J Extra Corpor Technol. 2020;52:196–202
The Journal of ExtraCorporeal Technology

Extracorporeal Membrane Oxygenation during Percutaneous Coronary Intervention in Patients with Coronary Heart Disease

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Abstract: Extracorporeal membrane oxygenation (ECMO) has become an effective method in the treatment of adults and children with severe cardiac and pulmonary dysfunction that is resistant to conventional therapy. The aim of this article was to summarize an experience of ECMO usage for cardiac dysfunction, which develops in patients with coronary heart disease (CHD) during percutaneous transluminal coronary angioplasty. The study comprised a retrospective, single-center analysis of 23 patients with CHD (19 men and four women, average age 65.7 ± 12.3 years), who undertook the ECMO technique during percutaneous transluminal coronary angioplasty. A total of 13 (56.52%) patients died directly in the hospital, or 30 days after a discharge. Independent predictors of fatal outcomes were diabetes mellitus (odds ratio [OR] = 17.58; 95% confidence interval [CI] = 6.47–47.48; p = .00125), chronic renal failure (CRF) (OR = 20.81; 95% CI = 5.95–72.21; p = .00014), and damage to the right coronary artery (RCA) (OR = 25.51; 95% CI = 8.27–79.12; p = .00013). For deceased patients, the "no reflow" phenomenon was indicated in a larger percentage of cases (23.1% in the group of deceased, vs. 10% in the group of survivors). A routine connection to ECMO before the occurrence of cardiac events was significantly more often used in the group of survived patients (90% of cases) than in the deceased (p = .0000001). Diabetes mellitus, CRF, and damage to the RCA were independent predictors of mortality during percutaneous transluminal coronary angioplasty in patients with CHD. The routine use of ECMO in high-risk patients with percutaneous transluminal coronary angioplasty was a positive prognostic factor of patient survival. **Keywords:** extracorporeal membrane oxygenation, myocardial infarction, percutaneous transluminal coronary angioplasty, cardiogenic shock. *J Extra Corpor Technol. 2020;52:196–202*

In recent years, the usage of extracorporeal membrane oxygenation (ECMO) has significantly increased in clinical practice. It became an effective method for the treatment of adults and children with severe cardiac and pulmonary dysfunction that has become resistant to traditional therapy (1,2). The indications for the use of this technique in intensive care units are expanding as a bridge to heart transplantation and for maintaining oxygenation in cases of severe pulmonary dysfunction (3–5).

Received for publication November 19, 2019; accepted July 16, 2020.
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The senior author has stated that the authors have reported no material, financial, or other relationship with any healthcare-related business or other entity whose products or services are discussed in this paper.

Extracorporeal Life Support Organization January 2019 registry report contained a cumulative of 112,231 patients who received extracorporeal support of vital functions (6). According to this report, cardiac ECMO and extracorporeal cardiopulmonary resuscitation ECMO survival rates were 43% and 29%, respectively (6).

According to Makdisi et al. (7), routine indications to provide adequate hemodynamic support for cardiac dysfunction include cardiogenic shock, severe heart failure caused by acute coronary syndrome, hemodynamically significant and non-stopping cardiac arrhythmias, sepsis with deep heart depression, an overdose or toxic effect of drugs that cause myocardial depression, myocarditis, pulmonary embolism, isolated heart injury, acute anaphylaxis, postcardiotomy syndrome, primary transplant failure after heart or lung transplantation, chronic cardiomyopathy (like a bridge before transplantation), and periprocedural

support for percutaneous coronary interventions in high-risk patients.

The studies of Shaukat et al. (8) had shown that the ECMO technique is applied more often to provide adequate hemodynamic support to patients with coronary heart disease (CHD) and those at a high risk of over-procedural complications when performing percutaneous coronary revascularization. Moreover, as noted in the work of Khera et al. (9), the frequency of using blood circulation mechanical support during percutaneous coronary interventions increased from 1.3% to 3.4% in the United States.

However, with all the positive qualities, the usage of ECMO could be accompanied by a number of serious complications. So, according to a scientific literature, the most common complication during this procedure is bleeding at a frequency ranging from 10% to 30% (10,11). According to various authors, a number of neurological complications varies from 4% to 41% (12,13). An intracranial hemorrhage occurs approximately in 10–15% of cases (7). Moreover, according to Davies et al. (14), 43% of lethal cases on ECMO are associated with intracranial hemorrhage.

At the same time, as indicated by Makdisi et al. (7), in most cases, these complications are a consequence of the condition that caused the need for ECMO therapy, rather than complications of the technique itself.

The aim of this article was to generalize the experience of using ECMO for cardiac dysfunction, which develops in patients with CHD during endovascular myocardial X-ray revascularization.

MATERIALS AND METHODS

The study includes a retrospective, single-center analysis of selected percutaneous transluminal coronary angioplasties (PTCA) in patients with CHD. The sampling period was from March 2014 to July 2018.

The study group included 23 patients with CHD on the basis of the Department of Endovascular Surgery and Angiography of the State Institution "Heart Institute Ministry of Health of Ukraine" (Kyiv). The PTCA was carried out using ECMO as a correction method for contractile cardiac dysfunction.

The clinical characteristics of the examined patients are shown in Table 1.

From the Table 1, the majority of patients included in the study were males (82.6%). At the same time, 95.7% of patients had arterial hypertension (AH) as a concomitant pathology, and 65.2% had previously suffered myocardial infarction (MI).

Only four of them had previously used surgical techniques for the treatment of CHD (one case: coronary artery bypass grafting [CABG], and three cases: stenting in case of a heart attack–associated coronary artery [CA]) (Table 1).

At the time of hospitalization, all patients had a diagnosis—acute coronary syndrome, or type 1 MI, with a disease duration of up to 2 days. In most cases (73.9% of patients), three or more coronary vessels were affected on control angiography.

The diagnosis of acute myocardial infarction was made on the basis of clinical data (more than 30 minutes of intense non-stop angina attack); electrocardiogram data (presence of a pathological Q wave, ST segment increase by at least 2 mm from the line or its depression by more than 2 mm, and T-wave inversion); indicators of a clinical blood test: leukocytosis, an increase in the level of enzymes in the blood serum (troponin I, creatine phosphokinase-MB [CPK-MB]); ultrasound data of the heart (identification of akinesia zones); and coronary angiography data.

All patients were examined according to the protocol which was adopted by the State Institution "Heart Institute of the Ministry of Health of Ukraine" for this category of

Table 1. Clinical characteristics of the examined patients (n = 23).

Indicator		Value
Gender (F/M)		4/19
Weight (kg)		60–118 (88.3 ± 21.5)
Age (years)		47–78 (65.7 ± 12.3)
Concomitant pathology	AH	22 (95.7%)
	Diabetes mellitus	4 (17.4%)
	Smoking	11 (47.8%)
	MI with a personal history	15 (65.2%)
	Stenting with a personal history	3 (13%)
	CABG with a personal history	1 (4.3%)
	CRF with a personal history	8 (34.8%)
	Cerebrovascular pathology with a personal history	7 (30.4%)
Number of affected coronary arteries	Average in group	2.9 ± 1.4
	≥3	17 (73.9%)
Type of infarct-associated CA	Left main CA	7 (30.4%)
	Left anterior descending	10 (43.5%)
	Left circumflex CA	1 (4.3%)
	RCA	5 (21.7%)
TIMI flow before PTCA		0–1 (.34 ± .09)

patients. The study protocol was approved by the Ethics Committee.

A total of two patients (8.7% of cases) were diagnosed with a CHD five or more years ago before admission to the clinic for x-ray endovascular treatment, and 91.3% had a CHD in a personal history for up to 5 years. Circulatory failure of functional class III–IV according to the New York Heart Association was diagnosed in 16 examined patients (69.6%).

The area of the left ventricular myocardium lesion when calculating the QRS index averaged 20.4 ± 1.2%. On admission, 16 patients (69.6%) had a heart failure of functional class II according to T. Killip.

The x-ray endovascular stenting procedure was performed in accordance with standard procedures. Routine therapy during angioplasty included nitrates, clopidogrel (a loading dose of 600 mg of clopidogrel with aspirin), and heparin (to achieve and maintain an activated clotting time [ACT] >250 seconds, the ACT was monitored every 30 minutes). Technical aspects of the procedure, including the choice of stent and balloon, the duration of inflation, and pressure, were determined individually by each operator. Hemodynamically significant occlusion of the artery lumen was considered more than 50%.

Radial access was used in 91.3% of cases (21 patients) and femoral in 8.7% (two patients).

When stenting of the CA was performed, an extent of the lesion to select the stent of the CA was calculated, taking into account the diameter of the stenotic lesion and the diameter of the unaffected site of the vessel. The second-generation everolimus-eluting or zotarolimus-eluting stent was used in all patients.

The average number of drug-eluting stents implanted in a patient was 2.3 ± 1.2, and the average length of the stent was 19.1 ± 3.7 mm. The average fluoroscopy time was 39.5 ± 24.7 minutes, and the radiation dose was within 1.68 ± .99 gray.

In 30.4% (seven patients) of cases, in connection with the detection of a CA thrombosis, endovascular interventions were combined with aspiration thrombo-extraction.

The main antegrade blood flow in the target CA was evaluated according to the thrombolysis in myocardial infarction (TIMI) 0–3 classification (13). At the beginning of the main stage of PTCA, the average TIMI blood flow was .34 ± .09 (Table 1).

All patients underwent protocol studies of blood biochemical parameters, including CPK-MB, using a Beckman Coulter AU480 automatic biochemical analyzer, Brea, CA.

A general blood test was performed in a Sysmex XS-500i hematology analyzer, Sysmex Corporation, Kobe, Japan. Blood gas analysis during the procedure was performed using a gas analyzer (ABL800 Flex Series 835, Radiometer, Copenhagen, Denmark), for analyzing pH, electrolytes, and metabolites in blood.

The level of troponin I was analyzed before angiography, and within 72 hours after x-ray endovascular procedures. Blood sampling was performed 6, 12, 24, 48, and 72 hours after x-ray angiographic recanalization of the CA. The study was performed using an immunoassay Beckman Coulter Access 2.

The Procedure for Venoarterial (V/A) ECMO Connection

ECMO connection was performed under general anesthesia. The choice of anesthetic support was based on the clinical status of the patient and the expected duration of the procedure.

ECMO circuit consisted of CentriMag centrifugal pump (Thoratec Corporation, Pleasanton, CA), PMP membrane oxygenator Quadrox-D (Maquet, Jostra Medizintechnik AG, Hirrlingen, Germany) with an inlet and outlet line, and Bio-Medicus® Femoral Venous and Bio-Medicus® Femoral Arterial cannulas (Medtronic Inc, Minneapolis, MN) for insertion in the patient's vascular bed. Tubing and oxygenator are all coated with Bioline Coating (Maquet, Jostra Medizintechnik AG). Heater Unit HU 35 (Maquet, Jostra Medizintechnik AG) was used for temperature control.

The hypocoagulant state was achieved by bolus dosing of unfractionated heparin (100 units/kg) and supporting of the activated coagulation time from 180 to 220 seconds with continuous intravenous infusion of unfractionated heparin (10–15 units/kg/h).

Bio-Medicus® Femoral Arterial cannulas of diameter 15 to 21 Fr and Bio-Medicus® Femoral Venous cannula of 22 to 26 Fr were used depending on the patient biometric data. A purse string suture was put around the cannula insertion site, and the cannulas were fixed at the insertion site. The distal end of the arterial cannula (inflow to the patient) was located in the common iliac artery or distal abdominal aorta. The distal end of the venous cannula (outflow from the patient) under ultrasound control was located at the junction of the right atrium and superior vena cava. Percutaneous cannulation was performed using a modified Seldinger technique under the control of fluoroscopic guidelines with vascular contrast and ultrasound imaging. After obtaining retrograde access to the common femoral artery, an ECMO arterial cannula was inserted after the dilatation procedure. The venous cannula was inserted in a similar manner.

The initial flow rate of ECMO was 2.4 L/min/m^2 (flow rate from 3.5 to 5.5 L/min), wherein non-pulsating blood flow was used. During the ECMO procedure, the pump flow increased or decreased when necessary (the mean blood pressure of 60–70 mmHg). The flow rate and supply of the oxygen–air mixture were corrected by measuring blood gases depending on the calculated values of oxygen delivery/consumption for each patient.

Disconnection from ECMO was considered when the patient was hemodynamically stable at an ECMO flow rate

of less than 1 L/min/m^2 and had adequate indicators of the gas and acid–base blood composition. Decanulation was performed in the intensive care unit.

The obtained results were statistically assessed by Student's *t* criterion. The data are presented as mean arithmetic (M), based on the results of each investigation ±SD (m). If results showed skewed distribution, then they were summarized as median, and comparisons were performed using the Mann–Whitney *U* test. The differences at $p < .05$ (95.5%) were considered reliable. Logistic regressive analysis of the obtained results was performed by means of a computer program "XLSTAT" (Addinsoft Inc, New York, NY).

RESULTS

After PTCA, all patients who had an extracorporeal circulation support were divided into two groups: survived (group I) and those who died of various causes (deseade), directly in the hospital, or less than 30 days after discharge (group II).

Comparative clinical characteristics of the initial state of survived and deceased patients who underwent PTCA are shown in Table 2.

From Table 2, males prevailed in the group of deceased, which was 21.8% more in comparison with females, in relation to the total number of deaths. There were fewer affected arteries in this group of patients (more than three affected CAs in 61.5% of cases, vs. the same indicator in the group of survivors, which accounted for 90% of cases, $p = .00245$) (Table 2). This fact allows us to conclude that the number of affected CA was not a risk factor for death among the examined category of patients ($r = .009$; $p = .8746$). Wherein, the TIMI flow rates before PTCA also did not have significant intergroup differences ($p = .9231$).

A comparative analysis of concomitant pathology in patients of both groups revealed that diabetes mellitus was more common in patients of group II (23.1% of cases, vs. 10% of cases in group I, $p = .0236$) (Table 2). Data from a one-dimensional logistic regression analysis of indicators associated with mortality showed that diabetes was an independent predictor of fatal consequences (OR = 17.58; 95% CI = 6.47–47.48; $p = .00125$).

It is noteworthy that the presence of MI with a personal history in the II group of patients was less frequently observed in comparison with patients of the I group (53.8% vs. 80%, $p = .00243$) (Table 2). Moreover, in patients of group I, cardiac surgical interventions were significantly more often

Table 2. Comparative characteristics of the initial state of survived and deceased patients who underwent PTCA using ECMO (n = 23).

Indicator		Value		*p* Value
		Survived (n = 10)	Deceased (n = 13)	
Gender (F/M)		3/7	1/12	.04328
Weight (kg)		85.9 ± 24.7	90.2 ± 19.4	.6073
Age (years)		69 ± 5.8	63.2 ± 14.9	.2918
Concomitant pathology, n (%)	AH	9 (90)	13 (100)	.8618
	Diabetes mellitus	1 (10)	3 (23.1)	.0236
	Smoking	5 (50)	6 (46.2)	.6091
	MI in anamnesis	8 (80)	7 (53.8)	.00243
	PTCA in anamnesis	3 (30)	0 (0)	.03813
	CABG in anamnesis	1 (10)	0 (0)	.04892
	CRF in anamnesis	2 (20)	6 (46.2)	.03921
	Cerebrovascular; in anamnesis	4 (40)	3 (23.1)	.05935
Number of affected CA	Average in group	3.3 ± 1.4	2.6 ± 1.3	.00245
	>3	9 (90%)	8 (61.5%)	.00395
Type of infarction-associated CA, n (%)	Left main CA	4 (40)	3 (23.1)	.1181
	Left anterior descending	5 (50)	5 (38.5)	.2031
	Left circumflex CA	0 (0)	1 (7.7)	.3512
	RCA	1 (10)	4 (30.8)	.00013
TIMI flow before PTCA		.32 ± .08	.35 ± .07	.9231
Hemoglobin (g/L)		117.5 ± 16.8	107.3 ± 20.5	.1183
pH		7.37 ± .16	7.39 ± .17	.4914
pCO2 (mmHg)		41.01 ± 5.31	38.8 ± 6.2	.6920
Lactate (mmol/L)		1.4 ± .4	4.7 ± 2.5	.03381
Creatinine (μmol/L)	Average in group	202.7 ± 114.6	275.7 ± 170.5	.05812
	≤200	3 (30%)	9 (69.2%)	.00281
CPK-MB (units/L)	Average in group	316.4 ± 264.4	540.8 ± 392.2	.00728
	>170	3 (30%)	11 (84.6%)	.00361
Troponin (ng/mL)	Average in group	10.5 ± 3.1	17.6 ± 2.9	.1329
	>.16	8 (80%)	12 (92.3%)	.0441
EF (%)	Average in group	38.1 ± 20.9	39.3 ± 24.2	.4326
	<40%	5 (50%)	6 (46.2%)	.5833

Table 3. Procedural parameters in survived and deceased patients who underwent PTCA using ECMO (n = 23).

Indicator	Value		*p* Value
	Survived (n = 10)	Deceased (n = 13)	
Thrombo-extraction, n (%)	2 (20)	5 (38.5)	.0374
"No reflow" phenomenon, n (%)	1 (10)	3 (23.1)	.0031
Cardiac arrest before ECMO, n (%)	1 (10)	9 (69.2)	.000154
CPR duration (minutes)	1 patient (10%)–40′	13 patients (100%)–35′–80′ (56.2 ± 19.4′)	.0098
Epinephrine (μg/kg/min)	1 patient (10%)–.08–.25	12 patients (92.3%) .12–.47	.0033
Norepinephrine (μg/kg/min)	6 patients (60%)–.04–.07	13 patients (100%)–.16–.5	.0442
Dobutamine (μg/kg/min)	9 patients (90%)–.06–.08	13 patients (100%)–12–15	.3091
TIMI flow after PTCA, grade	2–3 (2.4 ± .37)	1–2 (1.5 ± .48)	.00138

associated with a personal history (stenting of CA—30% of cases and CABG—in 10% of cases) (Table 2).

After analyzing concomitant pathology, it was found that the presence of chronic renal failure (CRF), which was 2.31 times more common in patients of group II (Table 2), was an independent predictor of mortality (OR = 20.81; 95% CI = 5.95–72.21; $p = .00014$) in the examined groups of patients. These predictors also included myocardial ischemia, which developed in the basin of the right CA (RCA) (OR = 25.51; 95% CI = 8.27–79.12; $p = .00013$). This lesion was found 20.8% more often in deceased patients, vs. a similar type of ischemia in group I (Table 2).

In group II of patients, ischemic myocardial damage was more pronounced, as evidenced by the indicators of CPK-MB and troponin I (Table 2).

When analyzing the condition of the examined patients during PTCA, it was revealed that the thrombo-extraction procedure was performed 18.5% ($p = .037$) more often in patients of group II (Table 3).

In this group, the "no reflow" phenomenon was also recorded in a larger percentage of cases (23.1% in group II, vs. 10% in group I) (Table 3). The aforementioned circumstances also led to a lesser procedural success in the examined group II (TIMI flow after PTCA in group I was 2.4 ± .37 vs. 1.5 ± .48 in group II) ($p = .00138$) (Table 3).

The fact of more frequent cardiac arrest in the operating room (10% in group I patients, vs. 69.2% in patients of group II) ($p = .000154$) (Table 3) and higher doses of adrenergic agonists, which were used to maintain the contractile ability of the myocardium (Table 3), is attributable to more severe myocardial ischemia (according to cardiospecific enzymes [Table 2] in patients of group II and lower procedural success of CA recanalization).

The recorded indicators during ECMO in survived and deceased patients after PTCA are shown in Table 4.

After analysis of these indicators, it was found that ECMO connection before cardiac events was significantly more often used in the group of survived patients (90% of cases in patients of group I and 0% of cases in patients of group II) ($p = .0000001$, Table 4).

The length of the ECMO run in hours was 17.8 ± 8.2 in patients of group I vs. 73.6 ± 23.7 hours in group II (Table 4) ($p = .00023$) patients. Complications arose that were significantly more common in patients of group II (Table 4). So, ischemia of the lower extremities, which was attributed to the prolonged presence of the cannula in the femoral artery area, was observed 18.8% more often in patients of group II ($p = .0014$), bleeding: 13.1% ($p = .0012$), and hemolysis: 7.7% (p = .129) (Table 4).

DISCUSSION

This study showed that the use of the ECMO technique with a V/A connection before PTCA was characterized by relative safety, low complications and a significant improvement in

Table 4. Indicators and complications during ECMO in survived and deceased patients (n = 23).

Indicator		Value		*p* Value
		Survived (n = 10)	Deceased (n = 13)	
Type of ECMO		V/A: 10 (100%)	V/A: 13 (100%)	–
Connection to ECMO before PTCA, n (%)		9 (90%)	0 (0%)	.0000001
Connection to ECMO during PTCA, n (%)		1 (10%)	13 (100%)	.0000001
ECMO duration (hours)		2.3–83.4 (17.8 ± 8.2)	13.5–128.2 (73.6 ± 23.7)	.00023
ECMO complications, n (%)	Lower extremity ischemia	2 (20)	4 (30.8)	.0014
	Bleeding	1 (10)	3 (23.1)	.0012
	Hemolysis	0 (0)	1 (7.7)	.1291
Mortality on ECMO, n (%)		0 (0)	10 (76.9)	–
Mortality during 30 days after PTCA, n (%)		0 (0)	3 (23.1)	–

survival in the examined patients with acute coronary syndrome or type 1 MI. Till date, no randomized studies regarding the effectiveness of ECMO during percutaneous coronary intervention (PCI), except retrospective, have been conducted, which requires further study of this problem.

Research on the efficacy of the prophylactic use of extracorporeal hemodynamic support during PTCA in high-risk patients has begun as early as the 1990s by Tierstein et al. (15). The researchers found that patients who received prophylactic cardiopulmonary support had a reliably frequent development of femoral complications and the need for blood products, against the background of a similar level of hospital mortality. At the same time, in this study, when comparing the efficacy of the prophylactic extracorporeal hemodynamic sublimation in patients with ejection fraction (EF) <20% with the application of this technique "in case of necessity," a significant decrease in hospital mortality was found.

Subsequently, Vainer et al. (16) described their experience in the prophylactic usage of ECMO among 15 patients who underwent PCI under general anesthesia. All patients were weaned from ECMO in an angiography laboratory and from mechanical ventilation in an average 5.1 ± 3.3 hours. Moreover, the study did not reveal a single case of hospital mortality or a single case of periprocedural heart attack. As the authors had noted, complications were associated with cannulation; there was one case of local inguinal bleeding that did not require surgical intervention. On average, patients were discharged after 3.2 ± 2.8 days. A possible reason for such good results of the usage of "prophylactic" ECMO was the absence of cardiogenic shock in patients included in the study because they should be considered at high risk for a relatively considerable age (average age 72 ± 9 years) and the presence of concomitant diseases.

At the same time, in a retrospective study by Sheu et al. (17), on the efficacy of early ECMO usage in acute myocardial infarction, it was found that deep cardiogenic shock after percutaneous coronary interventions, without the technical feasibility of using extracorporeal blood circulation support, had a 30-day mortality rate of 72.0% (18/25), whereas with ECMO, this indicator significantly decreased to 39.1% (18/46).

In our study of ECMO usage, both before and during PTCA, mortality rate was 56.52% (13/23). However, if we take into account patients who were connected to ECMO before revascularization and the onset of cardiac events, this indicator was 0%.

Starting ECMO before or during PTCA depended on the patient's initial severity status. To stabilize hemodynamics with high doses of inotropes, the first thing to do is to establish a recanalization of the affected CA. According to the authors, Li X., launching ECMO before PCI can delay the start of recanalization therapy from "door-to-balloon." However, during ECMO, myocardial oxygen consumption increases, which can lead to embolic complications and bleeding (18). Available scientific developments do not have much information that would use ECMO for PCI against the background of myocardial infarction with ST segment elevation. According to Lee et al. (19), this information does not carry statistical significance; however, the results are quite favorable. That is why it is important to study the results of therapy of such patients.

Favorable results from using routine ECMO during PTCA were also reported in a Van den Brink et al. (20) retrospective study (2018). So, the researchers found that among 12 patients who underwent ECMO during PTCA, the overall mortality rate was 42% (5/12), but mortality during ECMO was 33% (4/12). In the study, complications were recorded in six of 12 patients: one patient had serious neurological disorder, two patients had hemorrhage at the site of the cannula, two more had ischemia of the lower extremities, and one patient had hemorrhage in another place. It is worth noting that in this work, ECMO was connected already during PTCA when there was a life-threatening arrhythmia or when cardiac activity stopped.

At the same time, in our study, the 30-day mortality rate in patients who underwent ECMO connections when complications already appeared was 93% (13/14). Such high mortality rates, in our opinion, are attributed to several factors, including the presence of comorbid diseases in patients with diabetes mellitus and CRF. It should also be noted that in both studies, the interventricular branch of the left CA turned out to be a frequent heart attack–associated artery.

Another method of extracorporeal support in our cath lab is the use of intra-aortic balloon counterpulsation. Earlier, intra-aortic balloon counterpulsation was widely used for such patients, but today, it is not the primary treatment method. This conclusion is based on the latest guidelines in the SHOCK-2 study (21). In addition, the Cochrane study found that intra-aortic balloon contra-pulsation did not increase survival in patients with postinfarction cardiogenic shock and in patients who had PCI (94). In conclusion, the use of intra-aortic balloon counterpulsation method is not the gold standard for the treatment of patients with heart attack–related cardiogenic shock. Most likely, this technique is extremely subjective (22).

Thus, as the results of both our research and the work of the aforementioned authors had shown, the usage of ECMO in high-risk patients during PTCA is characterized by a significant increase in survival even in such a complex cohort of patients.

CONCLUSION

Diabetes mellitus, CRF, and damage to the RCA are independent predictors of mortality during PTCA in

patients with CHD. The number of affected coronary arteries was not a risk factor for death in the examined category of patients. The routine use of ECMO in high-risk patients (acute coronary syndrome and myocardial infarction) with PTCA is a positive prognostic factor for patient survival.

LIMITATIONS

Our study has several limitations. This is a retrospective study involving a small number of patients. In addition, in the study, we did not make provision for a control group of patients and its comparative analysis.

In future studies, there is need to randomize patients who underwent primary percutaneous coronary intervention in the presence of myocardial infarction followed by cardiogenic shock, compared with treatment with VA-ECMO.

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