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Echocardiographic assessment of left ventricular myocardial strain, as a non-invasive method for diagnosing pulmonary hypertension in patients with end-stage chronic kidney disease

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Abstract

Background. *Pulmonary hypertension is a common complication of chronic kidney disease, with incidence of up to 50%. Currently, the prognostic significance of non-invasive diagnostic methods for*

pulmonary hypertension in patients with chronic kidney disease remains relevant.

Aim. *To determine the significance of transthoracic echocardiography in diagnosing pulmonary hypertension in patients with end-stage chronic kidney disease.*

Material and methods. *The study group consisted of 53 patients with chronic kidney disease stage 5D who were evaluated for kidney transplantation at the N.V. Sklifosovsky Research Institute for Emergency Medicine in 2022. A control group was represented by 24 healthy volunteers. Transthoracic echocardiography was performed on all patients according to a standard protocol, with determination of left ventricular myocardial strain indices.*

Results. *A statistically significant correlation was found between the left ventricular global longitudinal strain and pulmonary artery systolic pressure - r=0.488 (p<0.001), as well as between the left ventricular global circumferential strain and pulmonary artery systolic pressure (r=0.545, p<0.001). Regression analysis showed that an increase in pulmonary artery systolic pressure by 1 mmHg increased the odds of lethal outcome by 13% (Odds ratio: 1.13; 95% Confidence interval: [1.05;1.22], p=0.002).*

Conclusions. *Hemodialysis patients are characterized by the development of pre-capillary pulmonary hypertension, which significantly affects their prognosis. Determination of left ventricular myocardial strain indices based on echocardiography provides additional information on the hemodynamics of the pulmonary circulation without using invasive diagnostic methods.*

Keywords: end-stage chronic kidney disease, pulmonary hypertension, chronic heart failure, global longitudinal strain of the left ventricular

myocardium, global circumferential strain of the left ventricular myocardium

Conflict of interests Authors declare no conflict of interest

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CHD, coronary heart disease
CHF, chronic heart failure
CI, confidence interval
CKD S5, chronic kidney disease, stage 5
CKD S5D, chronic kidney disease, stage 5 (dialysis)
CKD, chronic kidney disease
CO, cardiac output
EchoCG, echocardiography
EDD, end-diastolic dimension
EDV, end-diastolic volume
EEs, expected events
EF, ejection fraction
eMPASP, estimated mean pulmonary artery systolic pressure
ePAWP, estimated pulmonary artery wedge pressure
ESV, end-systolic volume
IVSth, interventricular septum thickness
LA, left atrium
LV, left ventricle
LVH, left ventricular hypertrophy
LVMMI, left ventricular myocardial mass index
LVPWth, left ventricle posterior wall thickness
MPAP, mean pulmonary artery pressure
OR, odds ratio
PASP, pulmonary artery systolic pressure
PCWP, pulmonary capillary wedge pressure
PH, pulmonary hypertension
PVR, pulmonary vascular resistance
RA, right atrium
RV FAC, right ventricle fractional area change

RV, right ventricle
SV, stroke volume
TAPSE, tricuspid annular plane systolic excursion

Introduction

According to world literature, chronic kidney disease (CKD), including its end stage, is accompanied by the development of pulmonary hypertension (PH) [1–3]. A sustained increase in pulmonary artery pressure can ultimately lead to decompensation of right ventricular failure and death [4]. In patients with chronic kidney disease, stage 5 (CKD S5), PH is an independent predictor of cardiovascular complications and mortality [5]. The exact mechanisms of PH development in CKD S5 are unknown. It is believed that the main pathophysiological components may be myocardial dysfunction, volume overload, and electrolyte imbalance [6]. Diagnosis of PH is based on measuring pressure in the pulmonary circulation system using invasive procedures, which is associated with the development of complications. Studying the incidence of complications related to catheterization of the superior vena cava system, including the internal jugular vein for the purpose of Swan-Ganz catheter placement, S.I. Lomeiko and E.N. Butova (2020) reported that the incidence of complications could reach 4.2% for patients in the general population [7].

Thus, there is a need to optimize making PH diagnosis in patients with CKD S5 in order to reduce the risks of complications. One of the promising non-invasive methods for diagnosing PH may be echocardiography (EchoCG), however, this technique has a limited ability to determine the impact of such hemodynamic parameters as cardiac output (CO), pulmonary capillary wedge pressure (PCWP) and pulmonary vascular resistance (PVR) in connection with the peculiarities

of PH pathogenesis in this group of patients, as well as the subjective nature of velocity measurements [8].

Aim. To determine the significance of transthoracic echocardiography in the diagnosis of pulmonary hypertension in patients with end-stage chronic kidney disease.

Material and methods

The study included 53 patients with associated cardiovascular diseases (22 men (42.6%) and 31 women (57.3%), the median age 50 (39;60) years) who received hemodialysis and were admitted in the hospital of the N.V. Sklifosovsky Research Institute for Emergency Medicine in 2022. Patients included in the study received a combination antihypertensive therapy (β-blockers, calcium antagonists, imidazoline receptor blockers). Specific therapy for pulmonary hypertension was not performed. The control group consisted of 24 healthy volunteers. Among them, there were 5 men (20.8%) and 19 women (79.2%); their median age was 27 (24;34.25) years. All study participants underwent transthoracic echocardiography according to a standard protocol. The comorbidities in the patients included in the study are presented in Table 1.

Table 1. The incidence rate of comorbid pathology in patients with end-stage chronic kidney disease

Comorbid pathology	
Arterial hypertension, n (%)	49 (92.45%)
Chronic heart failure (CHF), n (%)	27 (50.94%)
Previous myocardial infarction, n (%)	16 (30.18%)
Diabetes type 1, n (%)	6 (11.32%)
Previous stroke, n (%)	6 (11.32%)
Diabetes type 2, n (%)	5 (9.43%)

All patients underwent a transthoracic two-dimensional echocardiography (EchoCG) using a Phillips Epiq 7 device to determine the structural and functional parameters of the heart, and a speckle-tracking EchoCG to assess the longitudinal and circumferential left ventricular myocardial strain.

Patients with LV ejection fraction (EF) (calculated by Simpson method) <50% were diagnosed with LV systolic dysfunction. To define LV diastolic dysfunction, E/A and E/e' ratios were calculated using Doppler velocity measurements, E/A ratio <0.75 or >1.8 and/or E/e' >14 were qualified as LV diastolic dysfunction. Patients with interventricular septal thickness or LV posterior wall thickness ≥12 mm were diagnosed with LV hypertrophy (LVH).

Estimated mean pulmonary artery systolic pressure (eMPASP) was determined using continuous wave Doppler ultrasound using the formula: eMPASP = 4 × [TR Vmax]^2 + right atrium (RA) pressure, where TR Vmax is the peak velocity of tricuspid regurgitation. Mean pulmonary artery pressure (MPAP) was calculated using the formula: MPAP = 4 × [PR Vmax]^2 + right atrium (RA) pressure, where PR Vmax is Vmax of pulmonary regurgitation (PR); Vmax is the maximum velocity of the early diastolic PR peak in continuous wave Doppler mode [9–10].

To calculate pulmonary artery wedge pressure (PAWP), the following formula was used: PAWP = 1.24 × (E/e′) + 1.9, where E is the maximum velocity of the early peak of antegrade transmitral blood flow in pulsed-wave Doppler mode; e' is the mean of mitral annulus velocities of movements as measured at the annular interventricular septal and lateral wall sides in early diastole in pulsed-wave tissue Doppler mode [11].

Pulmonary vascular resistance (PVR) was calculated using the formula: (MPAP – PAWP)/cardiac output [12].

Depending on the presence of PH in patients receiving hemodialysis, two groups were formed: group I consisted of 22 patients with PASP values ≥ 35 mm Hg, group II included 31 patients with PASP values < 35 mm Hg.

Statistical processing

Statistical data processing was performed using jamovi software, version 2.1.16 for the macOS Monterey operating system. For statistical processing, methods of parametric and nonparametric statistics were used. To determine the normality of the distribution, the Shapiro–Wilk test was used. Quantitative data are presented using the median and interquartile range (Me (Q1;Q3)). Qualitative data are presented using absolute numbers and percentages (n (%)). Quantitative data were compared using the Mann–Whitney U test. Qualitative data were compared using the χ^2 -Pearson test for expected events (EE) > 10, the χ^2-Pearson test with Yates' correction for continuity for EE values from 5 to 9, and Fisher's test for EEs < 5. The correlation analysis was performed using non-parametric Spearman test, the closeness of the correlation relationship was assessed using the Chaddock scale. The probability of a fatal outcome was determined using the logistic regression method with calculation of the odds ratio (OR) and 95% confidence interval (CI). For all criteria, a statistical significance level of 5% was used, statistical differences were confirmed at $p<0.05$.

Results

The main parameters of echocardiography in patients on hemodialysis and the control group are presented in table. 2.

Table 2. Echocardiography parameters in the study groups

EchoCG parameter	Patients receiving hemodialysis (n=53)	Control group (n=24)	p-value
LV ejection fraction ***, %	60 (59.0;62.0)	62.0 (60.0;63.3)	**0.03***
EDV***, ml	92 (69.0;67.5)	75.0 (67.5;77.0)	**0.001***
ESV***, ml	35 (27.0;45.0)	30.0 (25.8;31.0)	**0.002***
SV***, ml	53 (42.0;65.0)	45.0 (40.5;46.0)	**0.002***
CO***, L	3.83 (3.04;4.55)	3.10 (2.51;3.32)	**0.001***
EDD***, cm	4.60 (4.0;4.90)	4.0 (3.75;4.00)	**<0.0001***
LA diameter***, cm	3.8 (3.4;4.1)	2.9 (2.45;3.25)	**<0.0001***
LA volume***, ml	60 (41.0;70.0)	32.0 (29.0;37.0)	**<0.0001***
RA volume***, ml	50 (35.0;60.0)	31.0 (28.0;36.0)	**<0.0001***
LVMMI***, g/m^2	104 (90.0;142)	60.0 (55.0;60.0)	**<0.0001***
IVSth***, cm	1.4 (1.2;1.5)	0.9 (0.8;1.0)	**<0.0001***
LVPWth***, cm	1.0 (0.9;1.10)	0.8 (0.7;0.825)	**<0.0001***
E/A***	0.9 (0.7;1.2)	1.2 (0.975;1.35)	**0.006 ***
E/e'***	6.80 (5.40;8.40)	5.70 (5.20;7.60)	0.07*
Type of diastolic dysfunction			
Type 1****	21 (39.62%)	0 (0%)	**0.0003****
Type 2****	6 (11.32%)	0 (0%)	0.08**
Type 3****	5 (9.34%)	0 (0%)	0.1241**
PASP***, mm Hg	32 (26.0;38.0)	22.5 (20.8;25.3)	**<0.0001***
ePAWP***, mm Hg.	10.4 (8.65;12.4)	9.03 (8.40;11.4)	0.07*
PVR***, Wood units	5.73 (4.43;6.81)	4.40 (3.89;4.87)	**0.00182***
Mitral valve insufficiency			
Grade1****	41 (73.35%)	20 (83.33%)	0.3422**
Grade 2****	12 (22.64%)	0 (0%)	**0.0117****

Tricuspid valve insufficiency			
Grade 1****	31 (58.49%)	22 (91.66%)	**0.0038****
Grade 2****	21 (39.62%)	0 (0%)	**0.0003****
Grade 3****	1 (1.88%)	0 (0%)	0.5**
Global longitudinal strain***, %	- 13.1 (-15.5;-11.2)	-21.1 (-21.1;-20.3)	**<0.0001***
Global circumferential strain***, %	-27.3 (-30.4;-21.4)	-33.1 (-33.2;-31.0)	**<0.0001***

Notes: * Mann–Whitney U test, ** Pearson χ^2 test, *** Me (Q1; Q3), **** n (%)

LVMMI, left ventricular myocardial mass index; EDV, end-diastolic volume; EDD, end-diastolic dimension; ESV, end-systolic volume; LV, left ventricle; LA, left atrium; PVR, pulmonary vascular resistance; RA, right atrium; ePAWP, estimated pulmonary artery wedge pressure; CO, cardiac output; PASP, pulmonary artery systolic pressure; LVPWth, left ventricle posterior wall thickness; IVSth, interventricular septum thickness; SV, stroke volume; E/A, the ratio of the maximum velocity of early diastolic filling of the left ventricle to the maximum velocity of filling of the left ventricle in atrial systole; E/e', the ratio of peak early diastolic transmitral flow velocity to peak early diastolic lateral mitral annular velocity.

According to Table 2, statistically significant differences in transthoracic echocardiography were observed for almost all parameters, except for E/e' and ePAWP. PH defined as an increase in PASP > 35 mmHg was identified in 22 patients (41.50%) receiving hemodialysis.

The rate of comorbidities in the groups formed with regard to the PH presence is given in Table 3.

Table 3. Incidence rate of comorbidities in patients receiving hemodialysis, with regard to the presence of pulmonary hypertension

Clinical characteristics	Group I (n=22)	Group II (n=31)	p-value*
Arterial hypertension, n (%)	21 (95.45)	28 (50, 90)	**0.0003**
Chronic heart failure, n (%)	15 (68.18)	12 (21.81)	**0.0001**
CHD, n (%)	11 (50)	5 (9.09)	**0.0001**
Previous stroke, n (%)	5 (22.72)	1 (1.81)	**0.0021**
Diabetes type 1, n (%)	2 (9.09)	4 (7.27)	0.7891
Diabetes type 2, n (%)	3 (13.63)	2 (3.63)	0.1098

Note: * Pearson χ^2 test; CHD, coronary heart disease

In patients with PH (group I), arterial hypertension, coronary artery disease, CHF, and stroke were statistically significantly more common.

The main EchoCG parameters in patients of group I and group II are presented in Table 4.

Table 4. Echocardiographic parameters in patients receiving hemodialysis, with regard to the presence of pulmonary hypertension

EchoCG parameter	Group I (n= 22)	Group II (n=31)	p-value
LV ejection fraction***, %	59.5 (58.0;60.0)	60 (59.0;62.5)	**0.01***
EDV***, ml	107 (80.5;127)	83 (63.5;93.5)	**<0.001***
ESV***, ml	40.5 (32.3;55.0)	34 (25.0;39.5)	**<0.001***
SV***, ml	62.5 (48.3;70.0)	50 (37.0;57.5)	**<0.001***
CO***, L	3.99 (3.36;4.96)	3.76 (2.95;4.18)	**<0.001***
EDD***, cm	4.90 (4.53;5.27)	4.30 (3.95;4.80)	**<0.001***
LA diameter***, cm	4.10 (3.68;4.75)	3.6 (3.30;3.90)	**0.003***
LA volume***, ml	74.5 (60.0;89.5)	48.0 (35.5;66.0)	**<0.001***
RA volume***, ml	56.5 (45.8;70.0)	43 (29.0;53.5)	**0.008***
LVMMI***, g/m^2	134 (101;153)	95 (86.0;116)	**0.012***
IVSth***, cm	1.45 (1.22;1.60)	1.40 (1.20;1.50)	0.36*
LVPWth***, cm	1.00 (0.900;1.10)	0.90 (0.80;1.10)	0.2*
E/A***	0.95 (0.725;1.45)	0.90 (0.65;1.00)	0.12*
E/e'***	8.25 (6.58;11.3)	5.80 (4.90;7.75)	**0.001***
Type of diastolic dysfunction			
Type 1****	7 (31.81%)	14 (45.16%)	0.3321**
Type 2****	4 (18.18%)	2 (6.45%)	0.1883**
Type 3****	4 (18.18%)	1 (3.22%)	0.0689**

RV EDD, cm	33 (31;35)	32 (29;34.8)	0.16
TAPSE, mm	22 (18;25)	23 (19.3;25)	0.682
RV FAC, %	45 (40;50)	46 (42;55)	0.044
PASP***, mm Hg.	40.0 (35.3;45.0)	26 (25.0;30.0)	**<0.001***
ePAWP***, mm Hg.	12.2 (10.1;16.0)	9.15 (8.03;11.6)	0.001*
PVR***, Wood units	6.23 (5.67;10.8)	5.30 (3.89;6.43)	**0.011***
Mitral valve insufficiency			
Grade 1****	13 (59.09%)	28 (90.32%)	**0.008****
Grade 2****	9 (40.90%)	3 (9.67%)	**0.008****
Tricuspid valve insufficiency			
Grade 1****	7 (31.81%)	24 (77.41%)	**0.001****
Grade 2****	14 (63.63%)	7 (22.58%)	**0.0029****
Grade 3****	1 (4.54%)	0 (0%)	0.2355**
Global longitudinal strain***, %	-12.3 (-16.1;-10.9)	-13.3 (-14.9;-12.5)	0.96*
Global circumferential strain***, %	-24.5 (-29.2;-19.7)	-28.4 (-33.2;-24.2)	**0.03***

Notes: * Mann–Whitney U test; ** Pearson χ^2 test; ***Me (Q1; Q3); **** n (%)

TAPSE (tricuspid annular plane systolic excursion;, RV FAC right ventricle fractional area change; LVMMI, left ventricular myocardial mass index; EDV,- end-diastolic volume; EDR, end-diastolic dimension, RV EDD, right ventricle end-diastolic dimension, ESV, end-systolic volume, LV, left ventricle; LA, left atrium; PVR, pulmonary vascular resistance; RA, right atrium; ePAWP, estimated pulmonary artery wedge pressure, CO cardiac output; PASP, pulmonary artery systolic pressure; IVSth, interventricular septal thickness; LVPWth, left ventricle posterior wall thickness; SV, stroke volume; E/A, the ratio of the maximum velocity of early diastolic filling of the left ventricle to the maximum velocity of filling of the left ventricle in atrial systole; E/e', the ratio of peak early diastolic transmitral flow velocity to peak early diastolic lateral mitral annular velocity.

As can be seen from the table presented, 4, patients with PH were characterized by statistically significantly higher rates of EDV, ESR, SV, and CO. Also noteworthy was the increase in the diameter and volume of the left atrium.

The RA volume was statistically significantly higher in group I, the RV size and function were within normal values.

When conducting a correlation analysis, a statistically significant correlation was revealed between the parameters of LV myocardial global longitudinal strain and PASP − r=0.488 (p<0.001).

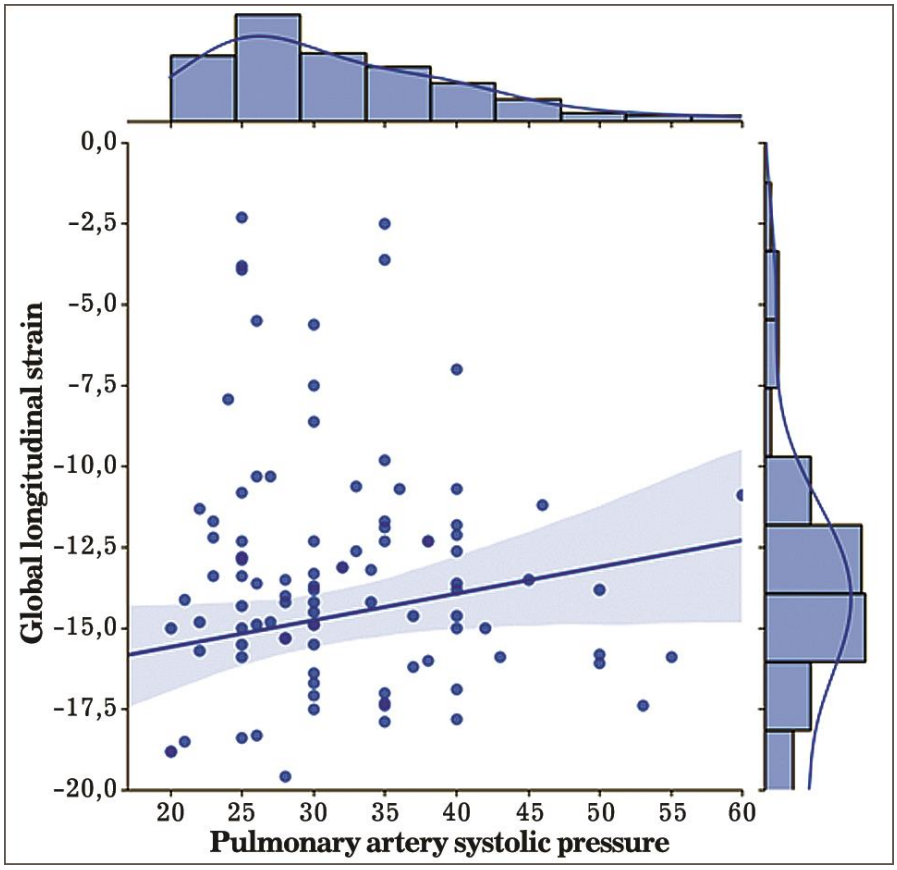

Fig. 1. Correlation between systolic pressure in the pulmonary artery and the global longitudinal strain of the left ventricular myocardium

This pattern was described by the equation:

$$Y_{PASP} = 40.872+0.657 \times X_{\text{global longitudinal strain}},$$

where Y_{PASP} is the predicted value of PASP, mm Hg;

$X_{\text{global longitudinal strain}}$ is the value of the LV global longitudinal strain in %

The resulting model was statistically significant ($p<0.001$). The coefficient of determination (R^2) was 0.114. A correlation was also identified between the parameter of the global circumferential myocardial strain and PASP ($r=0.545$, $p<0.001$), which was described by the following equation:

$$Y_{PASP} = 46.086 + 0.56 \times X_{\text{global circumferential myocardial strain}},$$

where Y_{PASP} is the predicted PASP value, mmHg.

$X_{\text{global circumferential myocardial strain}}$ is the value of the LV myocardial global circumferential strain in %.

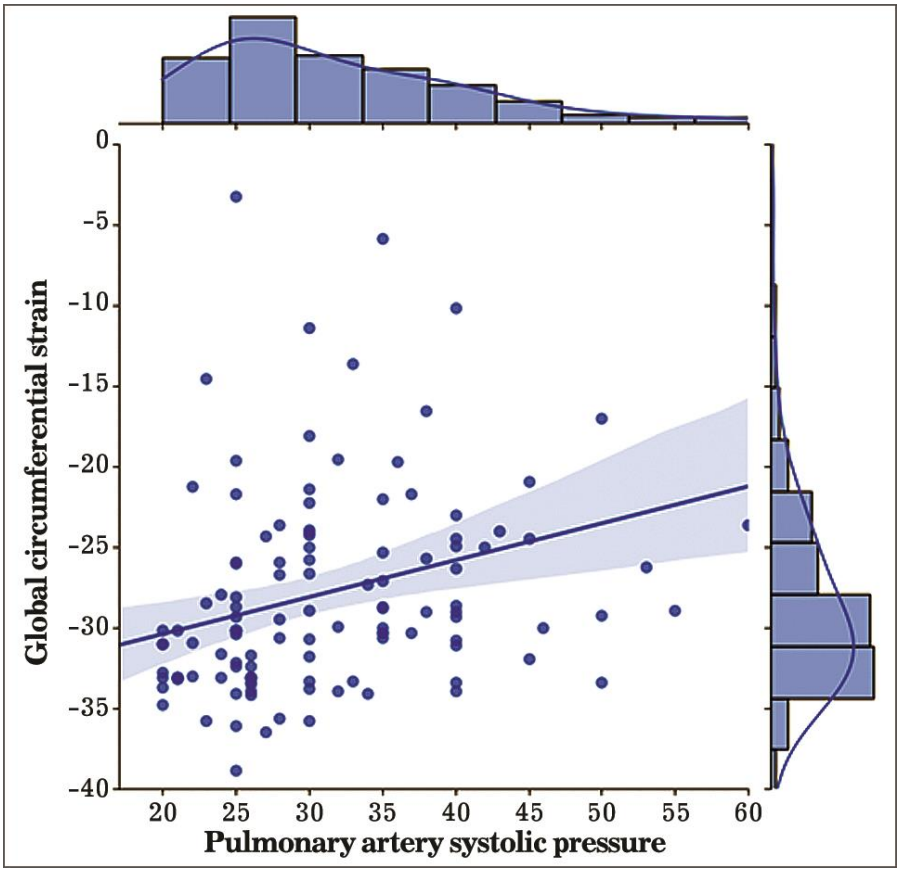

Fig. 2. Correlation between systolic pressure in the pulmonary artery and the global circumferential strain of the left ventricular myocardium

During the study period, 5 patients died. The causes of death in patients are presented in Table. 5.

Table 5. Characteristics of deceased patients

Patient No.	PASP, mm Hg	Cause of death
Patient 1	55	Multiple organ failure syndrome
Patient 2	70	Sepsis
Patient 3	30	Multiple organ failure syndrome
Patient 4	45	Sepsis
Patient 5	43	Acute cardiovascular failure

When conducting regression analysis, we found that with the PASP increase by 1 mm Hg, the odds of developing a fatal outcome increased by 1.13 times (OR: 1.13; 95% CI: [1.05;1.22], p=0.00177) (Table 6).

Table 6. Regression analysis results

Predictor	Coefficient	S.E.	Z	OR	95% CI	p-value
Constant	-9.278	3.0572	-3.03			
Increase in PASP by 1 mm Hg	0.138	0.0567	2.44	1.15	1.03;1.28	0.01468

Discussion

The results of our study confirm the world literature data, which indicate that patients on hemodialysis have pronounced structural changes in the myocardium expressed in an increase in LVMMI and hypertrophy of the LV walls [13]. Our results, demonstrated that in the group of CKD S5D patients, the LVMMI (see Table 2) was 104 (90.0;142) g/m^2, IVSth was 1.4 (1.2;1.5) cm, LVPWth was 1.0 (0.9;1.10) cm, which corresponded to moderate LVH. Diastolic dysfunction was diagnosed in 60.28%, which may indicate the presence of CHF with intact EF. The median LV EF was 60 (59.0;62.0)%, and despite the high prevalence of CHF in the study groups (68.18% and 21.81%, respectively), a decrease in LV EF <50% was observed only in 2 patients (9.09%) of group I.

There was a slight decrease in LV EF in group I (59.5 (58.0;60.0)%), compared to patients of group II (60 (59.0;62.5)%). Despite statistically significant differences in LV EF between groups I and II (p=0.01), this decrease in EF was not clinically significant and the parameter was corresponded to normal.

When stratifying the patient sample into groups with regard to the PH presence, we found that PH patients had statistically significant higher values of volumetric parameters of the heart chambers, which was apparently associated with volume overload and arterial calcification.

It is known that patients on hemodialysis often develop so-called uremic cardiomyopathy, which contributes to the pressure increase in the pulmonary artery system; and also the risk of developing pulmonary edema increases. An important feature of this cardiomyopathy is that LV dysfunction may not be detected, being latent [14].

When analyzing the EchoCG parameters reflecting the pressure in the pulmonary circulation system, we found that the median PASP was 32 (26.0;38.0) mmHg, the median PAWP was 10.4 (8.65;12.4) mmHg, the median PVR was 5.73 (4.43;6.81) Wood units.

By conducting a correlation analysis, we established a relationship between parameters of LV myocardial strain and PASP. Thus, moderate strength correlations were identified between the parameters of LV myocardial global circumferential strain and PASP (r=0.488 (p<0.001) and r=0.545 (p<0.001), respectively).

Increase in PASP was found to be associated with an increased risk of death in hemodialysis patients (OR: 1.13; 95% CI [1.05,1.22], p=0.00177). According to a study by M. Rroji et al that included 125 patients who had received renal replacement therapy for more than 3 months (the follow-up of 2 years), the PH prevalence, according to the transthoracic echocardiography results, was 28%, the mean PASP was

33.46 ± 5.38 mm Hg. The authors concluded that PH was a risk factor for mortality in this group of patients [15]. According to current clinical guidelines, an increase in PASP > 35 mm Hg, a decrease in PAWP < 15 mm Hg, and an increase in PVR > 3 Wood units made the criteria for precapillary PH. An important caveat is that to make a precapillary PH diagnosis, these parameters must be measured using a right heart catheterization [12]. The procedure of the right heart catheterization using a Swan-Ganz catheter may be associated with the development of a number of complications, including right atrium rupture, tricuspid valve damage, right ventricular perforation, the development of infective endocarditis, and pulmonary artery damage with subsequent thrombosis [16].

Thus, an urgent task is to search for alternative non-invasive methods for assessing the hemodynamics of the pulmonary circulation, and therefore additional studies are required to more accurately determine the diagnostic value of speckle-tracking imaging echocardiography in patients with CKD S5.

Conclusions

1. Determining the indicators of the left ventricular myocardium strain by echocardiography investigation provides additional information on the pulmonary circulation hemodynamics without the use of interventional diagnostic tools.

2. The study demonstrated the relationship between the parameters of the left ventricular myocardial strain and pulmonary artery systolic pressure. Moderate strength correlations were revealed between the parameters of the left ventricular myocardium global circumferential strain and the pulmonary artery systolic pressure ($r=0.488$ ($p<0.001$) and $r=0.545$ ($p<0.001$), respectively).

3. It has been established that with the increase in pulmonary artery systolic pressure by 1 mm Hg the odds of developing a fatal outcome increased by 1.13 times (odds ratio: 1.13; 95% confidence interval [1.05;1.22], p = 0.00177).

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