Evaluation of the Physical Parameters on Ischemic Heart Disease Patients Using Echocardiography

Marrwa K. Mohammed, Samar I. Essa*

Department of Physics, College of Science, University of Baghdad, Baghdad, Iraq

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Abstract

Background: Left ventricular function and volumes have major diagnostic and prognostic importance in patients with various cardiac diseases, such as ischemic heart disease which is a life-threatening heart disease condition characterized by systolic dysfunction and a decrease in cardiac output. According to left ventricular ejection fraction, the degree of ischemic heart disease was classified as mild, moderate, and severe. To determine cardiac function and hemodynamics, the echocardiography technique is used, which is a noninvasive diagnostic method.

Patients and Methods: The study included 216 patients between 25 and 75 years old; 121 males and 95 females; 265 normal individuals (age range: 25 to 75 years old); 84 males and 181 females. Doppler echocardiography was used to assess left ventricular function. The measurements included left ventricular end-diastole diameter (LVIDd), left ventricular end-systole diameter (LVIDs), and left ventricular ejection fraction (LVEF%).

Results show a significant correlation in the values of EDV, ESV and LVEF (all: p < 0.05). The change difference in left ventricular ejection fraction (LVEF) was (61.88%), (47.57%), and (36.76%), respectively, for all degrees of ischemia. While the change difference in left ventricular end diastolic volume (LVEDV) between patients and control groups was (173.38%), (248.83%), and (289.82%), respectively. On the other hand, the change in left ventricular end systolic volume (LVESV) was (258.73%), (495.13%), and (569.72%), respectively, for all degree of ischemia.

Conclusion: The findings suggest that diastolic dysfunction is more common in patients than in healthy people. This could be due to the adverse effects of ischemic heart disease on the cardiac muscle. These changes in left ventricular structure may include left ventricular hypertrophy, increase in stiffness, and reduction in compliance.

Keywords: Doppler Echocardiography, ischemic heart disease, left ventricular systolic dysfunction

*Email: samaroem78@yahoo.com
الخلاصة
وظيفة البطين الأيسر وأحجامه ليأى يوقفية تذكرة وانذارية كبيرة في السرضى الذين يعانهم من أمراض قلبية مختلطة مثل مرض نقص التروية هو مرض قلبي يهدد الحياة ويتميز بإلهام وظيفي انقباضي والاعتلال في النجا القلب. تم تصنيف درجة الإصابة بأمراض نقص التروية حسب النادر الناشئي للبطين الأيسر، متضمنة (خفيفة ، معتدلة ، وشديدة). تقنيات تخطيط صدى القلب هي طريقة تشخيصية غير جراحية تستخدم لمجرد وظيفة القلب والثانيات. تم ترشيح درجة الإصابة بأمراض نقص التروية حدب الكدر القذفي لمبطين الأيسر، متزسشة (خفيفة ، معتدلة ، وشديدة) . تقشية تخطيط صدى القلب يتيح تقييم وظيفة البطين الأيسر

البطين الأيسر عند نهاية الانبساط (LVIDd) ، وقطر البطين الأيسر عند نهاية الانقباض (LVIDs) 

المصنف للبطين الأيسر

بينت النتائج أن فرق التعبير في الحجم الانبساطي لنهاية البطين الأيسر بين السرضى ومجسهعات الاصحاء كانت (∑)73.8% (0.05) في البطين الأيسر، بينما كان التعبير في الجزء المصنف للبطين الأيسر (EF) %36.7% في البطين الأيسر (0.05). من ناحية أخرى ، كان النتائج في حجم انقباض نهاية البطين الأيسر (LVEF) %69.7% ، مع وجود فروق معينة (p <0.05) .

1. Introduction

Ischemic heart disease is a common cause of cardiovascular disease leading to ischemic cardiomyopathy and heart failure [1]. A decrease in coronary artery flow might be symptomatic or asymptomatic, occur during activity or rest, and result in myocardial infarction or angina [2].

The inability of the heart to pump enough blood to the body's needs is referred to as heart failure (HF). Most heart failures are associated with impaired left ventricular systolic function [3]. Depending on the left ventricle's ability to contract or relax, heart failure can be divided into two categories, heart failure with a reduced ejection fraction or heart failure with a preserved ejection fraction [4].

It is important that the classification of ischemia is according to the degree of the disease, mild, moderate, and severe. Assessment of left ventricular function may be obtained noninvasively using echocardiography or radionuclide techniques [5].

Echocardiography is a noninvasive diagnostic tool used to detect changes in cardiac parameters such as cardiovascular structure function and hemodynamics that characterize disease processes. Several echocardiographic measurements have long been known to provide useful cardiovascular outcome predictive information; such include left ventricular hypertrophy, aortic stenosis, and the left ventricle ejection fraction [6].

Although the left ventricular ejection fraction is a linear and continuous variable, gross classification into "normal" or "abnormal" has proven clinically useful. Utilizing left ventricular ejection fraction of 45% or above as normal and less than 45% as moderate. Patients with less than 35% ejection fraction have been found to have the severest degree of ischemia [7].

This study aims to assess the left ventricular hemodynamic changes measured by Doppler echocardiography in ischemic heart disease patients.
2. Patients and Methods

This study included 216 patients with ischemic heart disease (age range between 25 and 75 years old), 121 males and 95 females, with a mean age of 57.11 ± 12.94 years, and 265 normal individuals (age range 25 to 75 years old), 84 males and 181 females, with a mean age of 56.76 ± 12.24 years. The study was carried out at Baghdad Teaching Hospital/ Medical City/Baghdad, Iraq, in the echocardiography unit between December 2019 and December 2020. All patients and healthy volunteers were informed of the study's purpose and consented to participate.

The study's strategy included the following steps:

Echocardiography machine (GE Vivid E-9) was used, as well as a phased-array probe with a frequency of 2-4 MHz.

During quiet respiration with the patients lying in the left decubitus position echocardiograms were obtained, using M-mode echocardiography, two-dimensional (2D)-guided M-mode approach with the left ventricular (LV) alignment in the parasternal long axis view was on-axis. Using the leading-edge to leading-edge technique, measurements of LV end-diastolic diameter (LVEDD) were obtained in end-diastole, and LV end-systolic diameter (LVESD) were obtained in end-systole [8].

The modified Simpson's biplane method was used to calculate LVEF. LV volumes were traced at the interface between the compacted myocardium and LV cavity in both the apical 4- and 2-chamber views. LV end-diastolic volume (LVEDV) was measured at end-diastole and LV end-systolic volume (LVESV) at end-systole.

LVEF was calculated by

\[
LVEF = \frac{(LVEDV - LVESV)}{LVEDV} \quad \ldots \ldots (1)
\]

Where there was apical foreshortening in the apical 2-chamber view, LV volumes and LVEF were calculated using the single apical 4-chamber view. Where endocardial definition was poor in both apical four- and two-chamber views, the LVEF was visually estimated.

Definitions of preserved and reduced LVEF were LVEF ≥50% and LVEF <50%, respectively [8].

2.1 Statistical Analysis

Microsoft Office Excel 2010 was used to measure all the results. To compare variables between patients and control subjects, the student's t-test was used. A p-value of less than 0.05 was used to describe statistical significance. The mean, standard deviation, and percentages were used to represent all of the data.

3. Results

The measurements of the LVEF and volumes for all degrees of ischemia are presented in Table 1. The difference in LVEDV, LVESV, and EDV/ESV ratio between patient and control were (173.38%), (258.73%), and (62.33%), respectively, giving a significant p-value < 0.05, while the percentage change of LVEF% was (61.88%) with significant p-value < 0.05, for ischemia degree (1 and 2).

In the ischemia degree 3, the difference in LVEDV, LVESV, and EDV/ESV ratio between patient and control was (248.83%), (495.13%), and (46.04%), respectively, with a significant p-value < 0.05, whereas the percentage change of LVEF was (47.57%) with a significant p-value < 0.05.

The difference in LVEDV between patient and control for ischemia degree 4 was (289.82%) and a significant p-value < 0.05.

The change in LVESV between the patient and control group was (569.72%) also significant with a p-value < 0.05. The change in EDV/ESV ratio was (54.25%) with a significant p-value < 0.05, while the change in EF% was (36.76%) with a significant p-value < 0.05.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Control Mean ±SD</th>
<th>Patient Mean ±SD</th>
<th>(\frac{\text{change}%}{\text{Patient}} \times 100%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV EDV (ml)</td>
<td>80.98 ± 25.99</td>
<td>140.41 ± 36.50</td>
<td>173.38%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LV ESV (ml)</td>
<td>29.20 ± 14.19</td>
<td>75.55 ± 24.17</td>
<td>258.73%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EF%</td>
<td>67.69 ±10.27</td>
<td>41.89 ± 2.23</td>
<td>61.88%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EDV/ESV</td>
<td>3.08 ± 0.907</td>
<td>1.92 ± 0.44</td>
<td>62.33%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Degree of ischemic 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV EDV (ml)</td>
<td>82.68 ±25.77</td>
<td>205.74 ± 91.52</td>
<td>248.83%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LV ESV (ml)</td>
<td>30.81±13.98</td>
<td>152.55 ± 53.65</td>
<td>495.13%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EF%</td>
<td>66.57 ± 10.29</td>
<td>31.67 ± 2.12</td>
<td>47.57%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EDV/ESV</td>
<td>2.91 ±0.74</td>
<td>1.34 ±0.4</td>
<td>46.04%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Degree of ischemic 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV EDV (ml)</td>
<td>83.56 ±24.78</td>
<td>242.18 ± 54.11</td>
<td>289.82%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LV ESV (ml)</td>
<td>32.27±12.90</td>
<td>183.85 ± 79.32</td>
<td>569.72%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EF%</td>
<td>64.77 ±7.57</td>
<td>23.81 ± 1.041</td>
<td>36.76%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EDV/ESV</td>
<td>2.82 ±1.11</td>
<td>1.53 ± 0.66</td>
<td>54.25%</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

4. Discussion

Imbalance in myocardial oxygen supply and demand causes myocardial ischemia, which is most commonly caused by insufficient myocardial perfusion due to atherosclerotic coronary artery disease. Hypoperfusion causes an ischemic cascade of intracellular alterations, leading to a shift in cellular metabolism away from glucose and toward fatty acids [9]. Reuptake of calcium into the sarcoplasmic reticulum fails due to a decrease in ATP production due to a decrease in oxygen supply needed for aerobic metabolism. Consequently, the first manifestation is diastolic dysfunction, followed by systolic dysfunction and abnormal repolarization [10].

There are different degrees of ischemia since myocardial damage occurs during ischemia and reperfusion. Understanding the heart's reaction to injury like ischemia demands complete knowledge of myocardial structure, metabolism, perfusion, and function[11].

Hypertrophy and ischemia are two factors that can cause decreased left ventricular (LV) relaxation, and increased LV stiffness. There is much evidence in the literature that ageing can increase stiffening of the vasculature of the left ventricle due to the development of fibrosis [12].

Fibrosis, caused by an increase in collagen content in the muscle, is linked to myocardial stiffness, which, in turn, increases stiffness [13].

The study population was divided according to their degree of ischemia. This study showed that the increase in the size of end systolic volume was higher than the increase in end diastolic volume. This increase is proportional to the degree of severity of the disease. The ejection fraction decreased as the size of the end systolic volume increased, meaning that the greater the dilatation, the lower the ejection fraction (Table 1). The large increase in ESV for all degrees of ischemia in the patients group suggests that the contribution to heart failure(appeared on EF%) is significantly related to systolic stroke rather than diastolic, as the rise in end systolic volume is greater than the rise in end diastolic volume (Table 1). The results showed that the effects of dilation and myocardium weakness were more noticeable during systole than diastole since systolic stroke necessitates muscle strength for the heart to pump blood to various body parts.
In conclusion, the results showed that the reduced ejection fraction is associated with ischemia, and is related to end systole contraction, not diastole. Moreover, a lower ejection fraction is also related to the severity of the ischemic end-systolic volume, leading to more dilatation.

5. References


