Intraperitoneal Infusion of Dialysate: A Possible Cause of Increased Plasma Atrial Natriuretic Peptide Levels

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Dear Sir,

Atrial natriuretic peptide (ANP) is a hormone with natriuretic, diuretic and vasodilative properties.

Many papers [1-3] have shown very high plasma ANP levels before dialysis because of plasma volume expansion and reduced renal ANP catabolism. Moreover, they also demonstrated that a reduction of atrial distension, that is the major determinant in ANP release, caused a significant decrease in plasma ANP levels after dialysis. On the other hand, there are few reports [4] on ANP plasma changes in continuous ambulatory peritoneal dialysis (CAPD) patients. In particular, up to now, no specific influence of peritoneal dialysate exchanges on plasma ANP levels has been described.

The aim of our study was to evaluate the possible acute ANP changes in CAPD patients caused by dialysate infusion.

13 uremic patients (11 men and 2 women; mean age 72 ± 7 years; mean duration of CAPD 28.4 ± 16.1 months) were studied. None of them had supraventricular tachyarrhythmia or congestive heart failure. The underlying diseases were chronic glomerulo-nephritis in 2 patients, interstitial nephritis in 2, polycystic kidney disease in 1, diabetic nephropathy in 1 and hypertensive nephropathy in 1; in 6 patients, nephropathy was unknown.

The dialytic schedule consisted of 4 exchanges per day in 8 patients and 3 exchanges in 5. Glucose dialysate concentration was

2.27 g% in all patients. 2 liters of dialysate were infused at each exchange.

ANP plasma levels were assayed by direct radioimmunoassay (EIKEN, Japan). Two samples for the determination of plasma ANP were drawn in the morning after an overnight fast in patients maintained in the supine position for at least 30 min: the first sample was collected 30 min after the first dialysate discharge, the second 30 min after infusion of 2 liters of dialysate.

Cardiac function was evaluated using an impedance monitor (NCCOM3 BOMED Medical Manufacturing Ltd., Irvine, Calif. USA). The reliability of this method is similar to that of other standard methods for calculating cardiac hemodynamics such as thermo-dilution [5, 6]. The following parameters were examined: cardiac output (CO-l/min), stroke volume (SV-ml), heart rate (HR-beats/min), ejection ventricular index (EVI-Ω/s) and ventricular ejection time (VET-s).

Changes in mean arterial blood pressure (MAP) were calculated by the formula:

diastolic pressure + $\frac{1}{2}$ differential pressure,

and total peripheral resistances (TPR) were calculated by the formula:

MAP (mm Hg)/CO (ml/s) $\times 1,332 \text{ dynes} \times \text{cm}^5 \times \text{s}$.

All recordings lasted 5 min and were registered at the two above-mentioned times.

Statistical analysis was performed using Student's t test for paired data.

Plasma ANP values were $130.69 \pm 70.08 \text{ pg/ml}$ after removal of dialysate and $163.61 \pm 80.48 \text{ pg/ml}$ after infusion ($p < 0.05$; table 1). No significant changes were found in CO, SV, HR, EVI, VET, MAP and TPR (table 1).

Comparing a group of hemodialysis patients with a group of CAPD patients, Ando et al. [4] found very high levels of plasma ANP in hemodialysis patients before dialysis. After dialysis, ANP levels fell to values very similar to those in CAPD. The decrease in ANP levels after dialysis could be related to the reduction in plasma volume caused by ultra-filtration. The same authors considered that the lower volume expansion can account for the lower increase in plasma ANP levels in CAPD patients.

In these patients, Swartz et al. [7] and Acquatella et al. [8] found an increase in inferior vena-cava pressure caused by the compression of dialysate, which might be responsible for lesser atrial distension and reduction in ANP release by reducing cardiac preload. As a consequence, ANP levels should be lower after dialysate infusion in comparison to those 30 min after discharge. On the other hand, Kong et al. [9] reported no significant changes in cardiac hemodynamics after instillation of 2 liters of dialysate. Accordingly, in our study using impedance cardiography, we did not find any adverse effects on cardiac function.

Therefore, no variation might be expected in plasma ANP levels before and after the infusion of dialysate. Rather surprisingly, we found a statistically significant increase in ANP values after dialysate infusion.

The clinical data of CO, SV, HR, EVI, VET, MAP and TPR were unchanged. On the basis of our results, hemodynamic factors as a cause of the reported early increase in ANP could be excluded.

Further studies are needed to clarify causes and clinical importance of the early increase in plasma ANP after dialysate loading.

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