A Hemodialysis Patient with Heart Failure and Severe Overhydration but Near-Normal Plasma Levels of ANP

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Dear Sir,

Atrial natriuretic peptide (ANP) is released from the myocardial cells in response to increased atrial pressure [1–3]. Higher-than-normal plasma levels are seen in conditions of water and sodium retention, e.g. heart failure and severe renal failure [4–9]. They are especially high in patients with both cardiac and renal impairment [9]. The release stimulus is considered to be atrial distention or atrial-wall stress [1–3, 10]. Increased plasma levels of ANP in hemodialysis (HD) patients are regularly seen during volume overload and promptly return towards normal with adequate ultrafiltration [9, 11, 12]. We report here on a HD patient with overhydration and severe heart failure but near normal plasma levels of ANP.

The patient was a 59-year-old man with chronic renal failure. HD was started, but the patient was never well on dialysis treatment. He could never comply with fluid restriction and suffered from severe overhydration. During the hemodialysis sessions, adequate ultrafiltration could not be performed, because of severe hypotension episodes, malaise and muscle cramps. The patient developed severe fluid overload with heart failure, incipient pulmonary oedema and peripheral oedema. Chest X-ray revealed a greatly enlarged heart (1,000 ml/m² body surface) and incipient pulmonary oedema. Blood was drawn before dialysis for the determination of ANP. The method used was competitive radioimmunoassay after extraction using SepPac. Predialysis plasma levels of ANP were 42 pmol/l, and in the following week, 40 pmol/l. These ANP levels are lower than the mean predialysis and even postdialysis values in our HD population. In an earlier study of 20 HD patients, we found

ANP levels before dialysis of 110.5 ± 13.2 (mean $\pm$ SE) pmol/l and after dialysis 75.4 ± 10.4 pmol/l (range 28–640 and 20–264, respectively) [13]. Normal value for healthy controls in our laboratory is 20 ± 5 pmol/l. Shortly after this investigation, the patient died from pulmonary embolism. At autopsy, multiple pseudomyxomatous thrombi were found in the right atrium of the greatly enlarged heart. There was also severe pulmonary stasis. Evidently distention of the atrial wall had not taken place due to the atrial thrombi.

The patient illustrates that, in accordance with experimental work, distention or increased stress of the atrial wall is a necessity for increased plasma levels of ANP also in clinical situations. In heart disorders, interfering with cardiac distention secondary to fluid overload, plasma levels of

ANP may be unreliable as markers of heart failure or volume overload. In such cases, careful ultrasound examination of the heart may give clues to the underlying pathological process.

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