical calculations were presented in the format Z value, p, and when using the Shapiro-Wilk test - value W, p. Parametric methods of statistical analysis were applied to those series of variants in which there were no statistically significant differences with a normal distribution. If there were statistically significant differences between the variant and normal distributions (p < 0.05), we used nonparametric statistical criteria for assessing quantitative

parameters.

In the absence of statistically significant differences from the normal distribution, the descriptive characteristics of the variable series included: mean value, standard deviation and level of statistical significance ($M \pm S$ D). In the presence of statistically significant differences from the normal distribution, the descriptive characteristics of the variable series included: mode, median, 1st and 3rd quartiles of the data sample (*Mo, Me, Q*₁, *Q*₃).

To determine compliance with the null hypothesis (*H0*) of the subjects two groups of data that have a quantitative expression of a characteristic with a variant distribution that is statistically significantly different from the normal distribution, *the Mann* – Whitney *U test* was used *U-test*). When using this criterion, data were presented in the format *U value*, test value *Z*, statistical significance (p). In case of significant limitation of data categories, the two-sample Kolmogorov–Smirnov test was additionally used (*Kolmogorov–Smirnov for two independent samples* (*Z*, *p*)). To determine the correspondence of *H0* to three groups of data that have a quantitative expression of a trait with a variant distribution, the Kruskal-Wallis *H test was used*. When using this criterion, data were presented in the format *H value* (*df* - degrees of freedom, total sample size (n)), statistical significance (p).

To determine compliance with the null hypothesis (*H0*) of the two groups of data under study, which have a quantitative expression of a characteristic with a variant distribution that is not statistically significantly different from the normal distribution, $a \ t$ -test for unrelated data groups was used. When using it, data were presented in *df format* – degrees of freedom, t -test result (t), statistical significance (p). To determine the correspondence of *H0* to three groups of data that have a quantitative expression of a trait with a variant distribution that is not statistically significantly different from the normal distribution, ANOVA analysis of variance was used. When using it, data were presented in the format of Fisher's test value ($F \ -test$, F), statistical significance (p).

Comparison of two dependent samples (pre- and postoperative periods) was performed using the nonparametric Wilcoxon T test

(*Wilcoxon test, T*). When using this criterion, data were presented in the format *T value*, test value *Z*, statistical significance (p).

Odds ratios (*OR*) with 95% confidence intervals (*CI*) and their pvalues to identify predictors of 30-day mortality were performed using univariate logistic regression analysis. Continuous variables affecting mortality were converted to a binary value with a cutoff point determined by receiver *operating characteristic* (*ROC*) analysis to calculate the area under *the curve* (*AUC*) and identify the highest predictive value. Statistically significant variables were entered into a multivariate logistic regression model to identify factors independently associated with mortality. Predicted mortality was assessed using *OR* obtained in a multivariate regression model. The prognostic significance of the scale indicators for assessing the probability of death was analyzed using *ROC* analysis. The statistical significance of the calculations was accepted at the level of p <0.05.

RESULTS OF OWN RESEARCH

PECULIARITIES CURRENTS CORONARY HEART DISEASE IN PATIENTS WITH POST-INFARCTION RUPTURE OF THE INTERVENTRICULAR SEPTUM

The diagnosis of AMI upon admission was confirmed in patients of groups 1 and 2, in the frequency of which statistically significant differences were observed (p<0.001). Unstable angina was diagnosed in each of the clinical groups (group 1 - 1 patient, group 2 - 1 patient, group 3 - 2 patients). With tension angina pectoris I - III FC (CCS) noted only in the 2nd and 3rd groups of patients; with tension angina FC IV was noted in each of the clinical groups. A general clinical examination of patients with PVSD revealed statistically significant differences in both the incidence of signs of acute HF (p<0.001) and CNC (p<0.001) in patients with different periods from the onset of AMI. In addition, a statistically significant time from the development of AMI. Analysis of the presence of diabetes in patients of group 1, group 2 and group 3 revealed

statistically significant differences between them $\chi^2(2, n=90) = 8.399$; p=0.015. The severity of the patient upon admission was determined by the severity of clinical manifestations of HF, influencing the timing of the operation and the prognosis of its results. (Table 8).

Table 8

ma	Clinical nifestations	Group 1 (n=28)	Group 2 (n=26)	Group 3 (n=36)	Statistical significance
Dyspn	With physical function	8/28.6	17/65.4	22/61.1	$\chi^2(2, n=87)$ = 9.534; p=0.009
ea	At rest	19/67.9	8/30.8	13/36.1	– 9.554, p–0.009
Cyanos	sis	17/60.7	2/7.7	4/11.1	p<0.001
Edema		10/35.7	13/50.0	20/55.6	χ^2 (2, n=90) = 2.557; p=0.278
Hepato	omegaly	17/60.7	20/76.9	34/94.4	p=0.002
Hydrot	horax	10/35.7	12/46.2	14/38.9	$\chi^2(2, n=90) =$ 0.643; p=0.725

Frequency of signs of heart failure in patients with PVSD, n / %.

Clinical status of a patient with PVSD during hospitalization, which determines the timing of the operation and the possibility of delaying it, determines the prognosis of the disease. The severity of the condition of the patient with PVSD during hospitalization was assessed as satisfactory, moderate, severe and life-threatening (Table 9).

Table 9

Assessment of the severity of the clinical condition of patients with PVSD, n / %.

Patient's condition	Group 1 (n=28)	Group 2 (n=26)	Group 3 (n=36)	χ^2 , p
Satisfactory	1/3.6	3/11.5	2/5.6	$\chi^2(2, n=90)$
Moderate	6/21.4	9/34.6	21/58.3	= 9.574;
Heavy	5/17.9	11/42.3	11/30.5	p=0.008
Life threatening	16/57.1	3/11.6	2/5.6	

Data Table 9 demonstrates the relationship between the development of a moderately severe condition in patients with PVSD and a progressive decrease in the frequency of severe and life-threatening conditions as the time from the onset of AMI increases (χ^2 (2, n = 84) = 9.788; p = 0.007). A severe and life-threatening condition was observed in 75.0% of patients in group 1, 53.9% of patients in group 2 and 36.1% of patients in group 3.

Instrumental examination of patients with post-infarction rupture of the interventricular septum

Despite the widespread use of ECG to detect acute ischemic myocardial injury and its consequences, ECG signs that allow the assessment of myocardial integrity have not been clearly defined. The most pressing purpose of using ECG in patients with PVSD is to identify signs that reflect the dynamics of the formation of cardiosclerosis, making it possible to clarify the timing of operations (Table 10).

Table 10

ECG changes	Group 1 (n =28)	Group 2 (n =26)	Group 3 (n =36)	χ ² , p
ST segment deviation and/or T wave changes or small focal changes	15 /53.6	6 /23.1	3 /8.3	$\chi^{2}(2, n=90) = 1 $
Cicatricial changes in the myocardium (PICS)	11/39.3	20/76.9	31/86.1	$\begin{array}{l} \chi^{2}(2,n{=}90){=}\\ 17,214;\\ p{<}0.001 \end{array}$
Signs of the for- mation or presence of an LV aneurysm	4/14.3	6/23.1	10/27.8	χ^2 (2, n= 90) = 1.674; p= 0.43 3
Heart rhythm disturbances	9/32.1	6 /23.1	9/25.0	χ^2 (2, n= 90) = 0.652; p=0.722

ECG changes in patients with PVSD, n /%.

Designations: L V – left ventricle; PICS – post-infarction cardiosclerosis.

Analysis of samples of patients with breast cancer in each of the clinical groups indicates the absence of a normal distribution in the values of individual indicators of the functional state of the LV myocardium. For group 1 of patients with PVSD, the following results were obtained (LV end-diastolic volume (EDV): W = 0.870 p = 0.003; LV end-systolic volume (ESV): W = 0.977 p = 0.834; LV SV: W = 0.906 p = 0.140; LVEF: W = 0.954 p = 0.270; $\Delta \text{ P LV/RV}$: W = 0.935 p = 0.534 and defect size: W = 0.829 p = 0.005). For group 2 of patients with PVSD – LV EDV: W = 0.883 p = 0.007; LV ESV: W = 0.975 p = 0.831; LV SV: W = 0.946 p = 0.342; LVEF: W = 0.852 p = 0.002; $\Delta \text{ P LV/RV}$: W = 0.903 p = 0.391 and defect size: W = 0.877 p = 0.009. For group 3 of patients with PVSD – LV EDV: W = 0.976 p = 0.642; LV ESV: W = 0.980 p = 0.767; LV SV: W = 0.951 p = 0.679 and defect size: W = 0.911 p = 0.009 (Table 11).

Table 11

Data from preoperative ECHO-CG examination of patients with PVSD

with PVSD					
Indicators	Group 1 (N =28)	Group 2 (N =26)	Group 3 (N =36)	<i>N</i> , p	
LV EDV, ml . (Normal: 65-130) <i>Mo</i> <i>Meh</i> <i>Q</i> 1 : <i>Q</i> 3	(n =27) 151.0 153.0 146.0; 185.0	(n =26) 176.0 190.0 175.0; 250.0	(n =35) 244.0 201.0 175.0; 244.0	H = 1 2.58 5 (2, n = 88), p= 0.00 2	
LV ESV, ml. (Normal: 40-60) <i>Mo</i> <i>Meh</i> <i>Q</i> 1 ; <i>Q</i> 3	(n =24) 59.0 104.5 87.7; 119.0	(n =21) 108.0 127.0 100.0; 164.0	(n =34) 102.0 114.0 90.5; 137.0	N =4.030 (2, n =79), p=0.133	
UO LV, ml. (Normal: 55-90) <i>Mo</i> <i>Meh</i> <i>Q</i> 1 : <i>Q</i> 3	(n =14) 73.0 74.5 64.0; 87.5	(n =19) 68.0 82.0 68.0; 100.0	(n =34) 112.0 100.0 85.7; 112.2	<i>H</i> =9.404 (2, n =67), p=0.009	
LVEF, % (Normal: 55-60) <i>Mo</i> <i>Meh</i> <i>Q</i> ₁ ; <i>Q</i> ₃	(n =27) 40.0 41.0 36.0; 44.0	(n =26) 45.0 43.5 37.7; 49.0	(n =34) 47.0 45.0 39.7; 52.2	N = 4.978 (2, n = 87), p = 0.083	

ΔP LV/RV, mm Hg. <i>Mo</i> <i>Meh</i> <i>Q</i> 1; <i>Q</i> 3	(n =9) 28.0 44.0 33.0; 54.5	(n =6) 43.0 59.5 44.5; 71.2	(n =10) 70.0 57.0 41.5; 72.5	H =3.523 (2, n =25), p=0.172
Size of IVS	(n =17)	(n =23)	(n =34)	<i>H</i> =1.133 (2, n =74), p=0.567
defect, cm . <i>Mo</i>	1.5	2.0	1.0	
<i>Meh</i>	1.5 1.0;	1.8	1.5	
<i>Q</i> ₁ ; <i>Q</i> ₃	2.0	1.0; 3.0	1.0; 2.5	

Designations: LV - left ventricle; LV EDV - LV end-diastolic volume; LV ESV - LV end-systolic volume; LV SV - LV stroke volume; LVEF – LV ejection fraction; Δ P LV/RV – pressure gradient between the LV and the right ventricle; IVS interventricular septum. N – total number of patients in the group; n is the number of patients in whom the study was conducted.

According to the echocardiographic examination, there were no statistically significant differences between the clinical groups in the frequency of detection of thrombus in the LV, multiple IVS defects and disorders of local contractility of the LV.

Table 12

Characteristics of coronary artery lesions in patients with $\mathbf{DVSD} \mathbf{n} / 0_{0}$

CA	Group 1 (n =28)	Group 2 (n=26)	Group 3 (n=36)	χ ² , p
1 vessel	7/25.0	5/19.2	6/16.7	$\chi^{2}(4, n=90)$
2 vessels	6/21.4	10/38.5	16/44.4	= 3.793;
3 vessels	15/53.6	11/42.3	14/38.9	p=0.435

Designations: CA – coronary artery.

Analysis of coronary artery lesions in patients with LAD showed that hemodynamically significant stenoses were most often observed in the LAD and RCA. However, when comparing the frequency of atherosclerotic lesions between groups KA in general (χ^2 (2, n=270) = 0.124; p=0.939), no statistically significant difference was obtained.

We assessed the severity of atherosclerotic lesions of the main branches of the coronary artery in patients with PVSD in each of three clinical groups. In general, according to the results of the analysis of coronary angiography in these groups of patients, the LAD axis was most often observed. There was no significant difference in age between the patient groups (H=0.8961 (2, n=90), p=0.639).

RESULTS OF SURGICAL TREATMENT OF PATIENTS WITH POST-INFARCTION RUPTURE OF THE INTERVENTRICULAR SEPTUM

Modern surgical correction of PVSD is based on several principles:

1-introducing the patient into severe hypothermic cardiopulmonary bypass and ensuring the maximum possible protection of the myocardium

2- access to the LVAD through the infarcted LV tissue

3- careful treatment of the edges of the infarcted LV through which access to viable tissue was achieved in order to prevent delayed cutting of sutures

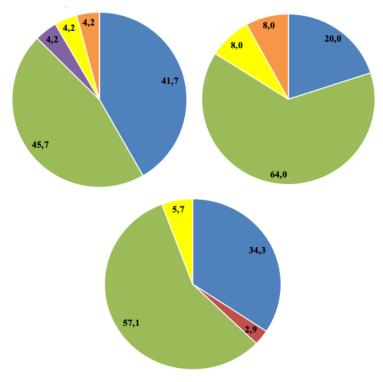
4-inspection of the papillary muscles and replacement of the mitral valve only in cases of severe rupture of the papillary muscles

5- closure of the primary gastrointestinal tract without tension, which is in the vast majority cases will require additional and abundant use of artificial prosthetic material

6-strengthening seam lines with strips of Teflon gasket for prevent seams from cutting through.

The determination of the optimal time for performing PVSD correction remains relevant and controversial. This is due to several factors: 1).in the acute phase of myocardial infarction, the tissues are not strong enough, and only after two weeks does the regenerative process and the scarring phase begin (up to 28 days after the development of myocardial infarction); 2).the high probability of surgical complications and mortality in the early stages after the development of AMI, force surgeons to postpone the operation for some time, if the patient's clinical condition allows; 3).high mortality in the early stages of the onset of AMI forces the use of interventional methods to temporarily stabilize the hemodynamics of patients with PVSD. The use of the latter in the early stages of the onset of AMI

makes it possible to delay the surgical correction of breast cancer, reducing the risk of complications associated with opening.



1 Group (n =24) 2 Group (n =25) 3 Group (n =35)

- PVSD plasty
- PVSD plasty+ LV trombectomy
- PVSD plasty+ LV aneurysm resection
- PVSD plasty+ LV trombectomy +LV aneurysm resection+occluder remove
- PVSD plasty + LV trombectomy +LV aneurysm resection
- PVSD plasty+ occluder remove

Figure. 5. Scope of surgery to restore geometryLV in patients with PVSD (%).

Designations: LV – left ventricle; RAL – resection of LV aneurysm; PVM – post-infarction rupture of the interventricular septum.

Among 90 patients, correction of PVSD was performed in 84 (93.3%). In 6 (6.7%) patients, the operation was not performed due to death before the operation, or patient refusal to undergo intervention. In 4 of 84 patients, occluder implantation was performed prior to surgery. U 79 (94.0%) of the 84 operated patients, correction of PVSD was performed simultaneously with CABG and/or mama-coronary bypass grafting (MCBG). CABG and/or MCS were performed in 22 patients of group 1; 21 - 2 groups; at 30 patients were in group 3 (Fig. 5). Mammarocoronary anastomosis was performed in 1 (4.3%) patient group 1; 3 (12.5%) - 2 groups; in 2(6.3%) patients 3 groups.

Possibilities of endovascular occlusion of the prostate esophagus and mechanical hemodynamic support to delay surgery

Endovascular correction of PVSD was used in 7 (7.8%) of 90 patients. In group 1 (n=28), EVO was used in 5 (17.9%) patients; at 2 group (n=26) - 2 (7.7%) patients. Occlusion of the VSD was performed in 6 patients with anterior and 1 patient with a posterior rupture. In the last patient (group 1), occlusion of the ventricular septum was accompanied by pacemaker implantation. In 4 cases, residual shunts were observed after occluder implantation. The occluder was implanted according to urgent indications: in the first group - three patients with anterior LVAD, one - with posterior LVAD; in the second group - 3 patients with anterior PVSD, two of them on day 2, one - on the 4th day of hospitalization.

In group 1 of patients, three patients died before surgery: one a patient with a posterior LBD and two with anterior LBD, among the latter was a patient with an implanted occluder on the 4th day of hospitalization. After installation of the occluder, 4 patients with anterior LBD underwent LAD correction. 2 patients were in the first and 2 patients in the second group. In one patient of the first and one patient of the second group, echocardiography verified signs of a large residual shunt after EVO. The use of endovascular occlusion of the LVAD in the early stages after the onset of AMI in 4 out of 7 patients made it possible to stabilize hemodynamics, facilitating delayed surgery on days 8, 23, 37 and 42 after the onset of AMI.

The use of mechanical support of systemic hemodynamics using intra-aortic balloon counterpulsation in patients with PVSD

Among of patients with PVSD hospitalized for surgery (n=90), 10.0% of patients (n=9) needed to connect an IABP before surgery. In group 1 (n=28), IABP was used in 25% of patients (n=7), including 2 patients with anterior and 5 - with posterior prostates; in group 2(n=26) - in two (7.7) patients with anterior PVSD. The IABP was connected according to urgent indications on the day of hospitalization (n=9). Before Two patients of the first group underwent surgery and died (1 patient with anterior and 1 patient with posterior PVSD); died in group 2 one patient with anterior PVSD. Both patients of the first group underwent EVO rupture at the same time. Correction of the LVAD was performed after connecting the IABP 6 patients: in group 1, one patient with anterior and four - with posterior cervical prostates; in the 2nd group - to a patient with anterior PVSD. Three patients died in this group. Reasons lethal outcomes were: progressive heart failure (HF) in 2 patients of the first group with posterior PVSD; gastrointestinal bleeding (GIB) in one patient of the second group with anterior PVSD. Connection of an IABP contributed to the stabilization of hemodynamics in 6 out of 9 patients, allowing the operation to be performed within 1 to 8 days hospital stay. In three cases that ended in death before surgery, the cause was the severe clinical condition of the patients rather than the lack of effectiveness of IABP.

13 (15.5%) out of 84 needed to connect an IABP before completion of the CPB. operated patients, including 9 patients of the first group, 6 patients with anterior and 3 patients with posterior PVSD; in group 2 – 1 patient with anterior and 1 patient with posterior PVSD; in group 3 – one patient with anterior and one patient with posterior PVSD. The average duration of IABP was 52.9 \pm 40.7 hours (Z =0.523, p =0.947). 31 % of those operated on with anterior and 38% with posterior LVADs required connection of an IABP before surgery; in 69% with anterior and 62% with posterior PVSD - in the intra- and postoperative period (Fig. 6). Among 17 operated patients with IABP connection, postoperative mortality was 35.3% (n=6). The causes of death were: acute heart failure in 3 patients with anterior PVSD of the first clinical group; Gastrointestinal tract and multiple organ failure in 2 patients with posterior PVSD; respiratory failure due to initial tuberculosis lungs in one patient with anterior PVSD of the second clinical group.

Analysis of the percentage distribution of patients according to the timing of IABP connection before-, intra- and postoperatively indicates that there is no significant difference between patients with anterior and posterior LVAD (p = 0.584); (Fig. 6).

An analysis of the mortality rate of patients who required an IABP connection showed no statistically significant differences between the groups with anterior and posterior LVAD (χ^2 (1, n =26) = 0.619; p =0.431).

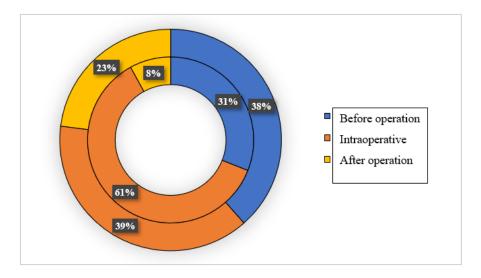


Figure 6. IABP connection in patients with anterior and posterior PVSD. Outer circle - patients with posterior PVSD (n = 13); Inner circle - patients with anterior PVSD (n = 13))

STRUCTURE OF POSTOPERATIVE MORTALITY AND COMPLICATIONS IN PATIENTS WITH POST-INFARCTION RUPTURE OF THE INTERVENTRICULAR SEPTUM

The severity of the clinical condition of patients with different localization of PVSD is reflected by the frequency of postoperative complications noted in 39.6% (n = 19) of patients with anterior and 66.7% (n = 24) - posterior localization of PVSD (χ^2 (1, n = 84) = 6.039; p=0.014)

To determine the optimal timing of surgical intervention, we assessed the dynamics of indicators of the postoperative period in the studied groups of patients with PVSD (Table 13).

Table 13

n /%							
Index	Group 1 (n =24)	Group 2 (n =25)	Group 3 (n =35)	Statistical significance			
Mechanical ventilation > 24 hours	18/75 %	14/56 %	24 / 68.6 %	$\chi^{2}(2, n=84) = 2.087;$ p=0.352			
Nitrate infusion	12/50 %	4/16 %	10/28.6 %	$\chi^{2}(2, n=84) = 6.782;$ p=0.034			
Inotropic support	18/75 %	12/48 %	23/65.7 %	$\chi^{2}(2, n=84) = 4.010;$ p=0.135			
Discharge through drains (> 500 ml / day)	8/33.3 %	1/4%	2/5.7 %	p<0.005			
Transfusion of blood and its components	12/50 %	3/12 %	4/11.4 %	p<0.005			

Characteristics of the postoperative period in patients with PVSD, n/26

Despite the absence of significant differences in the number of patients on mechanical ventilation, differences were identified in the

frequency of drug therapy between groups. In group 1 of patients, the frequency of use of nitrates, as well as sympatho- and adrenomimetic support, is higher than in other groups. In addition, the number of patients whose volume of drainage discharge exceeded 500 ml/day, requiring transfusion of blood and its components, was greater in group 1 than in other groups. The identified significant differences indicate a more severe course of the postoperative period in patients with PVSD who were operated on in the early stages after the development of AMI. Despite the absence of significant differences in the frequency of use of inotropic support, differences are observed between groups of patients with PVSD in the number of drugs used (Fig. 7). The data in Figure 7 indicate a decrease in the number of inotropic drugs in patients with PVSD operated on later after the development of AMI. Analysis of the need for the amount of such drugs in the postoperative period in surviving patients confirms this trend (p=0.033).

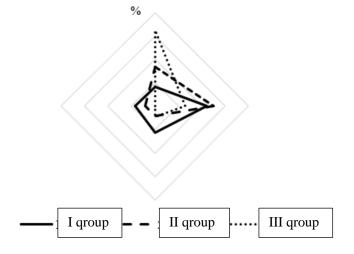


Figure 7. The need for inotropic support in patients with PVSD (1, 2, 3, 4 – number of drugs (norepinephrine, dopamine, adrenaline, dobutamine))

As was shown earlier, significant differences in the frequency of transfusion of blood and its components were identified between

groups of patients with PVSD. However, the very need for transfusion of blood and its components does not fully reflect the severity of the clinical condition of patients. An additional indicator of the latter is the number of blood components used (Fig. 8). We noted a decrease in the frequency of use replacement blood transfusion therapy, as well as a progressive decrease in the amount of blood components used in those operated on at a later date from the development of AMI (Fig. 8). The high frequency of use of blood and its components in patients 1 There is a body of evidence of both postoperative blood loss, periand as well as severe thrombocytopenia due to long-term use of ECMO or prolonged cardiopulmonary bypass. The identified relationship between the amount of blood and its components used and the duration of operations from the onset of AMI in patients with PVSD, there is evidence of a more severe postoperative period in patients 1 groups.

The severity of patients with PVSD is evidenced by postoperative complications in 75% (n = 18) of patients in group 1; 44% (n =11) - 2 groups; 40% (n = 14) - 3 groups, operated on at different times from the development of AMI (χ^2 (2, n = 84) = 7.717; p = 0.021).

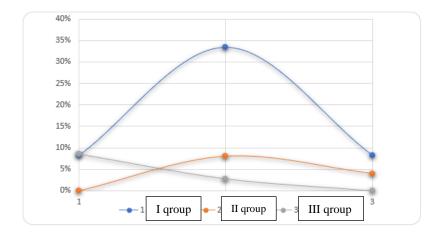


Figure 8. The need for transfusion of blood and its components in patients with PVSD (1, 2, 3 – number of drugs (fresh frozen plasma, packed red blood cells, platelet mass)

In the structure of postoperative complications in 84 patients with PVSD, low cardiac output syndrome dominated in 32.1% (n = 27), indicating a greater severity of the postoperative prognosis in patients with PVSD operated on early after AMI (Table 14).

The incidence of small output syndrome and acute renal failure differed significantly among the groups of operated patients. Acute renal failure was more often observed in patients of group 1, as well as before surgery. The data obtained are consistent with the idea of the role of renal failure as a factor complicating the course of PVSD and postoperative prognosis.

Complications	Group 1 (n =24)	Group 2 (n =25)	Group 3 (n =35)	Statistical indicators
Rethoracotomy	2/8.3	3/12.0	3/8.6	p = 0.901
LCO syndrome	16/66.7	6/24.0	5/14.3	$\chi^2(2, n=84) =$ 18.992; p< 0.00 5
Pulmonary disorders	4/16.7	1/4.0	3/8.6	p = 0.282
Neurological disorders	2/8.3	1/4.0	3/8.6	p = 0.763
Kidney failure	7/29.2	3/12.0	2/5.7	p=0.047
Gastrointestinal disorders	1/4.2	1/4.0	1/2.9	p=1.0
Arrhythmia	6/25.0	3/12.0	6/17.1	χ^2 (2, n= 84) = 1.43 2; =0.489
Bleeding	2/8.3	4/16	4/11.4	p = 0.766

Structure of postoperative complications in patients with PVSD, n / %.

Table 14

Designations: LCO – low cardiac output.

U 75% of patients with PVSD in each group had IC duration > 140 min. and aortic cross-clamping > 75 min. served as predictors

of a complicated postoperative period in 17.9% of 84 patients operated on with PVSD.

Analysis of postoperative mortality in patients with postinfarction rupture of the interventricular septum

Surgical intervention aimed at correcting the prostate rupture reduces the mortality rate during the natural course of the rupture to an average of 40-70%. The mortality rate is significantly influenced by the initial hemodynamic status of the patient, which correlates with the timing of the onset of AMI. According to the literature, postoperative mortality during operations performed in the acute and subacute stages of myocardial infarction is about 50%, reaching 80 % with the development of cardiogenic shock⁹. Based on what has been described, it was advisable to conduct an analysis mortality in patients with PVSD depending on the timing of the onset of AMI and the location of the rupture.

Of the 90 hospitalized patients with PVSD, two refused to intervene; four (4.5%) died before surgery. In-hospital mortality in 84 operated patients was 22.6% (n = 19). In the 1st group of patients there were 10, in the 2nd - 5, in the 3rd - 4 deaths (χ^2 (2, n = 84) = 7.577; p = 0.023), showing a progressive decrease as the duration of treatment increased after the development of AMI (Fig. 9).

The division of patients into three clinical groups is based on time intervals sufficient for the development of post-infarction "cardiac remodeling". At the same time, in each of the three groups there are patients with unstable cardiac hemodynamics requiring immediate surgical intervention. At the same time, the clinical condition of other patients in these groups made it possible to delay surgery to repair the IVS rupture. However, waiting for the formation of post-infarction cardiosclerosis can lead to the development of biventricular failure. Since not all patients can be stabilized for surgery within the time frame corresponding to PICS, it was advisable to determine the most optimal time intervals for performing operations, taking into account the risk of death (Fig. 10). According to Figure 5.6, starting from 3 weeks after the development of AMI, there is a decrease in operative mortality from

42.1% to 10.5-15.8%. In the future an increase in the time period from the development of AMI, no significant changes in mortality rates were noted. It can be assumed that the use of drug therapy, EVO rupture and mechanical support of systemic hemodynamics is justified in the early stages after the development of AMI.

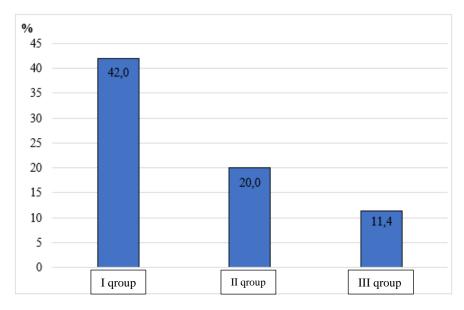


Figure. 9. Hospital mortality in operated patients with PVSD

This tactic of managing patients in the first 2 weeks after the development of AMI allows for delaying the operation to the most acceptable, from a prognostic point of view, timing. Considering the risk of mortality, it is most advisable to perform PVSD correction after 2 weeks from the onset of AMI. Since the processes of post-infarction "heart remodeling" and the development of decompensation occur in parallel, in patients of group 1, further expectant management does not help reduce operative mortality.

Analyzing the causes of deaths after surgery, We considered it appropriate to separately consider cardiac and extracardiac causes of death in patients with PVSD after surgery (Fig. 11).

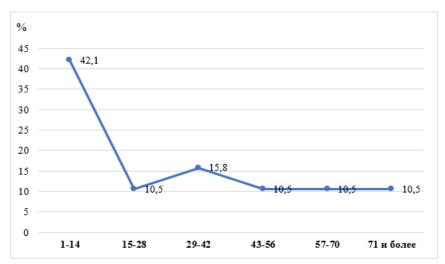


Figure 10. Mortality of patients with PVSD depending on the timing of the operation after the development of AMI (day)

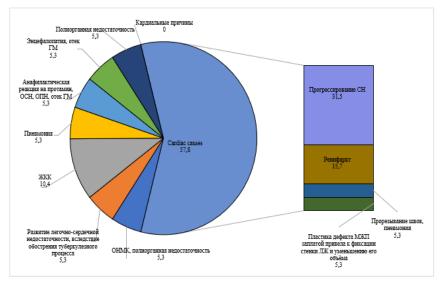


Figure 11. Structure of cardiac and extracardiac causes of deaths in patients with PVSD (%)

Designations: HF – heart failure; IVS – interventricular septum; LV – left ventricle; AHF – acute HF; GM – brain; AKI – acute renal

failure; GI bleeding – gastrointestinal bleeding; ACVA – acute cerebrovascular accident.

Among 84 patients operated on with PVSD, in 13.1% (n = 11) of cases, death was due to cardiac, accounting for 57.8% of total mortality. Taking into account the differences in the timing of post-infarction "cardiac remodeling" in patients with PVSD, we studied mortality from cardiac and non-cardiac causes in each of the clinical groups (Fig. 12).

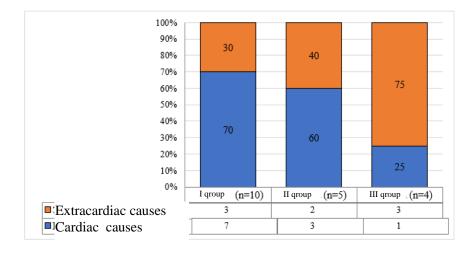


Figure 12. Postoperative mortality in patients with PVSD

Data in Fig. 12 indicate the predominance of cardiac causes of mortality in patients of groups 1 and 2 with PVSD. Noteworthy is the progressive decrease in mortality from cardiac causes as the time from the development of AMI increases. And an analysis of the causes of death in patients with PVSD showed that the most common the reason was the rapid progression of HF in the postoperative period (Table 15).

Table 15

Causes of deaths in the postoperative period in patients with PVSD, n/%

n / %.					
Cause		Group 1 (n =24)	Group 2 (n =25)	Group 3 (n =35)	
Cardiac (OSN)	Progression of heart failure	6/25.0	-	-	
	AMI (reinfarction)	-	2/8	-	
	Thrombosis of the shunt to the RCA in the right type of RCM, RV AMI	-	1/4	-	
Ca O	Cutting stitches, pneumonia	1/4.2	-	-	
	Plastic repair of the IVS defect with a patch led to fixation of the LV wall and a decrease in its volume	-	-	1/2.9	
	Cerebrovascular accident, multiple organ failure	-	-	1/2.9	
	Development of pulmonary heart failure due to exacerbation of the tuberculosis process	-	1/4	-	
rdia	Housing and communal services	1/4.2	1/4	-	
Extracardiac	Pneumonia	-	-	1/2.9	
	Anaphylactic reaction to protamine, AHF, acute renal failure, cervical edema	-	-	1/2.9	
	Encephalopathy, brain edema	1/4.2	-	-	
	Multiple organ failure	1/4.2	-	-	

Designations: HF – heart failure; AHF – acute HF; AMI – acute myocardial infarction; GI bleeding – gastrointestinal bleeding; RCA – right coronary artery; RV – right ventricle; IVS – interventricular septum; LV – left ventricle. Percentages are calculated based on the total number of patients operated on in each group.

In patients of group 1, acute HF was most often caused by an initially more severe clinical condition, early timing of operations and the volume of the latter; patients of group 2 – recurrent AMI; in

patients of group 3 - an expanded volume of intervention, corresponding to a longer duration of bypass and aortic clamping. Extracardiac pathology was the cause of death in 9.5% of cases (n = 8). Among noncardiac causes of death, despite preoperative gastroscopy and the use of proton pump inhibitors, GIB was the cause of death in 2 patients with PVSD. Analysis postoperative mortality and depending on the correction of the anterior and posterior LVADs show that correction anterior and posterior PVSD s were accompanied by a mortality rate of 20.8% (n = 10) and 25.0% (n = 9), respectively (χ^2 (1, n = 84) = 0.035; p = 0.851). Taking into account the different volume of myocardial damage in patients with anterior and posterior VSD, which affects postoperative mortality, we analyzed mortality in each of these groups, taking into account cardiac and extracardiac causes (Fig. 13).

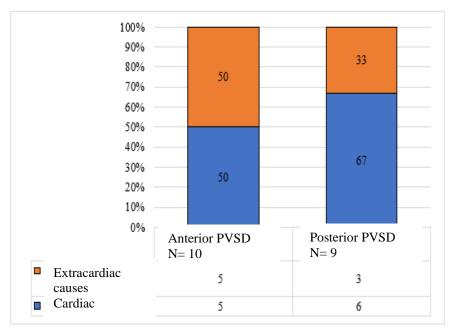


Figure. 13. Mortality of patients with different localization of PVSD

Cardiac pathology was the cause of death in 10.4% of patients (n = 5) with anterior and in 16.6% (n = 6) of patients with posterior VSD. When analyzing fatal outcomes, the most common cause of acute HF was the initial severity of the clinical condition. Extracardiac pathology was the cause of death in 10.4% (n = 5) of patients with anterior and in 8.3% (n = 3) of patients with posterior VSD. At the same time, cases of gastrointestinal bleeding were observed both in the group of patients with anterior and posterior localization of PVSD (Table 16).

Table 16

with different localization of PVSD, II / %			
Cause		Anterior localization of PVSD (n =48)	Posterior localization of PVSD (n =36)
	Progression of heart failure	3/6.3	3/8.3
	AMI (reinfarction)	2/4.2	-
Cardiac (OSN)	Thrombosis of the shunt to the RCA in the right type of coronary circulation, RV AMI	-	1/2.8
	Cutting stitches, pneumonia	-	1/2.8
	Plastic repair of the IVS defect with a patch led to fixation of the LV wall and a decrease in its volume	-	1/2.8
Extracardiac	Cerebrovascular accident, multiple organ failure	1/2.1	-
	Development of pulmonary heart failure due to exacerbation of the tuberculosis process	1/2.1	-
	Housing and communal services	1/2.1	1/2.8
	Pneumonia	-	1/2.8
	Anaphylactic reaction to protamine, AHF, acute renal failure, cervical edema	1/2.1	-

Causes of deaths in the postoperative period in patients with different localization of PVSD, n / %

Encephalopathy, cervical edema	1/2.1	-
Multiple organ failure	-	1/2.8

Designations: HF – heart failure; AHF – acute HF; AMI – acute myocardial infarction; GI bleeding – gastrointestinal bleeding; RCA – right coronary artery; AKI – acute renal failure; RV – right ventricle; IVS – interventricular septum; LV – left ventricle. The percentages indicated in the table are calculated for the total number of operated patients in each group.

Predictors of mortality in patients with post-infarction rupture of the interventricular septum

In our study, the age of the deceased patients was 63.3 ± 9.9 years; survivors - 58.9 ± 9.3 years. Proportion of patients with chronic PN in the anamnesis among the deceased was 47.8%, more than twice as high as the proportion of such patients among survivors (20.0%). Analysis of the hemodynamic profile during hospitalization indicated the existence of differences in the values of the shock index, heart rate, and class IV (NYHA) between deceased and surviving patients with PVSD. There were also differences between the groups of patients in the incidence of pulmonary edema and the timing of surgery from the development of AMI, SBP, and LV EDV (Table 17).

Table 17

	(n = 88)		
	Survivors	Dead	
	patients with	patients	n
Index	PVSD	with PVSD	р
	(n =65)	(n =23)	
Age (years)	58.9±9.3	63.3±9.9	0.011
Male	48 (73.8%)	15 (65.2%)	0.433

Characteristics of groups of patients during hospitalization depending on the postoperative outcome of PVSD correction (n - 88)

Body mass index kg/m2	26.9±3.9	29.1±6.5	0.169
Cardiac index (l/min/ m2)	1.9 ± 0.6	$2.0{\pm}0.8$	0.18 2
Heart power index (W/m ²)	0.38 ± 0.2	0.37 ± 0.2	0.364
Day from AMI to surgery	75.7±74.6	33.1 ± 37.7	< 0.001
GFR (ml/min/1.73 ^{m2})	80.3±47.9	62.3±40.4	0.068
History of CKD (GFR < 60 ml/min/1.73 m ²)	13 (20.0 %)	eleven (47.8%)	0.01 5
History of COPD	8 (12.3%)	3 (14.0 %)	1.0
Diabetes	19 (29.2)	12 (52.1%)	0.074
Arterial hypertension	18 (27.6%)	3 (13.0 %)	0.254
IV FC (NYHA)	30 (46.1%)	20 (8 7.0 %)	< 0.001
LVEF (%)	43.4±9.7	45.1±12.7	0.41 3
LV EDV (ml)	203.1±47.1	188.3±70.2	0.011
PRM size (mm)	17.8±9.9	20.1±14.1	0.46 3
Presence of LV aneurysm	16 (24.6%)	4 (17.3%)	0.640
Pulmonary edema accor- ding to ultrasound data	4 (6.1%)	7 (30.4%)	< 0.001
ST segment elevation	16 (24.6%)	7 (30.3%)	0.59 1
Heart rate (bpm)	81.5±13.4	94.5±14.2	0.002
SBP (mm Hg)	116.4±19.1	106.1±17.7	0.00 3
Pulse pressure (mmHg)	40.7±13.1	34.8±14.7	0.052
Shock index (HR/SBP)	0.9±0.2	1, 2 ± 0.2	< 0.001

Designation: AMI – acute myocardial infarction; GFR – glomerular filtration rate; HR – heart rate; CKD – chronic kidney disease; COPD – chronic obstructive lung disease; LV – left ventricle; LVEF – LV ejection fraction; LV EDV – LV end-diastolic volume; FC – functional class; Ultrasound – ultrasound diagnostics; SBP – systolic blood pressure; PVM – post-infarction rupture of the interventricular septum.

Univariate regression analysis revealed the predictive value of indicators unfavorable surgical outcomes for patients with PVSD, with statistically significant differences (Table 18).

Optimal timing of intervention remains the main problem in the correction of breast cancer. Analysis of the postoperative period patients with PVSD indicates the advisability of wait-and-see tactics surgical treatment provided that the patients are hemodynamically stable, because starting from 3 weeks after the onset of AMI, there is a decrease in operative mortality from 42.1% to 10.5-15.8%. Further wait-and-see tactics do not justify themselves, because does not reduce operative mortality in patients with PVSD.

Therefore, given the risk of mortality, it is most appropriate is to perform surgical interventions after 2 weeks from the development of AMI. At the same time, the need for emergency surgery in patients with PVSD should be based on ineffective attempts to stabilize systemic hemodynamics with drug therapy, IABP and endovascular occlusion.

Table 18

Results of univariate logistic regression analysis of in indicators of					
patients with PVSD					
Index	OR	95% CI	р		
Age (years)	0.988	0.966 - 1.004	0.051		
Day from AMI to surgery	1.119	0.983 - 1.338	< 0.001		
Heart rate (bpm)	4,079	1.032 - 18.98	< 0.001		
History of CP N	2,721	0.833 - 14.20	0.013		
LV EDV (ml)	0.015	0.008 - 3.997	0.268		
IV FC (NYHA)	0.937	0.840 - 1.009	0.003		
Pulmonary edema	0.988	0.966 - 1.004	0.601		
Shock index (HR/SBP)	1.119	0.983 - 1.338	< 0.001		
SBP (mm Hg)	4,079	1.032 - 18.98	0.030		

Results of univariate logistic regression analysis of n indicators of

Designation: AMI – acute myocardial infarction; HR – heart rate; CKD - chronic kidney disease; LV - left ventricle; LV EDV - LV end-diastolic volume; FC - functional class; SBP - systolic blood pressure; Ultrasound – ultrasound diagnostics; CI – confidence interval: OR – odds ratio.

CONCLUSIONS

1. The stabilization of clinical status of the patients with PIIVSR was based on inotropic support, ventilator, IABC, and endovascular intervention in 100%, 5,7%, 10,2% μ 7,8% of patients, respectively. Life support ineffectiveness was considered as an operation indication within \leq 24 hours. of hospitalization duration in 19% with a 37,5% mortality rate.

2. Due to endovascular occlusion (EVO) hemodynamical stabilization of the PIIVSR was achieved in 71,4%; decrease in shunt volume in 85,7%; the total rupture occlusion in 14,3% of cases. EVO mortality rate was 28,6%. The rupture EVO enabled operation postponed within 8-42 days of hospitalization in 57,1% of patients.

3. In patients who underwent operation within \leq 7 days, 7-28, and >28 days of hospitalization the survival rate was 58,3%; 80%, and 82,9%, with post-op complications in 75%; 44%, and 40%, respectively. In operated within \leq 24 чac. and \leq 7 days of hospitalization, the rate of "low cardiac output" syndrome was 56,3% and 66,7%, respectively; in operated within >7 days – 18,3%;

4. The mixed PIIVSR correction technique showed better results rather, than the isolated one, 77,2% and 0%, with better hospital survival and mortality rates in anterior ruptures superior to posterior ruptures, 79,2% and 20,8%; 75% and 25%, respectively.

5. The leading predictors of hospital and 30-day mortality after PIIVSR were as follows: a). Previous unstable hemodynamics / cardiogenic shock (p<0,001); 6). Conducting operations within \leq 7 days after the debuted AMI (p<0,001); B). Posterior PIIVSR; Γ). Chronic/acute kidney failure (p<0,015).

6. The hospital outcomes of the PIIVSR are not primarily determined by IABC, but, rather initial patient clinical status and time interval from debuted AMI till operation.

PRACTICAL RECOMMENDATIONS

1. During the pre-op management period of patients with PIIVSR the mandatory examinations are Echocardiography, coronary angiography/ventriculography, MSCT, and MRI, which enable the determination of IVS ruptures localization and number, their size and anatomy, coronary blood flow pattern, intracardiac hemodynamics disturbance, and myocardial viability.

2. During the planning of surgical intervention in patients with PIIVSR it should be used the risk scale scoring for mortality rate, which enables the distribution of patients based on main 30-day mortality predictors, which is essential in management tactics selection in patients with IVS defects.

3. Decision on urgent surgical intervention should be based clinical status of the patients with PIIVSR and, the probability of system hemodynamic stabilization in case of deterioration with the help of medical therapy and endovascular techniques.

4. In postponed surgical management of patients with PIIVSR it should be borne in mind the cardiosclerosis development and somatic compensatory mechanisms as well as the time from AMI onset. Two weeks after AMI onset. Are considered to be the most appropriate time for surgical intervention.

5. There is a certain risk of late LV aneurysm developing after isolated correction of anterior PIIVSR within an early period after the debuted AMI. Therefore, the surgical technique within an early period after the debuted AMI should cover extended resection of impaired myocardium.

6. Revascularization of infarct-dependent artery or multivessel coronary artery lesions in patients with PIIVSR is mandatory regardless of surgery time after debuted AMI and IVS defect localization.

7. The Biphasal hybrid intervention technic with endovascular occlusion of rupture is serving to increase the efficacy of surgical management. Meanwhile, the occluders are not recommended in cases of ruptures larger than 15mm, multiple PIIVSR, and posterior PIIVSR.

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- 2. Ретроспективный анали результатов хирургического лечения постинфарктного разрыва межжелудочковой перегородки // Ukrainian journal of cardiovascular surgery 2021; 42(1): 85-91
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- 6. Modern aspects of surgical treatment of post-infarction ventricular septal defect // Ürək-damar cərrahiyyəsi jurnalı 2020; 1(1): 50-66 (K.K. Musayev)
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- 16. Outcomes of post-mi VSR surgical repair in patients operated in different periods upon acute myocardial infarction

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ABBREVIATIONS

AH-arterial hypertension

AMI - acute myocardial infarction

ATS – atherosclerosis

BMI - body mass index

CA – coronary artery

CABG - coronary artery bypass grafting

COPD – chronic obstructive lung disease

CRF - chronic renal failure

CT -computer tomography

ECG-electocardiography

ECHO-CG-echocardiography

ECMO- extracorporeal membrane oxygenation

EVO - Endovascular Occlusion

HF - heart failure

IABP -- intraaortic balloon contrapulsator

IVS – interventricular septum

PCI - percutaneous coronary intervention

LAD – Left Anterior Descending artery

LV- left ventricle

LV EDV – LV end-diastolic volume

LV ESV - LV end-systolic volume

LV SV – LV stroke volume

LVEF – LV ejection fraction

MRİ – magnetic resonance imaging

OM LCA – Obtuse Margin branch of Left Coronary Artery

PICS - post-infarction cardiosclerosis

PVSD - Post-infarction ventricular septal rupture

RCA - right coronary artery

SBP – systolic blood pressure

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