Original article

Ultrasound diagnosis of pulmonary edema and systemic circulation congestion in patients with atrial fibrillation and chronic heart failure

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Abstract: Rationale — Fluid retention is among the most common causes of heart failure decompensation. The goal of our study was to evaluate the sensitivity and specificity of the ultrasound method for evaluating congestive phenomena in both systemic and pulmonary circulatory systems in patients with atrial fibrillation (AF) and chronic heart failure (CHF).

Material and Methods — The study included 28 patients with paroxysmal AF, with or without CHF, who were scheduled for radiofrequency pulmonary vein isolation. The maximum and minimum diameters of the right superior pulmonary vein (RSPV) and inferior vena cava (IVC) were measured via echocardiography on expiration. Mean blood pressure in both right and left atria was measured intraoperatively. We calculated the correlations between maximum and minimum diameters of the RSPV and a mean pressure in the left atrium, as well as between IVC on expiration and a mean pressure in the right atrium, and evaluated the sensitivity and specificity of ultrasound diagnostics for evaluating congestive phenomena in both systemic and pulmonary circulation.

Results — We established positive correlation between the minimum diameter of RSPV and invasively measured mean left atrial pressure (R=0.65, P<0.05), and between invasively measured mean right atrial pressure and IVC diameter on expiration (R=0.49, P<0.05). The proposed method sensitivity for detecting pulmonary venous hypertension (PVH) on the basis of the maximum RSPV diameter ≥21.7 mm was 75%, and the specificity was 86%. The sensitivity and specificity for detecting PVH on the basis of the minimum RSPV diameter ≥10.5 mm constituted 85% and 86%, respectively. When using the IVC diameter on expiration ≥18.5 mm as an indicator, the sensitivity and specificity for detecting PVH were evaluated at the levels of 100% and 92%, correspondingly.

Conclusion — The proposed ultrasound method of diagnosing congestion in pulmonary circulation via maximum and/or minimum diameter of the RSPV can be effectively employed in clinical practice in the same way as conventional technique of congestion diagnostics in systemic circulation via the diameter of IVC in patients with atrial fibrillation and chronic heart failure.

Keywords: hyperemia, pulmonary circulation, systemic circulation, diameter of the right superior pulmonary vein, diameter of the inferior vena cava, atrial fibrillation, chronic heart failure.


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Introduction

Heart failure is among the major causes of hospital admission with a high risk of readmission [1]. According to randomized studies, the risk of atrial fibrillation (AF) increases with a functional class (FC) of chronic heart failure (CHF) [2]. In large European registries, the prevalence of AF among patients, hospitalized for CHF ranged from 35-42% [3, 4]. Simultaneous presence of AF and CHF not only deteriorates the patient’s quality of life, but also increases the hospitalization rate and mortality [5].

Most patients with heart failure (76%), admitted for inpatient treatment, are ‘wet’ and ‘warm’—i.e., they represent the congestive type with a normal peripheral perfusion [6]. However, only 44% of patients complain of dyspnea at rest [7]. Therefore, it is important to perform instrumental diagnostics of fluid retention in patients with CHF as early as possible in order to prevent the decompensation, represented by progressive fluid accumulation in the body. When underestimated by clinicians, the severity of CHF may result in unsuitable treatment, including refusal from loop diuretics or prescribing wrong doses [8], and, consequently, the number of wet patients increases.

According to the recommendations for CHF diagnostics and treatment, congestion in systemic and pulmonary circulatory systems is diagnosed symptomatically; however, the symptoms exhibit either low specificity or low sensitivity, thereby allowing to diagnose venous congestion only at its later stages [9, 10]. Among...
instrumental diagnostic methods, chest radiography is capable of detecting the lung congestion and pleural fluid, but 20% of patients with congestion have a normal chest X-ray [11]. Compared with the chest X-ray, the number of B-lines on lung ultrasonography more effectively excludes the interstitial edema and pleural effusion. However, the disadvantage of this technique is represented by late diagnostics of congestion in the pulmonary circulation (i.e., at the stage of interstitial edema). Besides, the method does not allow differentiating among the causes of interstitial and alveolar pulmonary edema [11]. Determined via echocardiography, the ratio of the maximum filling velocity in the left ventricle (LV) during early diastole to the maximum velocity of movement of the mitral valve annulus in early diastole phase, E/e’ ≥ 15, correlates with an increased filling pressure of the LV, whereas E/e’ < 8 is indicative of the normal value of the latter. However, at values of E/e’ ranging 8-15, a wide range of early diastolic pressure values in the LV is detected, which requires additional diagnostics for this group of patients [12]. The sensitivity of E/e’>12 is 66%, while its specificity is 55% [11].

In the presence of right ventricular (RV) insufficiency, there is an increase in end-diastolic pressure in RV and pressure in the right atrium (RA), as well as a difficulty in blood flow towards the heart. As a result, the inferior vena cava (IVC) expands and its collapse during inspiration decreases. The expanded IVC without signs of collapse indicates a significant pressure increase in RA of over 15 mm Hg [13]. The sensitivity of the ‘IVC collapse less than 50%’ symptom as an indicator of RA pressure increase is 12%, and its specificity is 27%. The sensitivity of the ‘IVC diameter during inspiration below 12 mm’ indicator of RA pressure increase is 67%, and its specificity is 91% [11].

Cardiac catheterization with a direct pressure measurement in the atria and pulmonary artery with the estimate of pulmonary capillary wedge pressure (PCWP) is the gold standard of diagnosing the congestive phenomena in CHF. However, the invasiveness of this technique significantly limits its applicability in clinical practice. Hence, available and easily reproducible instrumental methods, allowing to diagnose the congestive phenomena in pulmonary and systemic circulatory systems even at early CHF stages, are in dire need. They are essential for the proper treatment, including diuretic therapy, in order to prevent CHF decompensation.

The goal of our study was to estimate the sensitivity and specificity of the newly proposed ultrasound method for diagnosing the pulmonary venous hypertension (PVH) by the maximum and minimum diameters of the right superior pulmonary vein (RSPV) in the same way as the conventional technique for congestive phenomena detection in systemic circulation by IVC diameter on expiration in patients with paroxysmal AF, and with or without CHF.

**Material and Methods**

**Patients**

We prospectively recruited 64 adult subjects who were examined for paroxysmal AF at the Department of Cardiology, Cardiology Research Institute of Tomsk, Russia, from March through May of 2019. The presence of cardiovascular diseases (coronary artery disease, essential hypertension), paroxysmal AF were the inclusion criteria. All patients with or without CHF were scheduled for radiofrequency pulmonary vein isolation (PVI). CHF was diagnosed sensu the latest recommendations on CHF diagnostics [9, 10]. Subjects without established CHF were asymptomatic patients with paroxysmal AF. The exclusion criteria encompassed the acute coronary syndrome, pulmonary embolism, congenital and acquired heart valve diseases, thyroid diseases, menopausal disorders, acute and chronic kidney diseases, acute and chronic pulmonary diseases, and cancer of all types. The total of 28 patients with paroxysmal AF were distributed among two groups, depending on the presence or absence of CHF in the anamnesis. Group 1 included 19 patients, 61.72±6.66 years of age, with CHF of FC I–III sensu the classification by New York Heart Association (NYHA). Group 2 comprised 9 patients 55.83±6.62 years old without CHF. The control group consisted of 44 virtually healthy individuals 35.02±2.44 years old who were previously examined at Uralskaya Clinic LLC (Ekaterinburg, Russia).

An informed consent to participate in the study was obtained from all participants. The study was approved by the local Ethics Committee at the Research Institute of Cardiology, Tomsk, Russia.

**Echocardiographic study**

Transesophageal echocardiography in the sinus rhythm was performed in all patients with the Philips HD-15 device (USA), according to the standard protocol. Additionally, the maximum and minimum diameters of RSPV were determined from the subcostal view. The maximum diameter of RSPV was measured in the ventricular systole phase; the minimum diameter was measured in the atrial systole phase [14, 15].

Besides, mean wedge pressure in the pulmonary artery (PAWP) was calculated by the following formula [16]: mean PAWP = 1.24 E/e’+1.9 (where E is the peak velocity blood flow from LV relaxation in early diastole, based the data by Doppler sonography of the transmitial flow; and e’ is the peak velocity blood flow of the early diastolic movement of the mitral valve lateral segment, based on the data of the pulse-wave tissue Doppler).

Also, mean right atrial pressure (RAP) was calculated by the following formula [17]: mean RAP = 1.7 E/e’+0.8, where E is the peak velocity blood flow from RV relaxation in early diastole, based the data by Doppler sonography of the diastolic flow through the tricuspid valve; and e’ is the peak velocity blood flow of the early diastolic movement of the tricuspid valve lateral segment, based on the data of the pulse-wave tissue Doppler.

**Invasive atrial pressure measurement**

Mean pressure in both atria was measured intraoperatively by the direct method in patients with paroxysmal AF prior to the radiofrequency PVI.

**Statistical analysis**

The use of chi-squared test showed that the available sample complied with the normal distribution. Statistical analysis of our research results was carried out via the Student’s t-test for a small sample. Statistically significant differences were considered at p <0.05. The data are presented in the form of M±m, where M is the mean of measured values, and m is the error of the mean. To determine the relationship between the indicators, the linear Pearson-Spearman correlation coefficient was calculated. Using ROC analysis, the reliability of the model of ultrasound diagnostics of PVH was verified by the maximum and minimum RSPV diameters. ROC curves were built using the IBM SPSS software. To
assess the quality of this model, we used the area under the ROC curve (AUC). The sensitivity and specificity of the ultrasound method for diagnosing the PVH were evaluated via the maximum and minimum diameters of RSVP [18].

Results
We examined 28 patients (12 men and 16 women) with paroxysmal AF, who were scheduled for the radiofrequency PVI. Commonly, AF progressed in patients with coronary artery disease (64%) and with essential hypertension (61%).

Table 1. Characteristics of study subjects

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Patients with AF and CHF (n=19)</th>
<th>Patients with AF and without CHF (n=9)</th>
<th>Control values (n=44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (male)</td>
<td>6 (31.6%)</td>
<td>6 (66.7%)</td>
<td>26 (38.6%)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>61.72±3.66</td>
<td>55.83±6.62</td>
<td>35.02±2.44</td>
</tr>
<tr>
<td>Coronary artery disease: effort of physical exertion</td>
<td>15 (78.9%)</td>
<td>3 (33.3%)</td>
<td></td>
</tr>
<tr>
<td>Arterial hypertension</td>
<td>14 (73.7%)</td>
<td>8 (88.9%)</td>
<td></td>
</tr>
<tr>
<td>Postmyocardial infarction cardiomegaly</td>
<td>0</td>
<td>1 (11.1%)</td>
<td></td>
</tr>
<tr>
<td>Acute stroke in the past</td>
<td>3 (15.8%)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>2 (10.5%)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>2 (10.5%)</td>
<td>1 (11.1%)</td>
<td></td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>19 (100%)</td>
<td>9 (100%)</td>
<td></td>
</tr>
<tr>
<td>Duration of AF, years</td>
<td>7.00±3.28</td>
<td>3.60±1.80</td>
<td></td>
</tr>
<tr>
<td>Duration of hypertension, years</td>
<td>20.75±4.88 *</td>
<td>9.43±3.98</td>
<td></td>
</tr>
</tbody>
</table>

CHF (FC)

FC I                              2 (10.5%) 0
FC II                             12 (63.2%) 0
FC III                            5 (26.3%) 0
Obesity                           8 (42.1%) 4 (44.4%)
Therapy: beta blockers            7 (36.8%) 2 (22.2%)
ACEI/ARB                          10 (52.6%) * 9 (47.4%) * 1 (11.1%) / 0
MCRA                             4 (21.0%) / 0
Indapamide and hydrochlorothiazide/ 5 (26.3%) * / 0/
Torsamide                          4 (21.0%) 0
Calcium antagonists               3 (15.8%) 0
Statis                            11 (57.9%) 3 (33.3%)
Antiarrhythmic therapy           13 (68.4%) 3 (33.3%)

AF, atrial fibrillation; CHF, chronic heart failure; FC, functional class; ACEI/ARB, angiotensin converting enzyme inhibitors/angiotensin receptor blockers; MCRA, mineralocorticoid receptor antagonists. * p<0.05 for patient groups compared with each other; ** p=0.05 for patient groups vs. control values.

Table 2. Echocardiographic and invasive parameters of hemodynamics

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control values (n=44)</th>
<th>Patients with AF and CHF (n=19)</th>
<th>P&lt;0.01</th>
<th>Patients with AF and without CHF (n=9)</th>
<th>P&lt;0.001</th>
<th>Patients without CHF (n=44)</th>
<th>P&lt;0.001</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAVI, ml</td>
<td>48.29±4.33</td>
<td>50.25±7.65</td>
<td>&lt;0.001</td>
<td>40.26±3.17</td>
<td>&lt;0.001</td>
<td>0.801</td>
<td></td>
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<tr>
<td>Simpson’s ejection fraction, %</td>
<td>62.89±1.47</td>
<td>58.63±6.79</td>
<td>0.750</td>
<td>65.5±2.66</td>
<td>0.808</td>
<td>0.176</td>
<td></td>
</tr>
<tr>
<td>Transmital EF/</td>
<td>1.61±0.07</td>
<td>0.77±0.05</td>
<td>&lt;0.001</td>
<td>0.75±0.08</td>
<td>&lt;0.001</td>
<td>0.672</td>
<td></td>
</tr>
<tr>
<td>Septal e’</td>
<td>13.25±0.63</td>
<td>7.03±0.74</td>
<td>&lt;0.001</td>
<td>6.88±0.79</td>
<td>&lt;0.001</td>
<td>0.847</td>
<td></td>
</tr>
<tr>
<td>LV lateral e’</td>
<td>17.85±0.94</td>
<td>10.03±0.87</td>
<td>&lt;0.001</td>
<td>10.62±1.94</td>
<td>&lt;0.001</td>
<td>0.486</td>
<td></td>
</tr>
<tr>
<td>Mean E/e’</td>
<td>6.83±0.29</td>
<td>8.92±1.73</td>
<td>&lt;0.001</td>
<td>8.62±1.31</td>
<td>&lt;0.001</td>
<td>0.650</td>
<td></td>
</tr>
<tr>
<td>Calculated PCWP, mm Hg</td>
<td>1.97±0.004</td>
<td>11.48±1.04</td>
<td>&lt;0.001</td>
<td>10.82±1.64</td>
<td>&lt;0.001</td>
<td>0.491</td>
<td></td>
</tr>
<tr>
<td>Maximum pulmonary vein diameter, mm</td>
<td>13.50±0.44</td>
<td>22.39±0.95</td>
<td>&lt;0.001</td>
<td>21.44±1.67</td>
<td>0.001</td>
<td>0.246</td>
<td></td>
</tr>
<tr>
<td>Minimum pulmonary vein diameter, mm</td>
<td>6.42±0.17</td>
<td>11.78±1.19</td>
<td>&lt;0.001</td>
<td>11.33±1.29</td>
<td>&lt;0.001</td>
<td>0.902</td>
<td></td>
</tr>
<tr>
<td>Invasively measured LA pressure, mm Hg</td>
<td>2-12 (7.9) [19]</td>
<td>14.68±1.40</td>
<td>&lt;0.001</td>
<td>15.00±2.63</td>
<td>0.850</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PASP, mm Hg</td>
<td>15.23±1.24</td>
<td>36.11±4.64</td>
<td>&lt;0.001</td>
<td>29.33±3.28</td>
<td>0.001</td>
<td>0.180</td>
<td></td>
</tr>
<tr>
<td>Transticuspidal E/A</td>
<td>1.58±0.07</td>
<td>0.82±0.04</td>
<td>&lt;0.001</td>
<td>0.91±0.12</td>
<td>&lt;0.001</td>
<td>0.004</td>
<td></td>
</tr>
<tr>
<td>RV lateral e’</td>
<td>15.00±0.82</td>
<td>8.73±0.83</td>
<td>&lt;0.001</td>
<td>8.30±1.12</td>
<td>&lt;0.001</td>
<td>0.466</td>
<td></td>
</tr>
<tr>
<td>IVC, cm</td>
<td>17.60±0.76</td>
<td>22.41±1.35</td>
<td>&lt;0.001</td>
<td>21.89±2.40</td>
<td>0.001</td>
<td>0.651</td>
<td></td>
</tr>
<tr>
<td>Calculated LAP, mm Hg</td>
<td>3.68±0.24</td>
<td>10.36±0.81</td>
<td>&lt;0.001</td>
<td>11.57±2.55</td>
<td>0.001</td>
<td>0.251</td>
<td></td>
</tr>
<tr>
<td>Invasively measured RA pressure, mm Hg</td>
<td>1-7 (3.9) [19]</td>
<td>6.75±0.63</td>
<td>0.001</td>
<td>6.13±1.13</td>
<td>0.288</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; CHF, chronic heart failure; LAVI, left atrial volume index; E/A, the ratio of peak velocity blood flow from left ventricular relaxation in early diastole (the E wave) to peak velocity flow in late diastole caused by atrial contraction (the A wave); LV, left ventricle; Mean E/e’, the ratio of early diastolic blood flow velocity to the velocity of the lateral and medial parts of the mitral valve annulus; PCWP, pulmonary capillary wedge pressure; LA, left atrium; PASP, pulmonary artery systolic pressure; RV, right ventricle; IVC, inferior vena cava; RA, right atrium. P denotes statistical significance of differences between each group of patients and the control group. P’ denotes statistical significance of differences between groups of patients.
In Group 2, the duration of AF and arterial hypertension was 3.60±1.80 years and 9.43±3.98 years, correspondingly. Patients in this group were receiving basic therapy without diuretic medications.

Patients of the control group were significantly younger than in two experimental groups, because we were not able to find virtually healthy people at ages similar to the subjects in Groups 1 and 2.

Patients with AF, and with or without CHF, compared with the control subjects, based on echocardiography data, had left atrial (LA) dilatation confirmed by the LA volume index (LAVI) values of 67.70±1.91 mL, 68.00±3.37 mL and 49.29±4.33 mL, respectively (Table 2); the maximum RSPV diameter of 22.39±0.95 mm, 21.44±1.67 mm and 13.50±0.44 mm, correspondingly (Figures 1A, 2A, 3A); and the minimum RSPV diameter of 11.78±1.19 mm, 11.33±1.29 mm and 6.42±0.17 mm, respectively (Figures 1B, 2B, 3B). As for the mean E/e’, it was very similar in both experimental groups: 8.92±0.73 vs. 8.62±1.31. This finding implied that values of the mean E/e’ were, so to speak, in the grey zone and required additional investigation in order to clarify the presence or absence of hypertension in the LA [10]. For the patients with AF, and with or without CHF, the calculated PCWP was significantly higher than in the control group, but still under 12 – i.e., it was within the range of reference values [19].

Hence, sensu lato, the existing ultrasound methods for detecting the congestion in the pulmonary circulation, patients with paroxysmal AF, regardless of CHF diagnosed by cardiologists, had no PVH. However, the invasively measured pressure in LA was elevated both in the group of patients with AF and CHF (14.68±1.40 mm Hg) and in the group with AF but without CHF (15.00±2.63 mm Hg). This finding implied the presence of the PVH and CHF in patients of both groups.

We established the positive correlation between the minimum diameter of RSPV and invasive mean pressure in the LA (r=0.65, p<0.05). We did not find statistically significant correlation between the maximum RSPV diameter and invasive mean pressure in the LA (r=0.06). Likewise, there was not significant correlation between the mean E/e’ and invasive mean pressure in the LA (r=−0.09) (Table 3).

We performed the ROC analysis to verify the reliability of the ultrasound diagnostic model for PVH based on the maximum and minimum RSPV diameters. For the maximum pulmonary vein (PV) diameter, AUC=0.599 (p<0.05), which indicated that this model had an average quality level.

The equilibrium point was the minimum diameter of RSPV of 10.5 mm, which corresponded to the norm (sensitivity of 85%, specificity of 86%).

Accordingly, the method for determining PVH via measuring the maximum and/or minimum diameters of the PV could be effectively used as noninvasive diagnostics of the venous congestion in the pulmonary circulation.

For the patients with AF and with or without CHF, the calculated pressure in the LA was 10.36±0.81 and 11.57±2.55 (mmHg), respectively; the invasive measurement method yielded the values of 6.75±0.63 and 6.13±1.13 (mmHg), respectively. This finding implied that the pressure calculated via echocardiography was overestimated.

There was a positive correlation between invasively measured mean pressure in the RA and IVC diameter on expiration (r=0.49, p<0.05). For the maximum IVC diameter, the ROC curve plot was obtained with AUC=0.832 (p<0.05), which indicated that this model had a very good quality level. The equilibrium point was the value of the IVC diameter of 18.5 mm, which corresponded to the norm (sensitivity of 100%, specificity of 92%).

Discussion

It is known, that in arrhythmogenesis of the AF, PVs play an essential role as a trigger or driver of AF [20, 21]. Prior studies using cardiac imaging modalities demonstrated that AF patients had significantly enlarged PVs, compared with the control [22]. This finding suggests that structural alteration of PVs is related to the AF development. Our study demonstrated that patients with paroxysmal AF, and with or without CHF, compared with the control group, according to echocardiography, exhibited the dilatation of both maximum and minimum RSPV diameters. There was a positive correlation between the minimum RSPV diameter and invasive mean pressure in the LA (R=0.65, P<0.05). Our study revealed that asymptomatic patients with AF had an augmented invasively measured pressure in LA and structural changes in LA and PVs that did not differ from those of the patients with simultaneous presence of AF and CHF. This finding suggests that in CHF diagnostics, it is preferable to rely on changes in the structure and function of the heart, detected by the echocardiography, rather than on symptoms and manifestations alone.

In this study, the sensitivity and specificity of ultrasound methods of congestion diagnostics in the pulmonary and systemic circulatory systems were determined in comparison with the gold standard – i.e., the invasive pressure measurement in the LA and RA of the heart.
Figure 1. Maximum (A) and minimum (B) diameter of RSPV in patients with AF and CHF. The maximum diameter of RSPV was 26.2 mm; the minimum diameter of RSPV was 17.0 mm.

Figure 2. Maximum (A) and minimum (B) diameter of RSPV in patients with AF and without CHF. The maximum diameter of RSPV was 21.8 mm; the minimum diameter of RSPV was 10.7 mm.

Figure 3. Maximum (A) and minimum (B) diameter of RSPV: control values. The maximum diameter of RSPV was 12.1 mm; the minimum diameter of RSPV was 5.6 mm.
The method of noninvasive diagnostics of PVH is based on measuring the maximum diameter of any of the visible pulmonary veins pushing blood into the LA during diastole, and the minimum diameter during atrial systole. If the maximum diameter of any visible pulmonary vein is greater than 18 mm, and its minimum diameter is greater than 9 mm, then the venous congestion in the pulmonary circulation is diagnosed [14]. The sensitivity of the method for the maximum RSPV diameter greater than 21.7 mm, as an ultrasound criterion for PVH, was 75%, and its specificity was 86%. The sensitivity of the method for the minimum diameter of the RSPV greater than 10.5, as an ultrasound criterion for PVH, was 85%, and its specificity constituted 86%.

In this study, in 24 of 28 patients with AF, the pressure in LA (14.79±1.18 mm Hg) was elevated, whereas mean E/e’ values in patients with AF, and with or without CHF, were 8.92±0.73 and 8.62±1.31, respectively, which implied the gray zone of values, requiring additional examination methods to determine the pressure in the LA [10]. We did not find significant correlation between the mean E/e’ and invasive mean pressure in the LA (r=-0.29). A systematic review of nine studies has reported only modest correlations of E/e’ with invasive filling pressures in CHF patients with preserved ejection fraction, because some of these studies did not reveal correlations similar to ours [23].

It is well-known that the presence of congestion in the pulmonary circulation and the necessity to take diuretics in addition to primary therapy prevents the development of CHF decompensation [10]. In this study, in addition to the primary therapy, 9 patients were prescribed diuretic medicines: MCRA and indapamide. In 4 patients, torasemide at a dose of 2.5-5 mg was prescribed. At the same time, PVH was detected in 24 patients via using invasive and newly proposed ultrasound technique of measuring the maximum and/or minimum diameter of the PV. Fluid retention in the body with CHF may lead to the development of atrial myocardial edema [24]. However, an appointment of adequate diuretic therapy in patients with simultaneously present paroxysmal AF and CHF reduces the frequency of arrhythmia recurrence [25].

Previously known ultrasound methods for detecting congestion in the pulmonary circulation did not show signs of increased pressure in the LA. The calculated PAWP in patients with AF, and with or without CHF, was not informative as well, considering that identified values were within the reference range ~ 11.48±1.04 and 10.82±1.64 (mm Hg), respectively. Hence, the diagnostics of CHF and congestion phenomena in pulmonary circulation and, consequently, inappropriate treatment was not unusual, including the lack of prescribing loop diuretics or their incorrect dosages.

It should be noted that the method of ultrasound diagnostics of PVH by examining the maximum and minimum PV diameters in patients with CHF has also allowed evaluating the effectiveness of the diuretic treatment. In patients with venous congestion in the pulmonary circulation, the maximum and minimum diameters of visualized PVs decreased in response to an adequate diuretic therapy [26].

Determining the congestion in systemic circulation does not usually cause difficulties for clinicians, because contrary to congestion in pulmonary circulation, the clinician can objectively assess the presence of edema and increase in the liver size by percussion and palpation. However, it is imperative to differentiate between the edema of the lower limbs that occurs in varicose veins, lymphostasis, and an increase in the liver size caused by liver diseases, including steatohepatitis, which has become widespread in recent years.

Our study determined the sensitivity and specificity of the proposed ultrasound method for congestion diagnostics in systemic circulation via the diameter of IVC on expiration and via the gold standard of invasive measurement of pressure in RA. The specificity of the IVC diameter exceeding 18.5 mm on expiration was 100%, and the specificity was 92%. We established positive correlations between invasively measured mean pressure in the RA and systolic pressure in the pulmonary artery (r=0.54, p<0.05), and maximum diameter of IVC as well (r=0.49, p<0.05).

Limitations

Our sample size was rather small. The studied patients did not have wide range of atrial pressure values. Consequently, actual the relationship between the maximum and/or minimum PV diameter and blood pressure in the LA could have been underestimated. Hence, further research is needed on a large sample of patients.

Conclusion

The proposed ultrasound method for diagnosing congestion in the pulmonary circulation via maximum and/or minimum RSPV diameter in the same way as the conventional technique of detecting the congestion in systemic circulation via IVC diameter in patients with AF and CHF can be effectively employed in clinical practice, providing an adequate diuretic therapy and significantly reducing the number of CHF decompensation cases.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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