Blood pressure control in dialysis patients: HD vs CAPD

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INTRODUCTION

Mortality in dialysis patients has been attributed mainly to cardiovascular disease (CVD) and there has been an effort to address this issue even before patients start dialysis[1,2]. At the stast of dialysis, 80-90% of the patients are hypertensive and the majority suffer from severe systemic consequences: left ventricular hypertrophy, arteriosclerosis and coronary, cerebral and peripheral vasculopathy. Hypertension has been considered to be one of the major risk factors for the unacceptable high cardiac mortality and morbidity among the renal patient population[3]. The National Kidney Foundation Task Force on CVD has recently labeled this problem to be of epidemic proportions[4].

CARDIOVASCULAR DISEASE

In many studies cardiovascular deaths were similar for CAPD and HD but in the USRDS 1995 report there were more deaths with CAPD at 1 year for myocardial infarction and «other cardiac» causes, except sudden death[5]. It was surprising since CAPD has some hemodynamic advantages over hemodialysis: no hypercirculation due to an AV fistula, constant acid-base balance, constant concentrations of sodium, potassium, calcium and other solutes and a continuous removal of waste products that are possible etiological factors in uremic cardiomyopathy. Furthermore, intra or interdialytic changes in cardiac filling and major fluctuations in blood pressure are absent in CAPD[6].

Among the risk factors for cardiovascular disease are uremic cardiomyopthy, with myocardial hypertrophy and intermyocardiocytic fibrosis, coronary artery disease, hypertension and dislipidemia.

RATIONALITY FOR BLOOD PRESSURE CONTROL IN CAPD PATIENTS

Blood pressure control on CAPD is simple due to continuous sustained ultrafiltration and sodium removal, which maintains patients at their dry body weight. The reduction in blood pressure is most marked during the initial weeks of therapy and additional decreases occur over the next few months. The blood pressure response to CAPD correlates well with the reduction in fluid body weight, emphasizing the importance of fluid volume in the pathogenesis of the hypertension[7].

During CAPD exchanges, net water as well as sodium is removed. A typical CAPD patient may lose about 1-1,5 L/day of ultrafiltrate with a sodium concentration of about 132 mEq/L. The total sodium loss can be readily calculated[8]. With low sodium diets, a CAPD patient may become sodium depleted. Multiple antihypertensive drugs may be discontinued.

LEFT VENTRICULAR HYPERTROPHY (LVH)

LVH has many possible causes in uremia: sustained high blood pressure, anemia, hyperparathyroidism, rennin system (local), endothelin, sympathetic overactivity, salt/ouabain-like factor, pulsatile *vs* steady LV workload[9]. Reduction of LVH with CAPD and not with HD has been related to the nocturnal blood pressure reduction seen only on CAPD and to lower norepinephrine levels on CAPD than HD[10].

CEREBROVASCULAR DISEASE (CVD)

On the basis of the USRDS data, Bloemberg y cols., found that patients on PD had a 20% higher probability of dying from stroke than their HD counterparts[11].

However, the 2001 USRDS Report revealed a simlar mortality rate between HD and CAPD/CCPD patients due to cerebrovascular disease: 13.7 *vs* 13.3, respectively (rates per 1,000 pt years at risk, period 1997-1999). Although in the 45-64 age group the rate for HD was 10.7 *vs* 11.7 in CAPD/CCPD and at the group 65 + it was 19.4 *vs* 23.4 respectively[12].

Maiorca y cols., also noted a higher percentage of deaths due to stroke in CAPD than in HD patients[13]. In their series 21 out of 297 CAPD patients (23/1,000

patient-years) and 6 out of 281 HD patients (7/1,000 patients-years) died of stroke, but there were more diabetics in the CAPD group.

In our own series we have found stroke to be the leading cause of death in our CAPD population[14].

CONTROL OF BLOOD PRESSURE

The 1987 Report of the NIH-CAPD registry revealed that 30% of 400 medicated patients were converted to normal within 1 year and others tended to take less medication[15]. In two other studies[16,17], CAPD appeared sto be as effective as HD, whereas in another one[18] there was better control of blood pressure with CAPD than with HD. Maiorca y cols., were not able, in randomly selected elderly patients to find differences between the two methods[19].

Velasquez y cols.[20], studied 21 patients, 10 of them transferred from CAPD to HD and 11 from HD to CAPD and found a higher prevalence of hypertension with PD (71%) than with HD (43%). The authors attributed the difference to higher fluid expansion in PD.

Sezer y cols., followed 34 patients (mean age 43.5 ± 14.5 years) than had been on HD for 36.6 ± 24.7 months and had been transferred to CAPD and were followed for 19.8 ± 11.0 months[21]. Among other parameters, they have noted a decrease in mean systolic blood pressure (139.4 ± 22.8 mmHg to 114.4 ± 21.0 mmHg, p = 0.001) and diastolic blood pressure (85.7 ± 12.6 mmHg to 73.5 ± 17.6 mmHg, p = 0.002) after the switch to CAPD. Similarly, the number of patients on antihypertensive therapy decreased from 13 (38.2%) to 3 (8.8%, p = 0.02); systolic dysfunction from 18 (52.9%) to 5 (14.7%, p = 0.001); diastolic dysfunction from 24 (70.5%) to 11 (32.3%, p = 0.01) and left ventricular hypertrophy from 19 (55.8%) to 6 (17.6%, p = 0.001).

Although earlier studies have reported good control of blood pressure on CAPD patients, yet the prevalene of hypertension remains high[22,23]. Several recent studies have revealed that CAPD patients require more antihypertensive drugs than HD patients to maintain control of blood pressure, probably because of progressive loss of residual renal function[24,25]. Shoda y cols., evaluated blood pressure before and after introduction of CAPD and compared the office visit measurement with readings obtained during 24 hours ambulatory blood pressure analysis[26]. The study showed that mean daytime systolic and diastolic blood pressure were significantly reduced after the introduction of CAPD but these values were not significantly reduced during sleep. The authors pointed out that these results suggest that hypertension is not adequately controlled for almost half the day.

CONCLUSIONS

The prevalence of hypertension in PD patients is lower than in HD patients, at least in the first years of treatment, although not all investigators agree[20,27]. Achievement of dry weight is more sucessful in PD patients probably because of larger residual diuresis in PD patients or more favorable clearance of various humoral pressor factors. The more efficient rate of removal of sodium and water in PD than in HD may play also a role. The different prevalence of hypertension tends to disappear with longer duration of PD which could be due to loss of residual renal function or even ultrafiltration failure.

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