Clinical Significance of Inferior Vena Cava Index in Monitoring Patients in Acute Exacerbation of Chronic Heart Failure

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Abstract

Background: Patients in acute exacerbation of chronic heart failure represent an important health problem and economic burden that requires proper intervention and treatment.

Aim of Work: To investigate the validity of Inferior Vena Cava (IVC) dynamics as a noninvasive diagnostic monitoring tool in patients with acute exacerbation of heart failure.

Methods: Thirty patients with decompensated heart failure (NYHA class III-IV) 16 males and 14 females mean age 50±13 years were included, admitted to ICU, and had full echocardiographic study including IVC dynamics: IVC diameter at end of expiration, at end inspiration, and collapse index (IVC-CI%) on admission, on days 5 and 10 of medical treatment. Also, ten volunteers were included as control group.

Results: After therapy, the IVC-CI% significantly increased on day 10 with mean 45.2±8.6% compared to IVC-CI% on day 5 with mean 27.7±8.6% compared to IVC-CI% on admission with mean 11.7±4.79% (p-value <0.001).

FS% increased significantly after 5 days compared to day 1 with mean 24.13±3.6%, however, there was no significant changes between day 10 and 5 values (p values <0.001 and >0.05 respectively).

EF% increased statistically after 5 and 10 days of therapy compared to day 1 values: 25.7±5.3%, 34.2±5.6%, 20.6±3.6% with p-values <0.001 and <0.05 respectively.

Conclusion: IVC dynamics appears to be simple reliable noninvasive bedside tool in management and monitoring therapy of patients with acute decompensated heart failure.

Key Words: Acute exacerbation of heart failure – Noninvasive diagnostic IVC index – Echocardiography.

Introduction

HEART Failure (HF) is a common and growing problem. The prognosis remains poor despite the identification of effective treatments, at least for patients with Left Ventricular Systolic Dysfunction (LVSD) [1]. The lack of widely accepted objective measures of cardiac dysfunction other than LVEF has hampered and continues to hamper clinical research in patients with HF. The non-invasive methods are kept as an alternative and have superiority as they are easily applied and do not raise the complications conditioned by the invasive measures [2].

Echocardiography can be a rapid, noninvasive, objective tool in the assessment of ventricular function and preload when compared with conventional invasive hemodynamic monitoring techniques [3]. Echocardiographic assessment of Inferior Vena Cava (IVC) diameter is simple and might be an objective, quantifiable measure, however, its relationship with other clinical variables and its potential prognostic role have received little attention. Therefore, it might represent an index of HF severity independent of Left Ventricular Ejection Fraction (LVEF). The relation between IVC diameter and other clinical variables and its prognostic significance in patients with HF has not been explored [4].

Aim of work:

The aim of the work is to determine the clinical significance and validity of measuring the inferior vena cava dimensions and dynamics as a bedside noninvasive hemodynamics monitoring tool in critically ill patients with congestive heart failure by echocardiography.

Patients and Methods

The study included 30 patients, 16 males (54%) and 14 females (46%), their ages range from 18 to 70 years with the mean age 53.2±13.09 years. All
were admitted to ICU suffering from acute decompensation of the already preexisted chronic Congestive Heart Failure (CHF).

**Inclusion criteria:**
- Male or female aged from 18 to 70 years.
- All are suffering from acute exacerbation of chronic congestive heart failure from:
  - Ischemic heart disease.
  - Dilated cardiomyopathy.
- All are in NYHA class III-IV.
- All in sinus rhythm.

**Exclusion criteria:**
- Patients who had the following were excluded:
  - Chronic Obstructive Pulmonary Disease (COPD).
  - On mechanical ventilation.
  - Atrial fibrillation.
  - Advanced hepatic cirrhosis and tense ascites.
  - Evidence of IVC obstruction.
  - Evidence or suspicion of pulmonary embolism.
  - Left ventricular outflow obstruction e.g. aortic stenosis.
  - End stage renal disease and those on dialysis.
  - Clinically significant abnormalities of hemopoietic and endocrine function.
  - Severe anemia with Hb <7g/dl.

The duration of the study was 10 days for each patient. On admission, all patients were subjected to full history taking, accurate clinical, electrocardiographic, radiographic, laboratory and echocardiographic examination. Under full clinical observation and appropriate medical treatment in ICU, all patients were followed-up and all tests were repeated on day 5 and on day 10 of treatment. Then, all results were recorded and tabulated and undergone statistical analysis.

1. **History and clinical examination:** On admission, meticulous history taking and clinical examination fulfilling the criteria for acute decompensation of chronic CHF based on evidence of new worsening or deterioration of signs and symptoms of HF [8].

2. **Right Atrial Pressure (RAP):** (RAP) can be measured noninvasively by the following equation: RAP= JVP + 5 in cm [6].

3. **Electrocardiography (ECG):** It was recorded on admission and on days 5 and 10 of treatment for all patients to detect signs of ischemia, chamber enlargement and exclude atrial fibrillation.

4. **Chest X-ray with Postero-Anterior Projection (PA-P).**

5. **Laboratory examination:** Urinary albumin was measured for all patients. A first morning urine, urinary albumin level was measured by radio immune assay (Eliza test). Then, collection of 24 hour urine was done for measurement of urine volume and calculation of urinary albumin excretion/24 hour in milligrams (mg) was done [7]. Measurements of urinary albumin excretion was done for all patients on admission, on day 5 and day 10 of medical treatment. Also, blood urea and serum creatinine were determined for all patients on admission and on day 5 and 10 of the study (patients with serum creatinine levels more than 2.5mg/dl were excluded).

6. **Echocardiographic examination:** Imaging was performed in the left lateral of supine decubitus positions using the ACUSON 128 system with a 2.5/3.5MHz dual frequency cardiac transducer. The study was recorded on video system and the measurements were obtained by two observers.

A. **Left ventricular parameters:** From M-mode, we obtained measurements of Left Ventricular End Systolic and End Diastolic dimensions (LVEDD and LVEDV respectively) and Left Atrial diameter (LA).

Estimation of Ejection Fraction (EF%) was calculated using the Acuson Software Cardiac Analysis Package. This method applies Simpson's formula to calculated ventricular volumes (end systolic and end diastolic volumes) (LVEDV and LVEDV respectively) using disc summation before obtaining a mean value for EF% from each scanning planes.

\[
\text{LVEDV} - \text{LVEDV} \times 100\%.
\]

1- Ejection Fraction (EF%):

\[
\frac{\text{LVEDV} - \text{LVEDV}}{\text{LVEDV}} \times 100\%.
\]

2- Fractional Shortening (FS%):

\[
\frac{\text{LVEDD} - \text{LVEDD}}{\text{LVEDD}} \times 100\%.
\]

3- Stroke Volume (SV):

\[
\text{SV} = \text{LVEDV} - \text{LVEDV} \text{ ml}.
\]

4- Cardiac Output (CO):

\[
\text{CO} = \text{SV} \times \text{HR} \text{ Liter/min}.
\]

5- Cardiac Index (CI):

\[
\text{CI} = \frac{\text{CO}}{\text{BSA}} \text{ Liter/min/m}^2 \text{ BSA}.
\]

B. **Parameters of IVC dynamics:** IVC study was performed while the patient lies supine by anterior subcostal approach. By 2D image in the longitudinal plane, IVC appears as a tubular structure with echo=free lumen. By upward movement of the transducer, the point of entrance into RA can be clearly visualized. By rotating it 90°, IVC appears in the transverse plane as a circular or oval
structure to the right of aorta [8]. Measurements of IVC dimensions was obtained on 2D or M-mode images at 2 centimeter below the entrance into RA.

IVC dimension was measured at end-expiration to obtain the maximum IVC diameter (D-max). Also, IVC dimension was measured at end-inspiration to obtain the minimum IVC diameter (D-min).

The dispensability index of IVC or IVC collapse index (IVC-CI%) was derived as the ratio of difference between maximal and minimal diameter to the maximal diameter, and expressed as percentage.

\[
\text{IVC-CI} = \frac{\text{D-max} - \text{D-min}}{\text{D-max}} \times 100\% \quad [9].
\]

7- All results were recorded, tabulated and undergone statistical analysis. Comparisons were made using the \( t \)-test, Mann-Whitney U test, Fisher’s exact test, and chi-square test as appropriate.

8- The control group: Ten normal persons were included in the study. They were subjected to the echocardiographic examination to obtain normal parameters for LV function and IVC dynamics. These parameters were used for comparison with those obtained from patients’ group. Also, serum creatinine levels were measured for all persons in this group.

9- Treatment: All patients received appropriate medical therapy for acute deterioration of CHF in the form of, intravenous IV diuretics using loop diuretics e.g., furosemide 80-240mg/day, IV vasodilator (nitroglycerin 5-20mg/min), IV inotropic (dobutamine 5-10mg/kg/min), oral ACE inhibitor (captopril 6/5-25mg/day) or an angiotensin receptor blocker (valsartan 40mg/day) plus Low Molecular Weight Heparin (LMWH) (enoxaparin 40mg/day) for prophylaxis against Deep Vein Thrombosis (DVT) and antibiotics when needed.

Results

The results of this study are demonstrated under the following headings:

1- Analysis of general clinical data.

2- Analysis of laboratory data.

3- Analysis of echocardiographic data, including:

A- Analysis of cardiac echocardiographic parameters.

B- Analysis of parameters of IVC dynamics.

1- Analysis of general clinical data:

The study included 30 patients who were admitted to ICU with acute exacerbation or decompensation of chronic CHF in the period between 1st of December 2005 to 1st of November 2006. The included patients were 16 males (54%) and 14 females (46%) with their ages ranged from 18 to 70 years with the mean age 53.2±13.09 years.

The (Table 1) reveals that all patients (100%) in the study group had dyspnea grade III to IV and basal lung crepitation.

On admission, all patients were classified according to NYHA into: 24 patients (80%) were in NYHA class IV and 6 patients (20%) were in class III. Significant improvement occurred on day 5 of treatment with 17 patients (57%) were in class II and 13 patients (43%) were in class I (\( p \)-value <0.001) and on day 10 of treatment with only 5 patients (17%) were in class II and 25 patients (83%) were in class I (\( p \)-value <0.001).

Table (3) reveals no statistically significant differences between blood pressure values (systolic or diastolic) on admission, on day 5 and day 10 of treatment.
treatment ($p$-value = NS). Also, it reveals statistically significant differences between heart rate on admission, on day 5 and day 10 of treatment ($p$-value: <0.01).

2- Analysis of laboratory data:

On admission, there was evident increase in Urinary Albumin Excretion (UAE) in all patients and it ranged between 118 to 800mg/24h with mean of 416 ± 188mg/24h.

After 5 days of treatment, UAE per 24 hour decreased significantly and ranged from 72 and 640mg/24h with mean of 279 ± 121mg/24h ($p$-value <0.001 as compared to the UAE values on admission). Also, on day 10 it furtherly decreased significantly to range from 33 and 410mg/24h with mean of 167 ± 87mg/24h ($p$-value <0.001 as compared to the UAE values on day 5). (Table 4).

Table (4): Albuminuria and S.creatinine on admission, on day 5 and day 10 after medical treatment.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On admission Mean±SD</th>
<th>On day 5 Mean±SD</th>
<th>On day 10 Mean±SD</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>UAE (mg/24h)</td>
<td>416±188</td>
<td>279±121</td>
<td>167±87</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>S.creatinine</td>
<td>1.60±0.4</td>
<td>1.56±0.3</td>
<td>1.47±0.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

Table (4) reports that the mean values of urinary albumin excretion on admission, day 5 and 10 of treatment were significantly deceased in response to successful treatment with $p$-value <0.001. However, there was no significant change in serum creatinine on admission and after day 5 and 10 days of treatment, but there was significant increase in serum creatinine in patient group on admission compared to the control group (0.8-2.1mg/dl with mean 1.6±0.4mg/dl, 0.7-1.2mg/dl with mean 1.03 ± 0.3mg/dl with $p$-value: <0.01).

3- Analysis of echocardiographic data:

A- Analysis of cardiac echocardiographic parameter:

LA dimension was significantly increased in patients group. On admission (day 1), it ranged between 47 to 57mm with mean of 51.0±3.29mm versus control group (range 29 to 40mm with mean of 32.0±4.94mm, $p$<0.05). LA dimension in patients on admission, on day 5, and on day 10 of treatment reports no statistically significant change detected after day 5 and day 10 of treatment [51.0±3.29mm, 49.59±2.8mm and 49.27±2.5mm respectively with $p$-value=>0.05 (NS)].

There were significant correlations between the change in Urinary Albumin Excretion (UAE) and the changes that occurred in EF%, CO and CI on day 5 ($r$=0.53, 0.48 and 0.62 respectively) ($p$ <0.05) and on day 10 of therapy ($r$=0.51, 0.54 and 0.63 respectively) ($p$<0.05).

Table (5): Correlations between Urinary Albumin Excretion (UAE) and EF%, CO and CI after 5 and 10 days of treatment.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>EF % ($r$=value)</th>
<th>CO ($r$=value)</th>
<th>CI ($r$=value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>On day 5</td>
<td>0.53</td>
<td>0.48</td>
<td>0.62</td>
</tr>
<tr>
<td>On day 10</td>
<td>0.51</td>
<td>0.54</td>
<td>0.63</td>
</tr>
</tbody>
</table>
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The (Table 5) shows a statistically significant correlations between the UAE and the change in parameters of LV function on day 5 and day 10 of treatment. The strongest correlations was between the change in UAE and the change in CI \( (p<0.05), r=0.62 \) and 0.63 for 5th and 10th days respectively.

**B- Analysis of parameters of IVC dynamics:**

On admission, the patients D-max (IVC diameter at end expiration) was ranged between 17-28 mm with mean 22.10±3.40mm and D-min (IVC diameter at end inspiration) was ranged between 14-25mm with mean 19.5±3.03mm. In the control group, the D-max was between 11-17mm with mean 13.10±2.33mm and D-min was 3-5mm with mean 3.80±0.79mm Fig. (3).

Fig. (3) demonstrates a statically significant increase in IVC dimensions (D-max and D-min) in patients group on admission as compared to the control group \( (p<0.001) \).

Also, there was significant decrease in IVC collapse index (IVC-CI%) in admission that ranged from 5-24% with mean 11.70±4.79% while in the control group it was between 58-79% with mean of 61.10±9.45% \( (p\text{-value}<0.001) \) (Table 6).

**Table (6): Parameters of IVC dynamics in patients on admission and the control group.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On admission</th>
<th>Control group</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>D-Max (mm)</td>
<td>22.1±3.5</td>
<td>13.1±2.3</td>
<td>( p&lt;0.001 )</td>
</tr>
<tr>
<td>D-Min (mm)</td>
<td>19.5±3.0</td>
<td>3.8±0.8</td>
<td>( p&lt;0.001 )</td>
</tr>
<tr>
<td>IVC-CI%</td>
<td>11.7±4.8</td>
<td>61.1±9.5</td>
<td>( p&lt;0.001 )</td>
</tr>
</tbody>
</table>

Table (6) reveals statistically significant increase in IVC dimensions (D-max and D-min) and decrease in IVC collapse index (IVC-CI%) on admission versus the control group (\( p<0.001 \)).

On the 5th day of treatment, there was significant decrease in IVC dimensions, D-max was between 14-26mm and mean 18.83±3.13mm while the D-min was between 8-18mm and mean 13.43±2.44mm and \( p\)-value <0.001 as compared to the values on admission. Conversely, IVC-CI% had been significantly increased between 11 to 38% with mean 27.70±8.69% compared to its values on admission \( (p\text{-value}<0.001) \) [(Table 7) and Fig. (4)].

After 10 days of therapy, there was another significant decrease in IVC dimensions, the D-max was ranged between 13-22mm with mean 16.70±1.91mm and D-min was between 7-13mm with mean 9.90±1.83mm \( (p<0.01) \) compared to the values encountered on day 5 of treatment. Also, IVC-CI% on day 10 was significantly increased to be between 24 to 56% with mean 47.17±8.64% compared to its on days 5 values \( (p<0.001) \) [(Table 8) and Fig. (4)].

**Table (7): IVC parameters for patients on admission and on day 5.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On admission</th>
<th>On day 5</th>
<th>( p)-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>D-Max (mm)</td>
<td>22.1±3.5</td>
<td>18.8±3.1</td>
<td>( p&lt;0.001 )</td>
</tr>
<tr>
<td>D-Min (mm)</td>
<td>19.5±3.0</td>
<td>13.4±2.5</td>
<td>( p&lt;0.001 )</td>
</tr>
<tr>
<td>IVC-CI%</td>
<td>11.7±4.8</td>
<td>27.7±8.7</td>
<td>( p&lt;0.001 )</td>
</tr>
</tbody>
</table>

Table (7) reveals statistically significant decrease in D-max and D-min on day 5 of medical treatment while IVC-CI% was significantly increased \( (p<0.001) \).
Table (8): IVC parameters for patients on day 5 and day 10.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>On day 5</th>
<th>On day 10</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>D-Max (mm)</td>
<td>18.8±3.1</td>
<td>16.7±1.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>D-Min (mm)</td>
<td>13.4±2.5</td>
<td>9.9±1.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IVC-CI%</td>
<td>27.7±8.7</td>
<td>47.2±8.6</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Table (8) demonstrates statistically significant decrease in D-min and significant increase in IVC-CI% while no significant decrease in D-max on day 10 of medical treatment compared to their values on day 5 of treatment.

Fig. (5): Linear change in D-max (*) and D-min (^).

Fig. (5) demonstrates progressive decrease in D-max and D-min through the study (from day 1 through day 5 and day 10).

Table (9): Correlations between IVC parameters and the change in EF%, CO, CI and UAE on 5th and 10th days.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Day 5</th>
<th>Day 10</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>D-max</td>
<td>D-min</td>
<td>IVC-CI%</td>
</tr>
<tr>
<td>EF%</td>
<td>0.46</td>
<td>0.57</td>
<td>0.51</td>
</tr>
<tr>
<td>CO</td>
<td>0.53</td>
<td>0.59</td>
<td>0.56</td>
</tr>
<tr>
<td>CI</td>
<td>0.61</td>
<td>0.78</td>
<td>0.68</td>
</tr>
<tr>
<td>UAE/24h</td>
<td>0.58</td>
<td>0.60</td>
<td>0.67</td>
</tr>
</tbody>
</table>

*D-min : IVC diameter at end inspiration.
D-max : IVC diameter at end expiration.
IVC-CI% : IVC collapse index.

Table (9) reveals that the strongest correlations (r) were found between the change in IVC dynamics and the change in CI and UAE. (p-value in all <0.01).

Discussion

The clinical diagnosis of HF is fundamentally based on demonstrating objective evidence of cardiac dysfunction in the presence of symptoms, such as breathlessness, and signs, such as peripheral edema. Echocardiographic assessment focusing solely on LV function might be misleading. Advanced heart failure is associated with frequent hospitalizations, poor quality of living and increased mortality. Despite optimal medical management, readmission rates remain high and account for approximately two thirds of all costs related to HF management. If congestion is the hallmark of HF, then distension of the great veins might be the best marker on imaging. The IVC diameter is usually easy to measure in patients with HF and has low inter-observer variation. The IVC diameter is a summary measure of cardiac function as well as a marker of venous congestion, thus, increasing IVC diameter is associated with a worse prognosis [4].

Evaluation of patients with HF is critical for the appropriate selection and monitoring of therapy. This evaluation can be complex and relies on integration of the bedside information available from meticulous clinical examination, noninvasive and other invasive diagnostic techniques [10].

Patients with decompensated chronic congestive heart failure present with mild proteinuria which is reversible after treatment. In our study, albuminuria was evident in all patients with mean of 416±188mg/24hr on admission. After 5th and 10th day of treatment albuminuria regressed to mean 279±121mg/24hr and 167±87mg/24hr and p-value: <0.001 respectively.

Our results coincide with the results obtained by Eiskjaer et al., [11] who found increase in urinary protein excretion in patients with CHF that reverses promptly upon successful therapy. The urinary excretion of albumin was correlated to NYHA class (r=0.68) and plasma rennin (r=0.89) (p<0.05). Albuminuria in CHF carries the risk of poorer outcome, longer hospital stay and higher readmission rates [12] and has been found to be a strong predictor of cardiovascular events in CHF [13]. Explanation of albuminuria may be due to increased intra-renal angiotensin II generation, increased plasma ANPs levels that increase renal venous pressure and impaired function of glomerular electrostatic barrier. All these cause an increased renal endothelial permeability and protein infiltration in urine [11].

Echocardiography plays a central role in the diagnosis and assessment of HF. It is now established as an accurate, reproducible and noninvasive technique that is well suited for the evaluation of LV function and successfully used for diagnosis,
screening and monitoring therapy in patients with CHF [14]. In our study, all patients were examined by echocardiography for evaluation of LV function before therapy, on day 5 and 10 of treatment respectively.

Also, significant deterioration in parameters of LV function has been found in patients on admission (mean EF% 19.60 ± 3.64% and mean CI 1.59 ± 0.25 L/min/m²) as compared to (mean EF% 71.20 ± 5.55% and mean CI 2.64 ± 0.33L/min/m²) in the control group (p < 0.01).

These results confirm and coincides with that obtained by several studies as Capomolla S. et al., [15] and Traversi E. [16], who confirmed and strongly recommended the reliability of echocardiographic measurements (cardiac dimensions, EF% and CI) in diagnosis, screening and hemodynamic profile evaluation of patients with CHF.

In association with the decrease in cardiac dimensions, there were a significant improvement and increase in EF% and CI on day 5 and day 10 of treatment. The mean values of EF% were 25.70 ± 5.33% and 34.17 ± 5.65% on day 5 and 10 respectively (p-value < 0.05) and the mean values for CI were 1.84 ± 0.38L/min/m² and 2.03 ± 0.34L/min/m² on day 5 and 10 respectively (p-value < 0.001). These values denote a significant improvement in cardiac function in response to successful medical treatment.

All these results coincide with that obtained by Nohria et al., [17] and Cheesman et al., [14] who demonstrated that accuracy and reliability of the echocardiographically measured and derived parameters of LV function in hemodynamic monitoring for management of patients with CHF.

As the clinical conditions were improved, significant reduction in urinary albumin excretion were clearly observed. The of decrease in urinary albumin was significantly correlated to the improvement in cardiac function as detected by echocardiography.

**IVC dynamics in LV dysfunction and monitoring of LV function:**

Briefly, in spontaneously breathing subjects, intrathoracic pressure decreases during inspiration, thereby increasing venous return and inducing collapse of the IVC; inversely, during expiration, venous return decreases, so causing an increase in the diameter of the IVC. High right atrial pressures dilate the IVC and worsen this normal IVC collapsibility. According to these observations, congestion would be indicated by relatively small IVC-CI values. Therefore, large, non-collapsible IVCs reflect high right atrial pressures [18].

We found statistically significant different values between the two groups. There are significant increase in the maximum IVC diameter at end-expiration (D-max) in patients on admission with mean 22.10 ± 3.48mm versus mean 13.10 ± 2.33mm in the control group and significant increase in minimum IVC diameter at end-inspiration (D-min) in patients with mean 19.40 ± 3.30 versus mean 3.80 ± 0.79 in control group (p-value < 0.001). Also, the results revealed that, all patients in the control group had IVC-CI > 50% while all patients on admission had IVC-CI < 50% (the maximal value was 24%). These results demonstrated the change in IVC dynamics in patients with acute exacerbation of CHF in the form of increase in IVC dimensions and decrease in the of inspiratory collapse.

De Vecchis et al., [18] confirmed in a recent study that a IVCCI ≤ 15% was highly sensitive and specific for the diagnosis of acute decompensated heart failure. Furthermore, all of the patients presenting with IVCCI ≤ 15% were found to have a clinical picture of combined right and left heart failure, this finding being presumably associated with both high CPWP and right atrial pressure.

Nath et al., [19] stated that the dilated IVC in CHF is a marker of poor survival independent of a history of HF, ventricular function and pulmonary artery pressure. He reported IVC diameter in 3,729 patients, almost exclusively men, having echocardiograms at 1 of 3 U.S. Veterans hospitals.

Patients with a dilated IVC that did not collapse with inspiration were older, were more likely to have HF (38%), and had a 33% mortality at 1 year.

The findings by Pellicori et al., [4] also confirm and support the notion that IVC diameter might offer diagnostic advantages similar to NT-proBNP as a nonspecific marker of global cardiac dysfunction but might be less influenced by non-cardiac factors such as renal dysfunction or AF. Where echocardiography is not available, for instance in primary care, a blood test as the first diagnostic step for suspected HF is convenient and efficient. When echocardiography is available, IVC diameter might provide similar information. They found that IVC diameter was a strong predictor of prognosis, providing information similar to NT-proBNP (widely considered to be1 of the most robust prognostic markers in patients with HF). The IVC diameter is easy to measure and provides similar prognostic information as plasma concentrations of NT-proBNP in outpatients with chronic HF.
In our study, successful treatment is evidenced by significant clinical improvement, significant improvement in NYHA class (lung congestion) and marked improvements of hemodynamic parameters of LV function.

These changes were associated with statistically significant decrease in IVC-D-min on day 5 with mean of 13.43 ± 2.49mm and day 10 with mean of 9.90 ± 1.83mm ($p < 0.001$). Another significant decrease in IVC-D-max on day 5 in mean of 18.83 ± 3.13mm ($p < 0.001$) and on day 10 with mean 16.7 ± 1.9mm ($p < 0.001$). Also, there was significant improvement in IVC-CI% on day 5 (mean 27.70 ± 8.69%) and day 10 (mean 43.17 ± 8.64%) of therapy ($p < 0.001$).

Strong and significant correlation were found between echo-determined parameters of LV function on day 5 and day 10 respectively and parameters of IVC dynamics. The strongest correlation were between the changes in D-min and CI on day 5 and 10 of treatment ($r=0.78$ and 0.69 respectively) and also between the changes in IVC-CI% and CI on day 5 and 10 ($r=0.68$ and 0.72 respectively).

These results mean that changes in IVC dynamics in patients with acute exacerbations of CHF reflect changes in parameters of LV functions during aggressive medical therapy in ICU and also proved the validity of IVC dynamics (measured simply by M-mode and 2D-echocardiography) in hemodynamic monitoring of LV functions in HF patients.

These results coincide with the results obtained by Hollerbach et al., [20], who studied the validity of IVC dynamics as a clinical tool to assist in monitoring of therapy in patients with CHF. In their study, IVC diameter decreased continuously and significantly ($p < 0.003$) from day 1 to day 10 of diuretic therapy. Also, at the beginning of therapy the IVC-CI% was significantly lesser in patients that in controls. However, after 10 days of therapy this index reached similar values to those observed in controls. They concluded that ultrasonic measurements of IVC diameters and inspiratory movements are a quantifiable and reliable approach to assess the hypervolemia associated with CHF. Also, normalization of IVC-CI% correlates with successful diuretic therapy and can be reliably used for beside assessment and monitoring treatment in CHF patients.

**Albuminuria and IVC dynamics:**

In our study we have studied the reliability of proteinuria as a monitoring tool for successful therapy for CHF in association with other echocardiographic parameters. We found a statistical correlations between the change in albuminuria and the change in D-min and IVC-CI% on day 5 of treatment ($r$-value=0.60 and 0.67 respectively, $p$-value <0.05) and on day 10 of treatment ($r$-value =0.61 and 0.70 respectively, $p$-value <0.05). Also, decrease in albumin excretion was correlated significantly to change in parameters of LV function (EF% and CI) on day 5 of treatment ($r$-value=0.53 and 0.62 respectively, $p$-value <0.01) and on day 10 of treatment ($r$-value=0.56 and 0.63 respectively, $p$-value <0.01).

These results confirmed the association between IVC dynamics, echocardiographic parameters of LV function and the albuminuria as markers for CHF and their reliability as noninvasive tools for monitoring therapy.

**Conclusion:**

- IVC dynamics are valid and reliable as a simple, noninvasive hemodynamic tool in diagnosis and monitoring therapy for acute exacerbation of CHF.

- IVC dynamics can replace echocardiographic parameters of LV function at beside evaluation in ICU for monitoring therapy of acute exacerbation of CHF.

- IVC dynamic changes correlate with albumin excretion.

**References**


