

COMMENTARY

Congested states: is there a price for confounding cardiac and renal failure?

Es J. WILL

Department of Renal Medicine, St James's University Hospital, Leeds LS9 7TF, UK. Fax: (+44) 113 244 0499

Keywords: *cardiac failure, congested states, renal failure*

Cardiac failure is congestive by virtue of sodium retention. A secure diagnosis and the assessment of severity must therefore take other causes of positive sodium balance into account. In elderly patients and those with diabetes the contribution of renal impairment is particularly important because primary cardiac and primary renal disease often co-exist and interact [1]. The syndrome of congestive cardiac failure (CCF) is itself associated with a reduction of glomerular filtration rate, but this is usually modest [2–4]. Likewise, some primary renal disorders are easily misdiagnosed as CCF—for example, atherosclerotic renal artery stenosis—but the relevance of vascular or parenchymal renal disease in true combination with cardiac disorder is less clear-cut in clinical practice [5]. Even moderate renal impairment interferes with treatment of the heart by reducing the response to diuretics and creating difficulty in the use of angiotensin-converting enzyme (ACE) inhibitors because of hyperkalaemia (for example [6, 7]). It hazards a *congested state*, in which the renal component directly confounds the assessment of residual cardiac capacity and prevents the accurate prediction of outcome. Only the relief of 'fluid overload' by dialysis or filtration can reveal the degree of specifically cardiac failure, compared with the secondary consequences of sodium retention, and in fact the extent of cardiac recovery is often surprisingly good [8].

Distinguishing between cardiac and renal components

Unfortunately, clinical symptoms and signs cannot be expected to allow discrimination of cardiac and renal components, particularly when both are present. Pulse volume, blood pressure and Valsalva manoeuvre may be useful but are not definitive. Radiological techniques are also of limited value. Pulmonary oedema from cardiac or renal causes is essentially indistinguishable [9]. Routine echocardiography is better at delineating valvular disorders than assessing the failing heart, especially

when the ejection fraction is well maintained in 'diastolic failure' [10, 11]. At ward level, it is often relatively untrained junior staff who attempt a reconciliation of clinical and echocardiographic findings. More sophisticated techniques may not be pursued in very elderly patients or those with much co-morbidity.

The confusion is not helped by widespread misunderstanding of the extent to which serum creatinine reflects glomerular filtration rate in old age. The degree of renal failure is often seriously underestimated from a creatinine level which is only modestly raised. This is even more misleading in malnourished individuals [12]. The terminology of heart failure is also unhelpful, since refractoriness to diuretics and ACE inhibition in the presence of renal impairment need not imply the less commonly diagnosed, and frequently implicit, 'end-stage heart'.

Dialysis for older patients

These problems would be less important if patients with a combined disorder were commonly sent for a trial of dialysis, but there is a substantial shortfall in the numbers of elderly and diabetic patients referred for renal replacement therapy in the UK [13]. The few renal units are geographically, and philosophically, 'out of the way' for most medical departments [14]. It seems likely that the largely implicit (mis)diagnosis of 'end-stage heart disease' is one of the factors that allow physicians to rationalize the decision not to refer for renal replacement therapy in these populations. This would go some way to explain the demographic picture and has the advantage of being based on very understandable difficulties of differential diagnosis.

Several systematic improvements could be made to avoid these problems. Laboratory reporting of calculated glomerular filtration rate as well as serum creatinine would improve the ascertainment of renal failure. Wider awareness of renal arterial disease should reduce adverse reactions to ACE inhibition and allow a greater frequency of interventional treatments. Formal

training of physicians in the interpretation of routine echocardiogram reports could increase awareness of the limitations of the technique. A terminology for congested states that included the description of combined primary cardiac and primary renal disease would fix the issue on the medical map. Such steps would go a long way to correct the 'cardio-renal' clinical pitfall to which clinicians are otherwise exposed and could start to correct the exclusion of treatable elderly patients from dialysis programmes.

References

1. Packer M, Meller J, Medina N, Gorlin R, Herman MV. Hemodynamic evaluation of hydralazine dosage in refractory heart failure. *Clin Pharmacol Ther* 1980; 27: 337-46.
2. Fliser D, Franek E, Joest M, Block S, Haas M, Ritz E. Decrease of renal function in the elderly—effects of hypertension and cardiac disease. *Nephrol Dialysis Transplantation* 1996; 11: A150.
3. Anand IS, Ferrari R, Kalra GS, Wahi PL, Poole-Wilson PA, Harris PC. Edema of cardiac origin. Studies of body water and sodium, renal function, hemodynamic indexes, and plasma hormones in untreated congestive cardiac failure. *Circulation* 1989; 80: 299-305.
4. Dzau VJ. Renal and circulatory mechanisms in congestive heart failure. *Kidney Int* 1987; 31: 1402-15.
5. Missouri CG, Buckenham T, Vallance PJ, MacGregor GA. Renal artery stenosis masquerading as congestive heart failure. *Lancet* 1993; 341: 1521-2.
6. Packer M, Lee WH, Medina N, Yushak M. Influence of renal function on the hemodynamic and clinical responses to long-term captopril therapy in severe chronic heart failure. *Ann Intern Med* 1986; 104: 147-54.
7. McLay JS, McMurray JJ, Bridges A, Struthers AD. Acute effects of captopril on the renal actions of furosemide in patients with chronic heart failure. *Am Heart J* 1993; 126: 879-86.
8. Konig PS, Lhotta K, Kronenberg F, Joannidis M, Herold M. CAPD: a successful treatment in patients suffering from therapy-resistant congestive heart failure. *Adv Peritoneal Dialysis* 1991; 7: 97-101.
9. Rocker GM, Rose DH, Manhire AR, Pearson D, Shale DJ. The radiographic differentiation of pulmonary oedema. *Br J Radiol* 1989; 62: 582-6.
10. Franciosa JA, Park M, Levine TB. Lack of correlation between exercise capacity and indexes of resting left ventricular performance in heart failure. *Am J Cardiol* 1981; 47: 33-9.
11. Brutsaert DL, Sys SU, Gillebert TC. Diastolic failure: pathophysiology and therapeutic implications. *J Am Coll Cardiol* 1993; 22: 318-25.
12. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976; 16: 31-41.
13. Health Care Strategy Unit, Department of Health. Review of Renal Services, 1994.
14. Boyle PJ, Kudlac H, Williams AJ. Geographical variation in the referral of patients with chronic renal failure for renal replacement therapy. *Quart J Med* 1996; 89: 151-7.