CHANGES IN LEFT VENTRICULAR ANATOMY DURING
HAEMODIALYSIS, CONTINUOUS AMBULATORY PERITONEAL
DIALYSIS AND AFTER RENAL TRANSPLANTATION

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Summary

The changes in left ventricular anatomy in 30 patients with end-stage renal dis-
ease and stable cardiac function, undergoing regular haemodialysis (10 patients),
continuous ambulatory peritoneal dialysis (10 patients) and after successful
renal transplantation (10 patients) were evaluated by M-mode echocardiography.
Initially all had evidence of left ventricular hypertrophy and dilatation. Re-
evaluation after a mean follow-up of 22 months on each mode of treatment
showed that in the haemodialysis group the left ventricular mass and volume
were increased, while in continuous ambulatory peritoneal dialysis (CAPD) and,
especially renal transplantation, the hypertrophy and dilatation were reversed.
This improvement was probably due to a reduction of cardiac workload.

Introduction

It is well known that chronic renal failure and its maintenance treatment pro-
dcedures predispose to cardiovascular disease [1]. Although chronic haemodialysis
is successful in making productive life possible, 25 to 40 per cent of patients
have evidence of left ventricular hypertrophy and dysfunction [1,2]. Several
factors such as long-standing hypertension, accelerated atherosclerosis, hyper-
dynamic circulation and uraemic ‘cardiomyopathy’ have been incriminated
[1,2]. However in the recent literature there is some evidence that successful
transplantation and continuous ambulatory peritoneal dialysis (CAPD) have a
beneficial effect on cardiac size and function in patients with chronic renal
failure [3,4]. This study further defines the changes in left ventricular anatomy
and function by M-mode echocardiography in patients on long-term haemo-
dialysis or CAPD and after successful renal transplantation.

Materials and methods

Thirty patients with end-stage chronic renal failure were studied, group A: 10
patients on regular haemodialysis, group B: 10 patients on CAPD and group C:
10 patients who were on regular haemodialysis and underwent successful renal transplantation. None of them had evidence of other systemic disease, severe hypertension, cardiac failure or pericarditis during the study. In group C the arterio-venous fistula was patent. Measurements were made within one month after the initiation of regular haemodialysis or CAPD and before renal transplantation and 22 months (range 20–24 months) following the application of each procedure. M-mode echocardiograms were obtained with an Ekoline 20A (Smith, Kline & French) machine attached to a VR-6 Electronics for Medicine osciloscopic recorder. The patients were studied in the left decubitus position by the standard technique.

The left ventricular internal diastolic dimension (LVIDd), systolic dimension (LVIDs), left ventricular posterior wall thickness (PW), interventricular septal thickness (IVS), aortic root dimension (ARD) and left atrial dimension (LAD) were calculated according to the recommendations of the American Society of Echocardiography [5]. From these data the following parameters were calculated: a) Per cent shortening fraction of the left ventricle (ΔD% = LVIDd-LVIDs/LVIDd x 100); b) LAD/ARD ratio; c) left ventricular mass (LVM = 0.77 [(LVIDd+PW+IVS)² - LVIDd²]+2.4); d) the end-diastolic volume (EDV = LVIDd³), stroke volume (SV = EDV - ESV) and cardiac index (CI = SV x heart rate/m²); e) the relative left ventricular wall thickness (R/Th ratio = LVIDd/2PW). All measurements represent the average value of five cardiac cycles.

Data were analysed using the Student's 't' test for paired data.

Results

Clinical data from the three groups are shown in Table I. There were no significant differences in the mean age, the mean concentration of serum creatinine, haematocrit, mean blood pressure and the mean heart rate at the time of the initial evaluation. The changes of the above clinical features during the follow-up period are given in the same table. As during the study the calculated body surface area of the patients did not change the absolute values for chamber size and cardiac performance were compared.

Table II shows that the mean LVIDd increased by 4.8 per cent (p<0.01) in haemodialysis patients but decreased by 2.5 per cent and 2.9 per cent (p<0.01) in CAPD and renal transplantation patients respectively. There was also a significant reduction of the mean values of LVIDs and IVS by 3.7 per cent and 17 per cent (p<0.01) in group C. Although the initial mean values of LVIDd, LVIDs, PW and IVS were significantly greater than those of the control group, a tendency towards normal in groups B and C was observed.

In all groups the initial left ventricular mass was greater than normal. Following haemodialysis the left ventricular mass increased by 24.3 per cent (p<0.01), but decreased during CAPD and, especially, after renal transplantation by 16.3 per cent and 20.7 per cent (p<0.01) respectively. The mean left ventricular end-diastolic volume increased by 15.3 per cent (p<0.01) during haemodialysis, but decreased after renal transplantation by 6.6 per cent (p<0.05). Although during CAPD there was a reduction in end-diastolic volume, this was not statistically significant. Finally, R/Th ratio showed a significant increase during CAPD and after renal transplantation.
### TABLE I. Clinical features of 30 chronic renal failure patients

<table>
<thead>
<tr>
<th>Data</th>
<th>Haemodialysis</th>
<th>CAPD</th>
<th>Renal transplantation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female</td>
<td>6/4</td>
<td>7/3</td>
<td>5/5</td>
</tr>
<tr>
<td>Age (years)</td>
<td>42±11</td>
<td>46±13</td>
<td>40±8</td>
</tr>
<tr>
<td>Duration of chronic renal failure (years)</td>
<td>8</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Serum creatinine (mg/100ml)</td>
<td>13.1±1.9 → 12.1±2.5*</td>
<td>12.4±3.8 → 7.5±1.6</td>
<td>11.8±3.4 → 1.7±0.8</td>
</tr>
<tr>
<td>Haematocrit (%)</td>
<td>23.4±5.8 → 28.2±5.2*</td>
<td>21.3±2.5 → 31.3±4.7</td>
<td>26.8±6.2 → 38.5±7.6</td>
</tr>
<tr>
<td>Mean blood pressure (mmHg)</td>
<td>131±16 → 122±12*</td>
<td>123±12 → 109±10</td>
<td>125±8 → 106±11</td>
</tr>
<tr>
<td>Heart rate (beats per minute)</td>
<td>77±11 → 73±13*</td>
<td>74±6 → 71±9</td>
<td>75±8 → 72±10</td>
</tr>
</tbody>
</table>

* Comparison of initial and follow-up mean values (±SD)

### TABLE II. Echocardiographic data of left ventricular anatomy and performance

<table>
<thead>
<tr>
<th>Data</th>
<th>Control group</th>
<th>Haemodialysis initial</th>
<th>Haemodialysis follow-up</th>
<th>CAPD initial</th>
<th>CAPD follow-up</th>
<th>Renal transplantation before</th>
<th>Renal transplantation follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular internal diastolic dimension (mm)</td>
<td>48±4</td>
<td>52±3</td>
<td>54±3*</td>
<td>53±4*</td>
<td>51±4*</td>
<td>52±4</td>
<td>50±4*</td>
</tr>
<tr>
<td>Left ventricular internal systolic dimension (mm)</td>
<td>32±4</td>
<td>34±4</td>
<td>36±3†</td>
<td>34±6</td>
<td>34±5†</td>
<td>35±4</td>
<td>33±4*</td>
</tr>
<tr>
<td>Left ventricular posterior wall thickness (mm)</td>
<td>9.5±1</td>
<td>10±2</td>
<td>11±2†</td>
<td>11±2</td>
<td>10±1†</td>
<td>10±2</td>
<td>9.0±1†</td>
</tr>
<tr>
<td>Interventricular septal thickness (mm)</td>
<td>10±1</td>
<td>12±2</td>
<td>13±2†</td>
<td>11±2</td>
<td>10±2†</td>
<td>12±2</td>
<td>10±2*</td>
</tr>
<tr>
<td>Left atrial dimension/aortic root dimension</td>
<td>1.1±0.2</td>
<td>1.3±0.3</td>
<td>1.2±0.3†</td>
<td>1.1±0.2</td>
<td>1.1±0.3†</td>
<td>1.2±0.3</td>
<td>1.1±0.2†</td>
</tr>
<tr>
<td>%ΔD</td>
<td>34±5</td>
<td>34±6</td>
<td>35±6†</td>
<td>37±8</td>
<td>36±7†</td>
<td>33±5</td>
<td>34±5†</td>
</tr>
<tr>
<td>Relative left ventricular wall thickness</td>
<td>2.5±0.3</td>
<td>2.5±0.5</td>
<td>2.4±0.4†</td>
<td>2.5±0.3</td>
<td>2.7±0.4**</td>
<td>2.7±0.5</td>
<td>2.9±0.5**</td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>158±25</td>
<td>209±38</td>
<td>260±38*</td>
<td>214±44</td>
<td>179±25*</td>
<td>203±57</td>
<td>161±38*</td>
</tr>
<tr>
<td>End diastolic volume (ml)</td>
<td>111±20</td>
<td>140±26</td>
<td>162±30*</td>
<td>149±39</td>
<td>139±35†</td>
<td>141±33</td>
<td>131±32**</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>79±16</td>
<td>100±19</td>
<td>115±20*</td>
<td>112±20</td>
<td>101±17†</td>
<td>99±21</td>
<td>94±17†</td>
</tr>
<tr>
<td>Cardiac index (L/min-m²)</td>
<td>3.7±0.4</td>
<td>5.1±0.5</td>
<td>5.4±0.6**</td>
<td>5.3±0.7</td>
<td>4.6±0.5*</td>
<td>4.8±0.5</td>
<td>4.4±0.5*</td>
</tr>
</tbody>
</table>

Mean values ±SD; * p<0.001; ** p<0.005; † Not significant
In all groups the initial values of stroke volume and cardiac index were greater than normal. During haemodialysis the stroke volume and cardiac index increased by 15.2 per cent and 6.5 per cent (p<0.01); CAPD and renal transplantation reduced stroke volume by 9.6 per cent and 4.9 per cent (NS), and cardiac index by 14.3 per cent and 7.8 per cent (p<0.01) respectively.

Discussion

The results of this study show that patients with end-stage chronic renal failure have evidence of left ventricular hypertrophy and dilatation. This is most probably due to the increased cardiac work imposed in chronic renal failure. While in patients undergoing regular haemodialysis the left ventricular mass, the end-diastolic dimension and volume and the cardiac index increase with time, CAPD and, especially, successful renal transplantation lead to an opposite effect by significantly reducing the aforementioned indices. This should be regarded as a beneficial effect of the last two procedures.

The increased workload faced by the heart in chronic renal failure is primarily caused by anaemia, hypertension and the presence of the arterio-venous fistula [1]. This may lead to left ventricular hypertrophy and cardiac failure [1,2]. It has also been suggested that ureaemia itself can cause myocardial dysfunction (uremic 'cardiomyopathy') possibly through a direct effect of some uremic toxins [1]. These changes are reversible, since it has been documented that left ventricular hypertrophy could regress after correction of left ventricular overload as has been shown after aortic valve replacement and in experimental hypertension [6]. Regression of left ventricular hypertrophy was also observed after effective parathyroidectomy in uraemic patients with secondary hyperparathyroidism [7].

The observed deterioration of left ventricular hypertrophy and dilatation following long-term haemodialysis has been previously reported [2,8]. The main factors contributing to left ventricular hypertrophy and dilatation are the persistence of chronic anaemia, the presence of the arterio-venous fistula and the hypertension which is common in patients with end-stage chronic renal failure even on long-term haemodialysis [1]. Under the above circumstances which lead to volume and pressure overload the left ventricle reacts with hypertrophy and dilatation [2,8]. It was found that the behaviour of the heart during haemodialysis depends on the pre-dialysis status of left ventricular function. Patients with depressed left ventricular function show an improvement of left ventricular ejection fraction while patients without heart failure remain stable or even deteriorate [9]. There is still controversy regarding uraemic toxins and uraemic cardiomyopathy which may have a negative inotropic effect on left ventricular contractility contributing independently to left ventricular dilatation [1,2].

The improved left ventricular hypertrophy and function in patients on long-term CAPD deserves special comments. It is supported that the beneficial effect of CAPD is mainly due to a reduction in volume and pressure overload [3,10]. This is reflected by a significant reduction of LVIDd, left ventricular
mass and cardiac index. It is most probably effected by improvement of anae-
mia, better control of uraemia and hypertension, absence of the arterio-venous
fistula and avoidance of the rapid haemodynamic fluctuations common during
haemodialysis [3,10]. However further investigations are needed as far as the
role of CAPD in removing uraemic toxins is concerned. Reversibility of left
ventricular hypertrophy and dysfunction after successful renal transplantation
has been recently reported [4,8]. Our findings are similar to those described in
the literature and furthermore show that renal transplantation is superior to
CAPD regarding the improvement of left ventricular hypertrophy and dilatation.
This should be expected since after successful renal transplantation most of the
previously described contributing factors to left ventricular hypertrophy and
dysfunction are effectively corrected.

It is concluded that left ventricular hypertrophy and dilatation in patients
with end-stage chronic renal failure and stable function deteriorate with regular
haemodialysis, while they regress with CAPD and especially after successful
renal transplantation.

References

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S179

No discussion owing to technical difficulties with recording.