CONGESTIVE HEART FAILURE IN PATIENTS WITH SYSTEMIC HYPERTENSION: A RETROSPECTIVE ANALYSIS WITH GATED BLOODPOOL SCINTIGRAPHY

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Abstract

Congestive heart failure (CHF) is a clinical syndrome that may be a manifestation of inadequate myocardial contraction (myocardial failure), volume overload or inadequate ventricular filling. In the Framingham Heart Study in participants aged 40 to 89 years and free of chronic heart failure, Levy, Larson, Vasan, Kannel & Ho (1996:1560) found a substantially greater risk for the development of CHF in both hypertensive men and women. The reason for CHF was not established i.e. systolic or diastolic dysfunction or both. More recently the effect of hypertensive left ventricular hypertrophy on left ventricular systolic function was reported in the Losartan Intervention for Endpoint (LIFE) study (Wachtell, Rokkedal, Bella, Aalto, Dahlöf, Smith, Roman, Ibsen, Aurigemma & Devereux, 2001:57). The left ventricular ejection fraction (LVEF) remains normal in most patients with left ventricular hypertrophy, but abnormal diastolic left ventricular filling in patients with high left ventricular mass was reported. The purpose of this study was to analyze the prevalence of systolic and diastolic dysfunction (or both) by means of radionuclide bloodpool scintigraphy in patients with the clinical diagnosis of hypertensive heart failure, referred to the Nuclear Medicine Department, Universitas Hospital, Bloemfontein. The LVEF did not differ significantly between the two groups, and remain within normal limits, but the mean PFR and TPF differ significantly between the two groups, indicating diastolic dysfunction in the study group. The differentiation between systolic and diastolic dysfunction has become of utmost importance because of differences in the therapeutic approach to congestive heart failure. Conventional treatment of heart failure with vasodilators, digitalis and diuretics, is irrelevant and even dangerous with diastolic dysfunction, and must be treated preferably with beta-blockers and Ca-antagonists.

1. INTRODUCTION

The heart is a muscular pump. In a healthy individual, the heart circulates blood through the body by systolic contraction and thus ejects blood into the great vessels and systemic circulation. Diastolic relaxation allows blood to fill the ventricle in preparation for the next systolic contraction. The proper function of the heart depends both on normal contraction in systole and normal relaxation in diastole. Many diseases can weaken the heart muscle and affect its ability to contract normally. Some of these diseases include ischemic heart disease, valvular heart disease, severe hypertension, thyroid disease, alcoholism, drug abuse, chemotherapy treatment, idiopathic dilated cardiomyopathy and viral infection. These diseases have varying mechanisms, but all lead to the final common result of a heart which is enlarged and contracts weakly. This is known as dilated cardiomyopathy, or “enlarged heart”, and could be the cause of congestive heart failure (MIMG, 2002:Online).

Congestive heart failure (CHF) is a clinical syndrome that is a manifestation of inadequate

myocardial contraction (myocardial failure), volume overload or inadequate ventricular filling (Dougherty, Naccarelli, Gray, Hicks & Goldstein, 1984:778). It is characterized by signs such as shortness of breath due to pulmonary congestion, as well as fluid retention with general oedema and ascites. With a low cardiac output, symptoms such as fatigue and poor exercise tolerance can occur (Cardinale, 2001). CHF, which is defined as the inability of the left ventricle to generate an adequate cardiac output, is usually associated with impaired left ventricular systolic function. However, symptoms of CHF may arise in the absence of any systolic abnormality (Spencer & Lang, 1997:1).

CHF is a common end point of most forms of serious heart disease. In severe congestive heart failure, both the left and right ventricles are usually performing inadequately. However, with most disease processes, one or the other chamber will usually become compromised first (Standler, 1994:218).

The two main categories of heart failure are systolic and diastolic heart failure. In systolic heart failure, the heart muscle pumps blood out with less strength than normal. Over time, the pumping chambers thin and enlarge, with blood backing up in the organs (University of Maryland Medicine, Online).

With abnormal diastolic function, the left ventricle is stiff and does not fill adequately at normal diastolic pressures. This leads to either decreased left ventricular end-diastolic volumes with a fall in cardiac output or a compensatory rise in left ventricular filling pressure with pulmonary congestion (Flavell & Stevenson, 2001:Online).

Hypertension is a cardinal precursor of congestive heart failure. Although other precursors have been identified, hypertension is the most common condition antedating heart failure in the general population. The population-attributable risk of hypertension for congestive heart failure, the time course of the progression from hypertension to congestive heart failure, and the relative contributions of several mechanisms by which hypertension contributes to the pathogenesis of congestive heart failure have not been fully explained (Levy et al., 1996:1557).

High blood pressure increases the workload of the heart, and over time, this can cause thickening of the heart muscle and symptoms of hypertensive heart disease (Medical Encyclopedia, 2003:Online). Prior studies documented the efficacy of hypertension treatment in reducing the incidence of congestive heart failure, but these intervention trials provide little insight into the mechanisms by which hypertension predisposes to congestive heart failure or by which hypertension treatment prevents or delays the onset of overt heart failure.
In the Framingham Heart Study done in participants aged 40 to 89 years and free of chronic heart failure, Levy et al. found that hypertension antedated the development of heart failure. After adjusting for age and heart failure risk factors in proportional hazard regression models, the hazard of developing heart failure in hypertensive versus normotensive subjects was about twofold higher in men and threefold higher in women. The heart failure criteria used by the Framingham Study are clinical, and were diagnosed prior to the introduction of echocardiography or radionuclide ventriculography. The reason for CHF (i.e. systolic or diastolic dysfunction, or both) was not established.(Levy et al., 1996:1557). A more recent LIFE Study reported normal left ventricular ejection fraction in most patients with left ventricular hypertrophy (Wachtell et al., 2001:58).

CHF resulting from diastolic left ventricular dysfunction may be clinically indistinguishable from systolic dysfunction. The clinical impression developed that diastolic dysfunction is underestimated as a possible cause for CHF (Levy et al., 1996:1561). To accurately distinguish between systolic and diastolic dysfunction, left ventricular function must be assessed. This can be accomplished amongst others with either echocardiography or radionuclide ventriculography (Spencer & Lang, 1997:5).

2. PURPOSE OF THE STUDY

The purpose of this study was to analyze the prevalence of systolic and diastolic dysfunction (or both) by means of radionuclide bloodpool scintigraphy in patients with the clinical diagnosis of hypertensive heart failure, referred to the Nuclear Medicine Department, Universitas Hospital, Bloemfontein. The major advantages of the radionuclide technique are the ability to measure relative volume changes with time without the geometric assumptions necessary for echocardiographic techniques, its ease of performance, lack of dependence on patient anatomy, production of data in a digitized format ready for computer processing, generation of the measurements of diastolic function, as well as evaluation of both ventricles at the same time (Soudry & Manting, 1994:Online).

3. MATERIALS AND METHODS

3.1 Study group

The study group consisted of seventeen patients (6 men, 11 women; mean age 64 years, SD 18 years) with the clinical diagnosis of hypertensive heart failure, who were referred to the Nuclear Medicine Department, Universitas Hospital, Bloemfontein for Gated Bloodpool Scintigraphy. They were studied retrospectively with specific reference to systolic ejection fraction and diastolic function i.e. Peak Filling Rate (PFR) and Time To Peak Filling (TPF).

Patients with Diabetes Mellitus, kidney failure, any ischemic or other cardiovascular illness, cerebrovascular illness or other severe systemic illness were excluded from the study. Patients with a technical unsatisfactory study were also excluded (e.g. Electrocardiogram (ECG) signal problems, patients with dysrhythmia and abnormal movement of the patient during acquisition).

A comparable group of twenty-four persons (13 men, 11 women; mean age 57 years, SD 12 years) without hypertension or congestive heart failure and a normal reported Gated Bloodpool Scintigraphy using the above-mentioned exclusion criteria, were studied in a similar manner. The data obtained were used to determine the normal values for PFR and TPF.

Ethical approval for this study was obtained from the Ethical Committee of the University of the Free State (25 May 2002, ETOVS NR 88/02).

3.2 Imaging technique

The method used for obtaining the images was Planar Gated Equilibrium Radionuclide

Ventriculography. Red blood cell labeling was performed by the in vivo technique with 25 mCi 99mTc-pertechnetate following intravenous stannous pyrophosphate. The images were performed in a modified left anterior oblique (LAO) position to give the best separation of the right and left ventricles.

A Gamma camera equipped with a low energy all purpose (LEAP) collimator was used to acquire the images. Gated equilibrium acquisition was carried out using 32 frames per cardiac cycle, for a total of 9 000 000 counts. Consecutive R waves (the first positive deflection on the electro-cardiogram reflecting early depolarization of the left ventricle of the heart) were used as gating signals.

3.3 Image processing

Standard semi-automatic edge detection and background subtraction were used in order to produce a radionuclide time-activity curve. All edges were visually inspected for accuracy. The background-corrected time-activity curve obtained was differentiated using a digital filter to yield a derivative curve from which the peak filling rate was obtained.

The most rapid change in ventricular volume is called peak filling rate (PFR). It usually occurs in the initial period of fast filling. The cardiovascular nuclear medicine computer can calculate the first derivative of the time-activity curve. This derivative curve represents the rate of change of counts over the ventricle. It is typically normalized to the end-diastolic volume (EDV), and expressed as EDV per second. The average normal PFR is > 2,5 EDV/s. The age and heart rate of the patient must be considered when diastolic measurements are assessed.

A second useful index of diastolic function is the time to peak filling rate (TPF) the interval in milliseconds, between a point at which the peak filling rate occurs. Normal values are < 180 ms.

There is an artifact in the later part of the curve as a result of intrinsic variability of heart rate with the later frames of the acquisition containing less activity that may be anticipated. It is therefore difficult to generate accurate information about atrial systole (Soudry & Manting, 1994:Online).

3.4 Statistical analysis

Continue data were expressed as the mean ± SD. The paired Student t-test was applied to the data to determine significant differences. Statistical significance was defined as p 0,05. The 95% Confidence Interval (CI) for the mean difference for paired data were calculated. If the CI excludes the value zero, there was a statistically significant difference.

4. RESULTS

The LVEF did not differ significantly between the two groups, and remain within normal limits. The mean PFR and TPF differ significantly between the two groups, indicating diastolic dysfunction in the study group. See Table 1.

4.1 Table 1.

Summary of the diastolic and systolic parameters in the normal and study group

	Normal	Study Group	Student t-Test	CI
PFR(EDV/s)	2.064±0.329	1.485±0.707*	0.005	0.246:0.9117
TPF(ms)	141±37	215±95*	0.006	-117.6:-30.97
LVEF(%)	63.5±9.2	58.9±14.7	0.431	-3.647:8.4605

* Indicates a statistically significant difference between values of the control- and the study group.

5. DISCUSSION AND CONCLUSION

The results of this study correspond with the LIFE study reporting that the LVEF remains normal in most patients with left hypertrophy, but that abnormal diastolic ventricular filling exists. The criteria of heart failure used by the Framingham Heart Study were clinical and diagnosed prior to the introduction of techniques such as echocardiography and radionuclide ventriculography. Criticism was mainly on its inability to distinguish between diastolic and systolic left ventricular dysfunction.

Systemic hypertension is probably a rare cause of a weak dilated myopatic ventricle judged to the LVEF that remained normal in the study group. The clinical syndrome of heart failure associated with hypertension was, in most cases, due to delayed ventricular filling on account of diastolic dysfunction.

Conventional treatment of heart failure with vasodilators, digitalis and diuretics, is irrelevant and even dangerous with diastolic dysfunction. Diastolic dysfunction must be treated preferably with beta-blockers and Ca-antagonists. Control of hypertension is very important, since left ventricular hypertrophy as a cause of diastolic dysfunction, is often reversible.

6. REFERENCES

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