Ambulatory blood pressure monitoring in patients receiving long, slow home haemodialysis

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Abstract

Background. Good blood pressure (BP) control has been reported previously in haemodialysis (HD) patients receiving 8-h dialysis sessions. Home HD allows patients to dialyse for long periods, but there are few data on the BP control achieved by these patients. We studied BP control, using ambulatory blood pressure monitoring (ABPM), in our home-HD patients who were receiving long-hours dialysis.

Methods. Twenty-four patients aged 52.7 ± 11 years underwent ABPM. They had been on home HD for 52.9 ± 39 months and dialysed for 7.2 ± 1.1 h thrice weekly. Two patients were taking antihypertensive drugs. Historical data on BP and weight gains were obtained from the patients’ own records. Left ventricular (LV) mass was assessed by echocardiography and total body water (TBW) by bioelectrical impedance.

Results. The mean 24-h BP was 129 ± 17 mmHg (systolic) and 83 ± 14 mmHg (diastolic). The daytime BP was 131 ± 17 mmHg (systolic) and 84 ± 14 mmHg (diastolic), while the night-time BP was 126 ± 22 mmHg (systolic) and 81 ± 17 mmHg (diastolic). Six patients (25%) had a normal circadian BP rhythm, but the rest showed a subnormal fall or an increase in BP at night. Mean 24-h BP did not correlate significantly with time on dialysis, dialysis session length, Kt/V, haemoglobin, interdialytic weight gain, or TBW. Twenty-one patients (87%) had LV hypertrophy and 16 of these had diastolic dysfunction. LV mass index was inversely correlated with nocturnal BP fall (r = −0.54, P = 0.03). Non-dippers had been treated longer than dippers (29 vs 59.2 months, P = 0.03) but they were similar in respect to age, dialysis session length or Hb concentration.

Conclusions. Long, slow haemodialysis at home provides satisfactory daytime BP control in the majority of patients without the need for antihypertensive drugs but abnormal circadian BP rhythm and LV hypertrophy remain common.

Key words: ambulatory blood pressure monitoring; circadian rhythm of blood pressure; haemodialysis; hypertension; left ventricular hypertrophy

Introduction

Hypertension is a risk factor for cardiovascular complications in patients on long-term dialysis [1,2]. Previous studies, which have utilized ambulatory blood pressure monitoring (ABPM), have shown that hypertension is common in dialysis patients, even when antihypertensive drugs are used [3–5]. The notable exceptions are the two studies from centres that use 8-h haemodialysis (HD) sessions [6,7]. Both reported excellent blood pressure control from dialysis alone. Charra and co-workers [2] suggested that good BP control was the principal reason for the low rate of cardiovascular complications in their patients.

Our nephrology unit, established in 1967, has provided only long-term HD at home. Patients dialyse for 6–10 h thrice weekly and less than 5% take antihypertensive medications. However, our HD survival (median 7.75 years) is inferior to that reported from Tassin [2], which raised questions about the adequacy of our BP control. The aim of this study was to determine the level of BP control achieved by long, slow haemodialysis performed at home. In addition, we sought to study the relationship between BP, hydration status, and cardiac morphology in HD patients who do not use drugs to control BP.

Subjects and methods

All patients who had been on home HD for more than 12 months agreed to undergo ambulatory blood pressure monitoring (ABPM). Those unable to have ABPM because of obesity (n = 2) or an absent brachial artery pulse (n = 1), and those whose ABPM recording was unsatisfactory (n = 4) were excluded. All patients performed thrice-weekly dialysis at home with a 0.8 m² cuprophane membrane (RE08H, Kawasumi, Japan), acetate buffer (sodium 138 mmol/l) and a 200 ml/min blood pump speed. The dialysis machines (Cobe Centry C2) had run logs that were used to check compliance with prescribed dialysis times. All patients kept a record of BP (self-taken with mercury sphygmomanometer) and weight pre- and post-dialysis.

ABPM was performed for 24 h prior to an end-of-week dialysis session on a non-working day using a Profilomat® monitor (Disetronic, Switzerland). This device has been validated and meets the British Hypertension Society criteria [8]. Calibration of the device was tested before use. The cuff
was placed on the non-fistula arm and its accuracy was tested against a mercury sphygmomanometer to ensure <5 mmHg discrepancy. BP was measured every 15 min from 0800–2200 hours and every 30 min from 2200–0800 hours. The patients kept a sleep and activity diary for the 24-h period. The mean 24-h, daytime and night-time BPs were recorded. Hypertension was defined as mean daytime BP >146/91 mmHg and/or night-time BP >127/79 mmHg [9]. BP load was defined as the percentage of readings >140/90 mmHg by day and/or >120/80 mmHg at night [10]. Nocturnal BP decrease (dipper status) was determined by comparing the mean arterial BP (MPB) of the night and day periods. Dippers exhibited a >10% fall in nocturnal MBP whereas non-dippers showed <10% reduction or an increase in MBP. The pre- and post-dialysis BPs taken by the patients over the previous month were recorded from their logs, and the predialysis BP recorded at clinic over the previous 2 years was retrieved from the clinical database (Protron® Clinical Computing Ltd, London).

Trans-thoracic m-mode echocardiograms (Hewlett Packard, Sonos 5500) were performed 12–16 h post-dialysis by a single operator who was blinded to the ABPM result. Left ventricular (LV) wall measurements were used to calculate LV mass, corrected for body-surface area, according to the formula of Devereux [11]. LV hypertrophy was defined by a single frequency bioelectrical impedance (SEAC (25%) (Proton Corporation, California) were measured on a pre- had been on dialysis for a shorter period than non-

Table 1. Results

<table>
<thead>
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<th>Systolic</th>
<th>Diastolic</th>
<th>MBP</th>
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<tbody>
<tr>
<td>24-h BP (mmHg)</td>
<td>129 ± 17 (103–160)</td>
<td>83 ± 14 (55–110)</td>
<td>99 ± 14 (70–120)</td>
</tr>
<tr>
<td>Daytime BP</td>
<td>131 ± 17 (105–169)</td>
<td>84 ± 14 (55–113)</td>
<td>100 ± 14 (72–124)</td>
</tr>
<tr>
<td>Night-time BP</td>
<td>126 ± 22 (100–151)</td>
<td>81 ± 17 (52–110)</td>
<td>98 ± 18 (68–122)</td>
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<td>% nocturnal dip</td>
<td>4 ± 6 (–15 to 15)</td>
<td>4 ± 5 (–7 to 12)</td>
<td>4 ± 5 (–13 to 14)</td>
</tr>
<tr>
<td>Patient BP, pre-dialysis</td>
<td>138 ± 34 (104–178)</td>
<td>83 ± 12 (62–104)</td>
<td>103 ± 14 (76–128)</td>
</tr>
<tr>
<td>Patient BP, post-dialysis</td>
<td>125 ± 20 (94–160)</td>
<td>73 ± 11 (51–87)</td>
<td>90 ± 13 (65–112)</td>
</tr>
<tr>
<td>Clinic BP, 2 years</td>
<td>144 ± 22 (110–166)</td>
<td>85 ± 11 (62–107)</td>
<td>104 ± 14 (78–119)</td>
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cystic disease (5), vasculitis (2), renovascular disease (2), reflux nephropathy (1), diabetic nephropathy (1) and unknown (2). The patients were aged 52.7 ± 12 (21–69) years and had been on home HD for 52.9 ± 39 (12–228) months. They dialysed for 7.2 ± 1.1 (6–9.5) h thrice weekly with a Kt/V of 1.2 ± 0.17 (1–1.6). Their weight gain over the studied interdialytic interval was 2.4 ± 0.9 (1–5) kg and their mean interdialytic weight gain over the previous month was 2.5 ± 0.8 (1.1–4.8) kg. The mean haemoglobin was 105 ± 22 (69–153) g/l. Ten patients were taking erythropoietin and two received antihypertensive therapy (Doxazosin in one patient, enalapril in another). Their percentage TBW ranged from 37 to 69%. TBW (by BIA) was 1.4 ± 2.5 (–3 to +8) litres above the TBW estimated using the Watson formula [15].

The BP results are shown in Table 1. Daytime mean BP results showed systolic hypertension in three (13%) patients and diastolic hypertension in four (17%). At night, systolic hypertension occurred in 8 (33%) patients and diastolic hypertension in 7 (29%). Mean 24-h BP correlated closely with both pre-dialysis (r = 0.89) and post dialysis (r = 0.79) BP from the patients’ records. Mean 24-h BP did not correlate with interdialytic weight gain, dialysis hours, Kt/V, Hb, or ‘hydration status’ (defined as TBW(BIA)−TBW(Watson formula)). A normal (>10%) fall in MBP at night occurred in six (25%) patients. These 6 patients (dippers) had less LV mass than non-dippers (NS), despite the fact that the mean 24-h BP of dippers was on average, 2.5/7.2 mmHg higher than non-dippers (NS). Age, dialysis hours, interdialytic weight gain and Hb concentration were similar in both groups (Table 2). Dippers had been on dialysis for a shorter period than non-dippers (29 vs 59.2 months, P = 0.03) although when one outlier, a non-dipper on dialysis >20 years, was excluded this difference was no longer significant. A scatter plot (not shown) revealed no significant relationship between dialysis duration and BP dipping (r = −0.3, P = 0.13).

Twenty-one patients (87%) had left ventricular hypertrophy (LVH) and 16 of these had diastolic dysfunction (mitral valve E/A ratio <1). There was a significant inverse relationship (r = −0.54, P = 0.03) between LVMI and the % fall in nocturnal MBP (Figure 1). Nocturnal dipping also predicted LVMI when considered with dialysis duration, mean 24-h and pre-dialysis BP using multiple regression analysis (r² =
Fig. 1. Relationship between LVMI (g/m²) and % change in nocturnal MBP. ((−)=fall, (+)=increase in MBP). A, patient taking enalapril; B, patient taking doxazosin.

0.3, \(P=0.04\)). Correlations between LVMI and other variables were not statistically significant. These included the duration of dialysis, mean 24-h BP and pulse pressure, the BP from patient or clinic records, dialysis hours, Kt/V, age, PTH, and haemoglobin concentration.

Discussion

This is the third report of ABPM in haemodialysis patients receiving long treatment times in a thrice-weekly schedule. Hypertension was more common in our patients than in the two previous studies [7,8]. Whether this is simply due to a different patient mix or to differences in treatment is unclear. Compared to patients treated in Tassin, our patients had slightly shorter HD sessions (7.2 vs 8 h) and lower Kt/V values (1.2 vs 1.7), possibly an effect of smaller (0.8 m²) dialysers and the calculation of Kt/V based on 30-min post-dialysis blood samples. However, neither session length nor Kt/V correlated with mean 24-h BP level and while the patients’ records for the previous month indicated that target weight was achieved with 84% of dialysis sessions, failure to identify changes in dry body weight and adjust targets accordingly may have played a role. These patients dialysed at home, often unsupervised, and lower target weights may have been possible had they been receiving long treatments in a hospital setting.

Compared to previous studies of ABPM in patients receiving 3–4 h HD sessions [3–5], however, the BP control of our patients was satisfactory. Their mean systolic BPs were within the recently published reference values, which predict the best prognosis in the general population [16] and were achieved without antihypertensive drugs. In the 1960s it was observed that most ESRF patients could have BP controlled by dialysis alone. The reports from Tassin, Manchester, and now our study show this still applies when long,
slow HD is used and that normotension can be maintained through the interdialytic period. However, despite good control of mean 24-h BP with this form of dialysis, normal circadian BP rhythm is seldom restored and LVH is common.

The causes of abnormal BP rhythm and its pathologic significance in patients on dialysis remain uncertain. Non-dipper status has been associated with LVH [17] and an increase in cardiovascular events [18] in essential hypertension. Goldsmith et al. [6] reported that diastolic BP fall during sleep was negatively correlated with ECG voltages. Our finding of an inverse relationship between LVMi and nocturnal BP dipping has not been reported previously for dialysis patients. Loss of the normal BP fall during sleep will increase an individual’s BP load, but the 24-h mean BP of our dippers tended to be higher than non-dippers. This suggests that altered BP rhythm may be an independent risk factor for LVH, or that LVH and BP rhythm disturbance share a common aetiology, e.g. altered sympathetic activity or levels of other vasoactive peptides.

As this is a relatively small cross-sectional study we cannot prove that relationships are causal. Nor can we rule out, despite the absence of statistically significant correlations, possible contributions from other factors to LV mass such as the duration of dialysis therapy. We can conclude from the multiple regression analysis, however, that non-dipping appears to be one contributing factor. The pathogenesis of LVH in dialysis patients is obviously multifactorial because LVH is still common in those with optimal BP control [19,20]. The relationship between BP variability, neurohormonal disturbances and LVH warrants further investigation in prospective studies, particularly since LVH is an important predictor of a worse outcome on dialysis.

In summary, most patients who receive long, slow dialysis at home are able to control BP without medication but abnormal diurnal BP variability and LVH remain common. Whether giving antihypertensive drugs to patients who dialyse in this way can restore diurnal BP rhythm and/or reduce the incidence of LVH, remains to be established.

Addendum

Since submission of our original manuscript, Covic et al. have reported an inverse association between LV mass on echocardiography and diurnal BP variability in long-hours haemodialysis patients and renal transplant recipients [Nephrology 1998; 4: 87–93]. Our results concur with their findings.

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References


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Editor’s note

Please see also the Personal Opinion by Scribner (pp. 2599–2601 in this issue).