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Review Article

Hypertension in peritoneal dialysis patients: epidemiology, pathogenesis, and treatment

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Abstract

Hypertension is prevalent in an estimated 29% to 80% of patients treated with peritoneal dialysis (PD). Cardiovascular disease represents the most common cause of mortality in this population, and hypertension (HTN) plays an important role. Volume overload is prevalent in PD patients because of liberal intake of fluids and loss of residual renal function (RRF). Noncompliance with salt restriction causes weight gain and makes HTN more difficult to manage. Physiology of the peritoneal membrane and its transport characteristics governs the ultrafiltration rate and consequently both volume and HTN. Therapeutic options for blood pressure control are ultrafiltration through the osmotic or colloid osmotic effects of dialysis solutions, salt restriction, and the use of antihypertensive medications such as diuretics, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers. Loop diuretics are used to maintain urine output in nonoliguric patients. Doses may exceed 250 mg of furosemide; ototoxicity is not problematic if blood levels are monitored carefully. Preservation of RRF is important for maintaining volume control and, thereby, control of HTN. J Am Soc Hypertens 2011;5(3):128–136.

Keywords: Peritoneal dialysis; end-stage kidney disease; residual renal function; peritoneal dialysis solutions; left ventricular hypertrophy.

Introduction

Peritoneal dialysis (PD) is one of the alternative renal replacement methods that provides solute clearance and fluid removal in end-stage kidney disease (ESKD). First used in 1923 by Ganter,[1] 26,517 patients are currently on PD in the United States.[2] In 2004, 149,000 patients were undergoing PD therapy worldwide, representing 11% of the total dialysis population.[3] Hypertension (HTN) is prevalent in PD patients. Although patients are initially improved by PD, they later tend to deteriorate because of volume expansion, coinciding with further loss of residual renal function (RRF).

It is generally accepted that chronic fluid overload is the main cause of HTN in patients with ESKD.[4] A randomized controlled trial of 150 hypertensive hemodialysis patients was designed to evaluate changes in systolic interdialytic ambulatory blood pressure with ultrafiltration (UF). One hundred patients were assigned to UF, and 50 patients served as controls. There was a –6.9 mm Hg change in systolic blood pressure (SBP) and a –3.1 mm Hg change in diastolic blood pressure (DBP) after a postdialysis reduction weight of 0.9 kg at 4 weeks into the study. Furthermore, changes of –6.2 mm Hg less for SBP and –3.3 mm Hg more for DBP after 1 kg reduction occurred in 8 weeks.[4]

Cardiovascular disease is by far the most common cause of mortality in ESKD. There are also structural changes associated with cardiovascular disease already present in the incident dialysis population. In a study involving 47 PD patients, Günal et al[5] were able to attain normotension

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in 91% with volume control through UF with different osmotic agents in peritoneal solutions, and in 42% with salt restriction. Few detailed data exist on the fluid status of PD patients, or on the relationship between fluid status, BP, and structural cardiovascular abnormalities in this population. Various authors have suggested a theoretical preference for PD because of the continuous removal of fluids and the fact that residual diuresis is often better maintained.

Age is another factor in the relationship between HTN and PD, a relationship that may differ from that in the general population. Rohrscheib et al[6] evaluated hemodialysis patients from the National Health and Nutrition Examination Survey participants (n = 9242), addressing the differences in the relationship between age and BP measurements. In contrast to the increase in SBP with age in the general population, SBP was elevated in young hemodialysis patients and declined among the elderly. The inverted ‘‘U’’ shape relationship between DBP and age was absent. DBP was elevated among patients younger than 50 years and declined in the elderly. Mean arterial pressure (MAP) and pulse pressure (PP) also showed similar trends. The plausible explanation is that in dialysis patients, the increased central artery stiffness at any age and the acceleration of cardiovascular disease translates into increased peripheral vascular resistance and decreased vascular compliance in young patients. Additionally, decreased cardiac output and a similar decrease in vascular compliance were observed in the elderly. These results may or may not be applicable to the PD population.

One of the main clinical benefits of PD is the preservation of RRF. Reanalysis of the CANUSA study in 2001 showed that RRF, and not peritoneal small solute clearance, was associated with survival.[7] Preservation of RRF is considered a marker of survival in observational cohort studies. Preservation of RRF at the expense of fluid excess represents a problem with BP management. There is a trade-off in PD between striving for euvolemia and maintaining RRF; episodes of hypovolemia predict loss of RRF.[8]

Lang et al[9] matched 15 patients in PD and hemodialysis (HD) according to cause of renal failure and RRF. RRF declined 3.7 mL/min and 1.4 mL/min after 12 months in HD and PD patients, respectively. HD patients required more aggressive UF with 15.6 hypotensive episodes per patient per year versus 0.46 in PD. In a study by Liao et al,[10] 270 patients on PD were stratified by decline rate in RRF between fast, intermediate, and slow decline groups. After 45 months, those with fast RRF decline had both poor survival (18.5% deaths) and poor compliance with the dialysis prescription. The median rate of RRF decline was 0.885 mL/min/1.73 m^2.

In this article, we review the epidemiology and pathophysiology of HTN in the PD population and its influence on cardiovascular mortality and patient survival. We also discuss the use of PD as a technique to optimize BP control through RRF preservation and volume control.

Epidemiology of Hypertension in Peritoneal Dialysis

The prevalence of HTN in the PD population ranges from 29% to 88% (Figure 1). Cocchi et al[11] evaluated 540 subjects in 27 centers belonging to the Italian Co-operative Peritoneal Dialysis Study Group; 88.1% were hypertensive according to World Health Organization/International Society of Hypertension and the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) criteria. The peritoneal dialysis core indicator study funded by the Centers for Medicare and Medicaid Services reported in 1998 that 26% and 16% of PD patients had SBP values higher than 150 and DBP values higher than 90 mm Hg, respectively.[12] Seventy-three percent of patients with continuous ambulatory peritoneal dialysis (CAPD) had uncontrolled HTN according to daytime ambulatory blood pressure monitoring (ABPM). Both systolic and diastolic ambulatory blood pressures were strongly related to left ventricular hypertrophy (LVH). This holds true for ambulatory but not office BP. ABPM data correlated better with end-organ damage compared with office measurements. The mean BP of the 1219 CAPD patients was 130/80 mm Hg.[12]

In some studies, SBP but not DBP, is a strong predictor of mortality at initiation of PD. A retrospective cohort study by Udayaraj et al,[13] using the UK Renal Registry, evaluated 2770 incident PD patients (19.8% diabetic patients, 58% men, 89.4% Caucasian). In years 4 to 5 of RRT, significant differences in association were observed between baseline SBP and mortality. For a 10 mm Hg increase of SBP, there was a 7% increase in time-stratified Cox proportional hazard in patients with diabetes (confidence interval [CI] 0.97–1.19) and a 7% decrease for those without diabetes. After 6 years, a significant association was observed among SBP, MAP, and mortality. It is important to consider

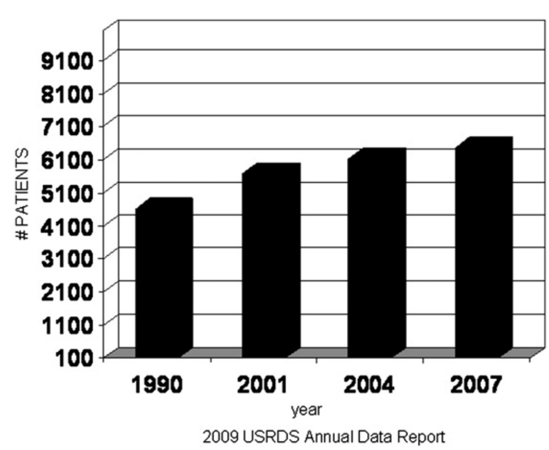

Figure 1. Prevalence of hypertension on peritoneal dialysis patients.