SOME ASPECTS OF DILATED CARDIOMYOPATHY
AS SEEN AT KENYATTA NATIONAL HOSPITAL WITH
EMPHASIS ON ECHOCARDIOGRAPHIC FEATURES.

BY

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A DISSERTATION SUBMITTED IN PART
FULFILLMENT OF THE DEGREE OF MASTER OF
MEDICINE (MEDICINE), UNIVERSITY OF NAIROBI.
DECLARATION

This dissertation is my original work and has not been presented for a degree in any other University.

Signature

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This dissertation has been submitted for examination with my approval as the University supervisor.

Signature

DR. WILLIAM LORE
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the Wellcome Foundation for allowing me to use their echocardiographic machine. I wouldn't end without mentioning my wife for her forbearance and for allowing me to turn her bedroom into one mess of a study! Finally but not least, many thanks to Mrs. Margaret Kamau for typing this manuscript.
A total of 37 patients were studied, 16 males and 21 females. The ages ranged from 13-78 (mean ± S.D. = 42.7 ± 17.9). Almost all the males (except one) were 30 years and above, while the females showed a peak in the twenties followed by another from the fifties.

All the patients presented in severe heart failure, most (81%) being in New York Heart Association (NYHA) class 4. Third heart sound was universal, while murmur of mitral regurgitation was heard in about two thirds. Chest pain was present in 10 patients (27%) but only in one case was it angina-like. Embolic phenomena occurred in two patients, both in association with pregnancy.

Mean rate(velocity) of circumferential fibre shortening (Mean Vcf) was depressed in all patients, range, 0.18-0.95 circ/sec, Mean ± S.D. = 0.48 ± 0.22 circ/sec.

Electrocardiographic abnormalities were present in all except one patient. ST - T changes were the most common (64.9%), followed by left bundle branch block (LBBB) (24.3%).
No single case of atrial fibrillation was seen.

Pregnancy was a strong contributory factor in the female population, being associated in 52% of the cases.

Association with alcohol was observed exclusively in the males. There was association in 62.5% of the males.

Elevated blood pressure was found in 6 patients (16.2%).
Cardiomyopathy is currently defined as heart muscle disease of unknown cause. This follows the recommendation of the World Health Organisation/International Society and Federation of Cardiology (WHO/ISFC) task force on the definition and classification of cardiomyopathies. Excluded by this definition are congenital heart defects, rheumatic heart disease, ischaemic heart disease and hypertensive heart disease. Also excluded are diseases of heart muscle but which are associated with specific disease entities as for example in amyloidosis, and systemic lupus erythematosus/hemochromatosis. These are called specific heart muscle diseases.

The cardiomyopathies are classified by the WHO/ISFC task force into...
(1) Dilated (congestive) cardiomyopathy, the subject of this study.

(2) Hypertrophic cardiomyopathy. This may or may not be accompanied by obstruction.

(3) Restrictive cardiomyopathy.

The basic abnormality in dilated cardiomyopathy is impairment of ventricular systolic function. The cardinal feature of dilated cardiomyopathy therefore is depressed left ventricular ejection fraction. This secondarily leads to elevation of left ventricular end diastolic volumes and pressures. The cardiac output may or may not be depressed depending on the stage of the disease. Similarly left atrial pressure may or may not be elevated depending among other things on venous return, sodium intake, ventricular compliance and
presence of anaemia. These are therefore not primary features of the disease and their absence does not invalidate the diagnosis. There is no impairment of diastolic properties and compliance is usually supranormal.

Clinically, usually the earliest presenting features are cardiomegaly and the presence of a third heart sound. A fairly common finding in the Western world is the presence of angina-like chest pain in the absence of other evidence of ischaemic heart disease with normal coronary arteries on angiography. One series reports 20.1% of the patients as having angina, with 10% having it as the presenting feature. Arrhythmias are said to occur commonly with atrial fibrillation being noted in 15-20% of cases. Radiography invariably shows cardiomegaly. Functional mitral regurgitation occurs in up to two thirds of cases. Various electrocardiographic (ECG) changes are seen and are not specific, being almost invariably present. The ST segment and T-wave changes being the most common, bundle branch block,
usually left, abnormal Q waves, prolonged Q interval, right ventricular hypertrophy (RVH), left ventricular hypertrophy (LVH), right axis deviation (RAD), left axis deviation (LAD), P wave abnormalities and atrio-ventricular (AV) blocks.\textsuperscript{3,9,10,11} The arrhythmias seen are atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia and ventricular ectopics.\textsuperscript{3,9,10,11}

Echocardiography is a useful non-invasive tool in the diagnosis of dilated cardiomyopathy and is usually recommended as the diagnostic method of choice.\textsuperscript{12,13} Echocardiography can show the features of dilated cardiomyopathy before heart failure is apparent.\textsuperscript{4,12} These features are:

1. dilatation of the ventricles
2. poor ventricular contractility
3. there may be poor separation of the mitral valve leaflets and eccentric position of the mitral valve.
Pathological examination reveals dilatation of all chambers\(^3,4\). This is invariably accompanied by hypertrophy which is however not proportional to the degree of dilatation\(^3,4\). Mild endocardial scarring is common\(^3\).

Histology shows varying degrees of cellular infiltrates and fibrosis which are non-specific and not useful in establishing an aetiological diagnosis, being found in other cardiomyopathies as well as other heart diseases e.g. rheumatic or ischaemic\(^4,12\). One study by Olsen et al showed neuronal depopulation in affected hearts as compared to controls\(^15\). The coronary arteries remain normal\(^4\).

By its definition, the aetiology of the condition is unknown\(^1,2\). The condition is thought to result from an interaction of a number factors, each of which is independently/incapable of causing the disease\(^6,9\). The factors mentioned include genetic predisposition, infections especially viral, disordered cellular immunity, hypertension, nutritional deficiencies, pregnancy and heavy alcohol intake\(^6,9,16,17\).
The finding of a syndrome resembling dilated cardiomyopathy in a region of China where selenium deficiency is common has suggested a possible role of mineral deficiency in pathogenesis. The possibility of viral aetiology has received the most attention. In one series 20% of cases were preceded by a viral-like illness. Elevated antibodies to viruses especially to Cocksackie B has been demonstrated. The postulation is that probably a viral infection triggers off an autoimmune process that damages the heart in a genetically predisposed individual, a process that is worsened by the coexistence of an extra factor like pregnancy or heavy alcohol intake.

Goodwin used the expression "conditioning factors" to refer to those conditions that are associated with dilated cardiomyopathy but which in themselves cannot be considered causes. These are, pregnancy, heavy alcohol consumption and elevated blood pressure, thus justifying terms such as alcoholic or peripartum cardiomyopathy.
Peripartum cardiomyopathy is defined as heart disease occurring in the last month of pregnancy or in the first five post-partum months in the absence of other demonstrable aetiology or preceding heart disease. Advanced age, high parity, twin toxæmia, hypertension and slow regression of the heart size have been identified as poor prognostic factors. Usually 50% of the patients improve with regression in heart size within six months while the other 50% follow a relentlessly downward course. Relapse in subsequent pregnancies is common. Most of the cases (82%) are post partum, 71% in third or subsequent pregnancy and 48% are over 30 years in one series. Alcohol is recognised as being deleterious to the heart both following acute ingestion and in chronic abuse. Alcoholic cardiomyopathy is now recognised as an entity distinct from the nutritional (thiamine) deficiency syndrome that is common in alcoholics. While in acute ingestion the damage is due to direct
toxic effects of alcohol and its metabolites on myocardial cell function, in the heart disease following chronic abuse, there is thought to be underlying factors such as infective, immunological or nutritive hence the term alcoholic cardiomyopathy. Abstinence has been shown to improve prognosis. Bed rest has also been used as a mode of therapy.

The relationship between hypertension and dilated cardiomyopathy is one that has been fraught with controversy. In the 1970's some workers postulated that dilated cardiomyopathy was indeed burnt out hypertension. Goodwin, however, strongly argued against this pointing at, for example, the differences in the degree of hypertrophy and electrocardiographic findings between hypertensive disease and dilated cardiomyopathy. Although dilated cardiomyopathy is no longer considered the same as hypertensive heart disease, the possibility of the coexistence of hypertension and cardiomyopathy is largely accepted. As stated above hypertension is thought to be one of
the multiple factors that interact to produce the syndrome. It is also considered a prognostic factor in peripartum cardiomyopathy. In one study by Falase et al they analysed the influence of lowering blood pressure on the clinical course of their patients, thus implicitly recognising hypertension as an independent variable.

Western literature points to a generally poor prognosis with a five year survival of 25-40% with the highest mortality being in the first year after diagnosis. In one series two thirds of the patients died within two years. Poor prognostic factors have been reported as age, biventricular heart failure, markedly depressed cardiac index, high left ventricular diastolic volumes and systemic embolisation. Ventricular hypertrophy and normalisation of blood pressure are good prognostic signs.

Reports from Africa indicate that cardiomyopathy is a commonly diagnosed condition. Parker in Nairobi and Nhonoli in Dar es Salaam found 10%
and 11% respectively of cardiovascular admissions to be due to cardiomyopathy. Barr found them to be third commonest cause of emergency cardiovascular admissions accounting for 8 out of 115 admissions. These studies have, however, been retrospective, except for Barr's series which was not specific for cardiomyopathies. In addition, these were very small series. Retrospective studies have certain inherent drawbacks among which is the lack of uniform and clearly defined diagnostic criteria. Rees et al found that a number of patients labelled cardiomyopathy actually had specific heart disorders. The aim of this study has been to undertake a prospective study in which the diagnostic criteria are clearly laid down, in this case the echocardiographic criteria mentioned above. It is the aim of the study to see how the pattern of presentation of our patients compares with reports from the western literature reviewed above in terms of clinical features, electrocardiographic abnormalities and association with alcohol, pregnancy and hypertension. Semi-quantitative assessment of ventricular function by echocardiography
as explained below is also undertaken.

**MATERIALS AND METHODS**

Patients seen at Kenyatta National Hospital in whom a clinical impression of dilated cardiomyopathy had been made were referred for screening.

The patients had a full clinical history taken and were subjected to complete physical examination. History was specifically taken for:-

- duration of symptoms,
- degree of disability, which was then classified according to the New York Heart Association criteria,
- presence of chest pain and its nature,
- intake of alcohol and an attempt to quantity both the quantity and duration,
- association with pregnancy, where the association exists, parity, presence of toxaemia and twins was looked into and any significant associated illness,
In the examination the following were specifically noted:

- blood pressure,
- third heart sound,
- apical systolic murmur and
evidence of pulmonary or systemic embolism.

The patients in whom the impression of dilated cardiomyopathy was confirmed were then subjected to an M-mode echocardiographic examination.

The echocardiographic features were used as the diagnostic criteria and only the patients who satisfied the criteria spelt out in the introduction were entered into the study.

The examination was done using a Honeywell VR-12 physiological recorder incorporating an echocardiographic module with a 2.25 MHz transducer and later a Siemens Echopan KS machine with a 2.0 MHz transducer. The examination was carried out with the patients lying in the left lateral position at about 30° with the transducer placed on the third or fourth intercostal space within 3-4 cm of the left sternal edge.
Left ventricular end diastolic diameter (LVEDD) was taken at the Q wave and the left ventricular end systolic diameter (LVESD) was taken at the point of the maximum posterior motion of the intraventricular septum as recommended by the American Society of Echocardiography and the WHO/ISFC task force on the standardisation of M-mode echocardiography \(^4\text{0},^4\text{1}\). The corresponding right ventricular dimensions were taken at the same point. Left atrial diameter was taken at the point of maximal anterior motion of the posterior aortic wall \(^4\text{0},^4\text{1}\). In all cases the leading echoes were used. \(^4\text{0},^4\text{1}\) The septum and left ventricular posterior walls were observed for degree of motion. Ejection time was determined as the total period of opening of the aortic valves in seconds. In the patients in whom dilated ventricles with poor wall motion was demonstrated and hence included in the study, a semi-quantitative assessment of left ventricular function using mean rate of circumferential fibre shortening (Mean Vcf).
This has been shown to be a good measure of myocardial contractility which compares well with angiographic parameters.\textsuperscript{14,42,43}. It is calculated by the formula:\textsuperscript{14}

\[
\frac{\text{LVEDD} - \text{LVESD}^*}{\text{LVEDD} \times \text{E.T.}} \text{ Circles/sec.}
\]

* LVEDD - left ventricular end diastolic diameter

LVESD - left ventricular end systolic diameter

E.T. - Ejection time.

The patients selected in the study also had the following done:

- chest x-ray - PA view

- twelve lead resting electrocardiogram.

Tests of significance were done using the students t test.
RESULTS

There were thirty seven patients seen, sixteen males and twenty one females giving a sex ratio of M:F = 1:1.3.

Figure I Showing general age distribution.

The histogram shows the age distribution of the studied population.
The age range is 13 - 78 years

Mean ± S.D. = 42.7 ± 17.94

The distribution shows two peaks in the third and fourth decades and in the sixth and seventh decades.

Sex specific Age distribution

Figure II Showing:

Female age distribution

The histogram above show the female age distribution.

The age range is 18 - 78 years

Mean ± S.D. = 42.4 ± 19.52

There is an early peak in the 20's and a late peak in the 50's.
Figure III Showing:

Male age distribution

Range 13 - 70 years

Mean ± S.D = 49.8 ± 15.19

Except for one boy/13 years, all the aged patients were/over 30 years. There is a uniform distribution through the forth to seventh decades.

Despite the difference in the means, there is no statistically/significant age difference between the males and females. However, when the boy aged 13
years is excluded, there is a statistically significant difference (P<0.05).

Clinical features:

(i) New York Heart Association classification

Table I

<table>
<thead>
<tr>
<th>NYHA</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>12</td>
<td>18</td>
<td>30</td>
</tr>
<tr>
<td>TOTAL</td>
<td>16</td>
<td>21</td>
<td>37</td>
</tr>
</tbody>
</table>

All the patients had at least NYHA class 3, with the majority (81%) being class 4. There is no significant sex difference in the class representation.
(ii) Third heart sound:

All the patients seen had a third heart sound on auscultation. Phonocardiography was not performed to corroborate this clinical finding.

(iii) Chest pain:

There was chest pain in 4 (25%) of the males and 6 (28.5%) of the males giving an average of 27%.

No statistically significant difference between the sexes was demonstrated.

- One case was anginal (2.7%)
- Three were pleuritic (8.1%)
- The rest were non-specific, usually left intramammary.

(iv) Apical systolic murmur was heard 24 of the 37 patients (64.9%). The sex specific proportions were:

- Men 10 out of 16 (62.5%)
- Women 14 out of 21 (66.7%)

There was no statistically significant sex difference.
(v) The mean duration of illness was 10.5 months in females and 14.5 months in males.

(vi) Embolic phenomena.

There was one case each of a systemic and pulmonary embolus (5.4%), both cases associated with peripartum cardiomyopathy.

Echocardiographic features:

(i) Mean Vcf (circles/second)

<table>
<thead>
<tr>
<th>Mean Vcf circ/sec</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0 - 0.19</td>
<td>0</td>
<td>2(9.5%)</td>
<td>2(5.4%)</td>
</tr>
<tr>
<td>0.20 - 0.39</td>
<td>9(56.25%)</td>
<td>9(42.9%)</td>
<td>18(48.6%)</td>
</tr>
<tr>
<td>0.40 - 0.59</td>
<td>5(31.25%)</td>
<td>3(14.3%)</td>
<td>8(21.6%)</td>
</tr>
<tr>
<td>0.60 - 0.79</td>
<td>1(6.25%)</td>
<td>5(23.8%)</td>
<td>6(16.2%)</td>
</tr>
<tr>
<td>0.80 - 0.90</td>
<td>1(6.25%)</td>
<td>2(9.5%)</td>
<td>3(8.1%)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>16(100%)</td>
<td>21(100%)</td>
<td>37(99.9%)</td>
</tr>
</tbody>
</table>

Range 0.18 - 0.95 circ/sec
Mean ± S.D. = 0.48 ± 0.22 circ/sec.
Males:

Range: 0.25 - 0.95 circ/sec

Mean ± S.D = 0.47 ± 0.22 circ/sec

Females:

Range: 0.18 - 0.89 circ/sec

Mean ± S.D = 0.49 ± 0.31

The difference is not statistically significant.

It is noteworthy that all the values fall below the normal range (1.02 - 1.94 circ/sec).
Table III showing:
(ii) Left ventricular end diastolic diameter

<table>
<thead>
<tr>
<th>LVEDD (cm)</th>
<th>MALE</th>
<th>FEMALE</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.5 - 5.9</td>
<td>2(12.5)</td>
<td>4(19)</td>
<td>6(16.2)</td>
</tr>
<tr>
<td>6.0 - 6.4</td>
<td>5(31.25)</td>
<td>9(42.9)</td>
<td>14(37.8)</td>
</tr>
<tr>
<td>6.5 - 6.9</td>
<td>4(2.5)</td>
<td>4(19)</td>
<td>8(21.6)</td>
</tr>
<tr>
<td>7.0 - 7.4</td>
<td>1(6.25)</td>
<td>3(14.3)</td>
<td>4(10.8)</td>
</tr>
<tr>
<td>7.5 - 7.7</td>
<td>1(6.25)</td>
<td>1(4.8)</td>
<td>2(5.4)</td>
</tr>
<tr>
<td>8.0 - 8.4</td>
<td>2(12.5)</td>
<td>0</td>
<td>2(5.4)</td>
</tr>
<tr>
<td>8.5 - 8.9</td>
<td>1(6.25)</td>
<td>0</td>
<td>1(2.7)</td>
</tr>
<tr>
<td>Total</td>
<td>16(100)</td>
<td>21(100)</td>
<td>27(99.9)</td>
</tr>
</tbody>
</table>

* Percentages in brackets

Mean ± S.D. = 6.69 ± 1.69 cms.

Age specific means ± S.D.:

Males: 6.96 ± 1.9 cms

Females: 6.48 ± 1.53 cms.

The difference not statistically significant.
Table IV shows ECG changes.

<table>
<thead>
<tr>
<th>ECG changes</th>
<th>No</th>
<th>% age</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST - T changes</td>
<td>23</td>
<td>62.2%</td>
</tr>
<tr>
<td>LBBB</td>
<td>9</td>
<td>24.3%</td>
</tr>
<tr>
<td>LVH*</td>
<td>4</td>
<td>10.8%</td>
</tr>
<tr>
<td>Ventricular ectopics</td>
<td>4</td>
<td>10.8%</td>
</tr>
<tr>
<td>RAD</td>
<td>2</td>
<td>5.4%</td>
</tr>
<tr>
<td>LAD</td>
<td>2</td>
<td>5.4%</td>
</tr>
</tbody>
</table>

All patients except one showed an ECG abnormality. There was no sex difference in representation.

LVH by voltage criteria, $SV_1 + RV_5 (6) \leq 35\text{mm}$. 
Association with hypertension

There was concomitant elevation of blood pressure in 6 patients (16.2%).

There were 4 females and 2 males.

The age range was 18 - 61 (mean ± S.D. = 39.7±14.8 years).

In five of the patients the hypertension was mild with diastolic pressures between 95-100 mmHg.

In one patient the diastolic pressure was 114 mmHg.

The echocardiographic parameters were:

LVEDD. Mean ± S.D. = 6.33 ± 0.87 cm.

Mean Vcf Mean ± S.D. = 0.48 ± 0.2 circ/sec.

None of the three parameters was statistically different from the total patient population.
### TABLE VI SHOWING ASSOCIATION WITH PREGNANCY

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Parity</th>
<th>HT*</th>
<th>Toxaemia</th>
<th>NYHA</th>
<th>LVEDD (cm)</th>
<th>Mvcf (circ/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.V.</td>
<td>35</td>
<td>4</td>
<td>+</td>
<td>0</td>
<td>4</td>
<td>6.5</td>
<td>0.27</td>
</tr>
<tr>
<td>P.K.</td>
<td>27</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>6.4</td>
<td>0.36</td>
</tr>
<tr>
<td>A.A.</td>
<td>35</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>5.6</td>
<td>0.85</td>
</tr>
<tr>
<td>I.A.</td>
<td>30</td>
<td>4</td>
<td>+</td>
<td>0</td>
<td>4</td>
<td>5.7</td>
<td>0.76</td>
</tr>
<tr>
<td>N.M.</td>
<td>18</td>
<td>1</td>
<td>+</td>
<td>+</td>
<td>4</td>
<td>7.2</td>
<td>0.60</td>
</tr>
<tr>
<td>R.A.</td>
<td>18</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>7.0</td>
<td>0.73</td>
</tr>
<tr>
<td>N.W.</td>
<td>30</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>5.6</td>
<td>0.38</td>
</tr>
<tr>
<td>G.A.</td>
<td>21</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>6.5</td>
<td>0.18</td>
</tr>
<tr>
<td>S.O.</td>
<td>38</td>
<td>8</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>6.3</td>
<td>0.55</td>
</tr>
<tr>
<td>P.O.</td>
<td>28</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>5.7</td>
<td>0.89</td>
</tr>
<tr>
<td>J.I.</td>
<td>30</td>
<td>5</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>6.1</td>
<td>0.70</td>
</tr>
</tbody>
</table>

HT* = Hypertension  

female  

In eleven of the twenty-one patients the disease was associated with pregnancy - (52%).  

When only women in the reproductive age (upto 45 years) are considered this percentage rises to 91.7%.
The three associated with high blood pressure all had mild elevation with diastolic of 100mmHg.

Six out of the ten had parity of 4 and above.

Mean age ± S.D = 27.36 ± 22 years statistically significant difference from the total female population studied (0.02>P 0.01).

Mvcf = 0.55 ± 0.22 circ/sec - significant (P<0.001)

LVEDD 6.2 ± 2.73 cm - Not significant.

One patient J.I had twins.
Table III Shows

Association with Alcohol

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>NYHA</th>
<th>Mvcf (Circ/sec)</th>
<th>LVEDD (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.W.</td>
<td>56</td>
<td>M</td>
<td>4</td>
<td>0.50</td>
<td>7.5</td>
</tr>
<tr>
<td>W.K.</td>
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<tr>
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The association with alcohol was observed exclusively in males, occurring in 62.5% of the male population. All the patients had an intake estimated to be equivalent to at least 80 gm of alcohol per day for at least 10 years. Further quantification was difficult:

One patient, G.N., had been a heavy drinker for many years but stopped 5 years earlier.

Age: Mean ± S.D. = 49.7 ± 11.83 years

Mvdf: Mean ± S.D. = 0.48 ± 0.08 circ/sec

LVEDD: Mean ± S.D. = 6.73 ± 0.63 cm.

None of these parameters showed a statistically significant difference from the whole male population studied.
DISCUSSION

Age Distribution

The age distribution reveals a significant sex difference. The male age distribution is characterized by the virtual absence of any patient under 30 years (except for one patient aged thirteen years). This tendency of the disease to begin later in life has been reported in other series in East Africa: Rees et al found only eight of thirty one patients were under 45 years of age. Nhonoli only found two of twenty five patients were under twenty, all the others being over thirty years.

The female age distribution shows an early peak in the 20's and a later peak beginning in the 50's. This earlier peak is mainly contributed to by peripartum cardiomyopathy and reflects its strong contribution in the female population studied. This is discussed further later under peripartum cardiomyopathy. Despite this difference in distribution, there was no statistically significant sex difference in the ages. This, however, became
significant when the single boy of thirteen years was excluded.

The age distribution in this study generally agrees with other series. Lore found an age range of 10-79 years (cf 13-79). Fuster et al found a medium age of 49 years. Shirley et al found a female mean age of 43.3 (cf 42.4) and male mean age 42.3 (cf 49.8).

Sex Distribution

Most of the reported series show a slight male preponderance. Thus in Lore's series the males were 21 (58%) and females 15 (42%). Nhonoli studied 14 men and 11 women. Shirley et al had 90 men and 44 women, while Fuster et al had 64 men and 40 women.

The present study was not designed to show prevalence rates, therefore the higher female ratio (58.8%) may be misleading. However, one possible explanation is the quite high rate attributable to pregnancy. In fact, peripartum cardiomyopathy contributes 27% of the total population studied.
This contrasts with other reports which have given figures of about 10\%.\textsuperscript{8,20}

**Clinical features:**

Our patients invariably presented in gross cardiac failure, the majority belonging to New York Heart Association (NYHA) Class IV. As noted earlier, cardiac failure is not a prerequisite for diagnosis and, in fact, represents a late stage of disease.\textsuperscript{3,4}

Literature reports incidence of heart failure between 38.5\% - 73\%, although the NYHA class is not specified.\textsuperscript{7,9} It is, therefore, apparent that our patients present to us quite late. One possible explanation is the generally, low socio-economic status of the patients, such that they report to hospital when severely symptomatic. Also important is the fact the study population came mainly from the patients who required admission thus having a bias for the sicker patients. Konyatta National Hospital being a referral institution adds to this bias.
All our patients had a third heart sound on auscultation. This is a common finding and has been noted to predate the development of heart failure, since all our patients had overt heart failure, this is not surprising finding. A reasonable proportion was found to have chest pain, however, as opposed to Western literature, in only one patient was it anginal. It is interesting that this was the only non-African in patients studied, being of Asian origin.

The finding of only two episodes of embolisation, one being systemic, contrasts with the reported common occurrence in western reports. The occurrence of pan-systolic murmur of mitral regurgitation (64.9%) compares well with the literature. The duration of illness at diagnosis is also comparable to the 6 months to 2 years reported. One report gave a mean duration of 1.3 years.

The chest x-rays invariably showed an increased cardiothoracic ratio as expected, though this does not appear in the analysis.
Echocardiographic features

The mean rate of circumferential fibre shortening (Mean Vcf) was universally depressed, the highest value being 0.95 cir/sec. This being a measure of myocardial contractility, is consistent with the fact that the basic pathophysiological abnormality is the impairment of myocardial contractility. No reports in the literature were found in which the same echocardiographic parameter was quoted, but angiographic studies give the same finding.

By definition the left ventricular end-diastolic diameter (LVEDD) was elevated in all the patients. There were no significant differences in any of the sub-groups.

Unfortunately, right ventricular dimension could not be analysed because of poor quality of the recorded echograms. This is a problem that has been noted by other workers. This measurement have been useful since dilated cardiomyopathy can be left ventricular, biventricular or right ventricular alone.
Electrocardiographic findings:

The finding of an ECG abnormality in almost all patients agrees with reports in the literature. For example, Lore found an abnormality in all patients in which a record was made while Fuster et al found abnormalities in 80%. The finding of ST-T changes as the most common abnormality agrees with these reports.

The finding of left ventricular hypertrophy in only 10.8% of patients is low for a condition in which, in addition to dilatation of chambers, there is invariably hypertrophy, though not proportional. One reason is the high prevalence of LBBB which invalidates the voltage criteria used. The voltage criteria in itself has been noted to be insensitive. ST-T wave change have also been thought to represent hypertrophy.

The low rate of arrhythmias is a departure from the literature reports which indicate that rhythm disturbances is seen in upto 65% of cases.
Atrial fibrillation alone is reported to occur in 15-20% of cases while none is noted in this study. Indeed, the only rhythm disturbance noted is ventricular ectopics. This not being a follow up study, it is possible that paroxysmal arrhythmias were missed, thus contributing to the lower rate of arrhythmias in this series. However this seems an insufficient explanation for the large difference in the rate of arrhythmias between this study and others in the literature. There seems to be a significant difference. The only local report had 2 patients with atrial fibrillation out of 36. The other significant finding is the absence of any degree of A-V block. Although complete A-V block is said to be uncommon, some degree of A-V conduction abnormality is said to occur in upto 20%. Finally, because of the echocardiographic bias of this study some of the ECG changes reported in the literature were omitted from the questionnaire and therefore were not systematically looked for. These are abnormal Q waves, prolongation of QT interval, P wave abnormalities and poor precordial
R wave progression. Electrocardiographic pattern in cardiomyopathy is obviously a large and interesting field and requires a study on its own to define the pattern of these abnormalities in our local setting.

Association with hypertension.

The association with hypertension in our series is low (16.2%). Goodwin quotes a figure of 25%, while Lore found a figure of 37%. The elevation of blood pressure in this group was generally mild except in one patient. The hypertensive group does not show any difference in terms of severity of disease or ventricular function from the general group. The male/female ratio of 1:2 is most likely spurious in view of the very small numbers. It would be of more interest to follow up these patients to see if the course of disease differs from the non-hypertensives and the influence of normalisation of blood pressure on this, as has been reported elsewhere.
Pregnancy comes out as a quite significant factor in the female population. Literature is scanty on peripartum cardiomyopathy as a proportion of cardiomyopathies in total. However, in Lore's local series it accounted for only 10% (cf 52%). Literature reports that peripartum cardiomyopathy occurs rather late with about 50% of the cases occurring after 30 years. This contrasts with our series in which most were in the 20's, giving us the early peak in the female age distribution. Indeed the age distribution was significantly different from the whole female population. A surprising finding was the significantly lower mean vcf. No explanation for this is apparent. Six out of the 10 patients were Para 4 and above, agreeing with reports of about 70% being in third or subsequent pregnancies. Because of the small number it was not possible to analyse the independent influence of such factors as age, parity,
hypertension on the presentation. This is another sub-group in which a follow up study would be enlightening. The influence of the various prognostic factors listed on the course of disease should be ascertained, as well as the influence of subsequent pregnancy.

**Alcoholic cardiomyopathy**

One of the apparent things in this series is the absence of drinking in the females. This probably reflects the drinking pattern in a low socio-economic, basically rural population from which the bulk of the patients come. Alcohol also comes out as a quite significant factor in the male population, being associated in 62.5%. Reports of these have varied in the literature. Rees et al found the association in 30 of 31 patients. 38 Lore quotes a 44% association but 39% of the group had no record of alcoholic intake noted. 8 In western literature, while Fuster et al reported a 21% association with heavy alcohol intake, Shirley et al
only had 58% of their group admitting to alcohol intake. Nyabundi in an analysis of otherwise healthy men who had heavy prolonged alcohol intake found no significant echocardiographic or ECG abnormalities. The problem of quantifying alcohol intake noted by other workers was encountered, and was worse in the case of traditional brews in which concentrations are not standardised. Follow-up of these patients to assess the influence of cessation of drinking and the reported beneficial influence of prolonged bed rest would be interesting.

Conclusion

As noted earlier, there is paucity of prospective studies on this subject locally. The aim of this study was to contribute to the filling of this gap. However the study suffered from some setbacks. Being an M.Med dissertation it had to be done within a defined, short time. It has also suffered from the technical and logistic problems currently besetting us. However, certain interesting facts are highlighted.
Among these are, the very late presentation of our patients. There is the absence of anginal chest pain as a mode of presentation, and the lack of embolic manifestations. The very high proportion contributed by peripartum cardiomyopathy, and the relatively younger age effected is also significant. There is also the difference in ECG presentation especially the absence of arrhythmias.

More prospective and larger studies, with proper logistical backing are necessary to confirm and better define these findings. It is also important to do follow up studies to see the disease progression in our local situation and define prognostic factors. There is also a need for independent studies of the sub-groups i.e. peripartum and alcoholic cardiomyopathy, and assessment of their contribution to the problem of dilated cardiomyopathy in our local setting.
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Echocardiographic tracing of a patient with dilated cardiomyopathy, showing:
- grossly dilated left ventricle
- poor contractile motion of the ventricular walls.

Legend:
- **LVEDD** - left ventricular end diastolic diameter
- **LVESD** - left ventricular end systolic diameter
- **RV** - right ventricle
- **IVS** - Intraventricular septum
- **LVPW** - left ventricular posterior wall.
Appendix II.

Echocardiographic tracing showing normal left ventricular dimensions and good contractility.

Legend:

LVEDD = Left ventricular end diastolic diameter
LVESD = Left ventricular end systolic diameter
IVS = Intraventricular septum

Right ventricle and left ventricular posterior wall not well outlined.
Appendix III

Selected tracings from the ECG of a patient with dilated cardiomyopathy, illustrating some of the common abnormalities, i.e.

- ST - T changes
- Left ventricular hypertrophy
- Ventricular ectopics.