

Teaching Point

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A hopeless case?

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Introduction

End-stage renal failure in an octogenarian with advanced congestive heart failure is a daunting challenge for the physician. Many of these patients cannot tolerate haemodialysis, due to severely impaired left-ventricular function, valvular disease or both. In such patients, peritoneal dialysis may be the only feasible option but home therapy is often impossible, due to age, comorbidity and lack of a suitable caregiver. In-centre intermittent peritoneal dialysis (IPD), which must often be performed for as many as 30 h/week, offers survival, at the expense of severely compromised quality of life. We present a seemingly hopeless case of end-stage renal failure in an 81-year-old patient with advanced congestive heart failure.

Case

An 81-year-old female patient was admitted in December 2005, with recurrent syncope and dyspnoea. She had a history of multiple hospital admissions because of congestive heart failure due to advanced aortic stenosis; comorbidities included hypertension, mild dementia and osteoarthritis of the knees. On examination, the patient appeared chronically and acutely ill with cyanosis and severe orthopnoea. Blood pressure was 105/70, pulse 105 beats/min, respiratory rate was 40/min and the patient was not febrile. The jugular venous pulse was elevated and there were coarse crackles over both lungs. The apical beat was irregular and palpable in the anterior axillary line and

there was a 4/6 crescendo–decrescendo systolic murmur at the second right interspace. Another soft pan-systolic murmur was audible over axilla and there was a third heart sound. Serum creatinine was 174 $\mu\text{mol/l}$ and urea 30.4 mmol/l; cystatin C was 6 mg/l and a previous serum creatinine was unavailable. An electrocardiogram confirmed atrial fibrillation. Echocardiography showed severe aortic stenosis, moderate aortic incompetence, severe mitral stenosis and moderate mitral incompetence, as well as impaired left-ventricular function, severe tricuspid regurgitation and pulmonary hypertension. Intravenous diuretics, digoxin and oxygen were begun while the patient remained on a peripheral ward and without inotropic support. The serum creatinine rose to 368 $\mu\text{mol/l}$, urea was now 38 mmol/l and the creatinine clearance was 12 ml/min. A femoral dialysis catheter was placed and haemodialysis was begun, which led to some improvement of the dyspnoea, although clinical examination and chest X-ray still showed marked pulmonary congestion (Figure 1). The haemodialysis sessions were complicated by hypotension, syncope and nausea. Cardiac catheterization confirmed multi-valvular disease with severe aortic stenosis (opening area 0.7 cm²) and severe mitral stenosis in the lead. Severe stenosis of the right and left anterior descending coronary arteries was also detected. The cardiac surgeons felt that double-valve replacement and bypass surgery was inappropriate; the forced expiratory volume was 1.15 l/s. A peritoneal dialysis catheter was placed and the patient was referred for in-centre IPD while she was on home oxygen, torasemide 50 mg/day and digoxin.

On the first day of in-centre intermittent peritoneal dialysis on 1 February 2006, the patient was bedridden, confused and incontinent. A urinary catheter was in place. Continuous oxygen was administered at 8 l/min but there was severe dyspnoea at rest and orthopnoea. A Karnovsky index of 25% was estimated. The medical team felt that death due to intractable congestive heart failure was imminent and resuscitation was believed to be futile in the event of any further deterioration. Laboratory studies showed no signs of

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Fig. 1. Chest X-ray after 2 weeks of haemodialysis; note cardiomegaly, pulmonary vascular engorgement and interstitial changes in keeping with chronic congestive heart failure as well as a jugular dialysis catheter.

infection. Peritoneal dialysis was begun with 15l 2.27% glucose. Spironolactone was begun because of hypokalaemia. A marked improvement was noted during the following weeks. Oxygen therapy was withdrawn in mid-February. Another assessment in April showed a patient in no distress and without any dyspnoea at rest. Remarkably, the patient was also partly orientated, cooperative and without incontinence. A urinary catheter was still in place and the urine output was estimated at 1.5l/day. The serum creatinine was 1.3 mg/dl. A nurse suggested that residual renal function might be sufficient to stop dialysis but the physician was reluctant and feared the risk of deterioration. Dialysis was stopped in April 2006, and the patient was seen in weekly intervals as an out-patient. During the following weeks, the patient improved even more. The general practitioner now saw the patient on a weekly basis and performed exit site care. When last seen on 30 November 2006, the patient was in good condition (Figure 2), no longer bed-ridden, partly orientated and without dyspnoea. A Karnovsky index of 55% was estimated. All attempts to measure the creatinine clearance failed but serum creatinine was 1.13 mg/dl and measurement of cystatin C indicated a glomerular filtration rate (GFR) of 27 ml/min. There have been no episodes of exit site infection and not a single episode of syncope or hospital admission while the patient was on aspirin, torasemide, spironolactone, digoxin and low-dose metoprolol. Removal of the peritoneal dialysis catheter was contemplated but postponed.

Discussion

The case presented here provides three different teaching points. First, it underscores the efficacy of peritoneal dialysis in patients with concomitant severe congestive heart failure. Patients with severe aortic stenosis have frequent and severe episodes of

cardiovascular collapse during the haemodialysis sessions, as illustrated by the present case. An abnormal Bezold–Jarisch reflex [1], i.e. hypotension and bradycardia mediated by left-ventricular baro-receptors, has been postulated as a mechanism for collapse and sudden death in aortic stenosis. During haemodialysis, this mechanism as well as exercise-induced vasodilatation in the presence of a fixed cardiac output may cause syncope. Our patient had intractable congestive heart failure due to multi-valvular and coronary heart disease. Haemodialysis ameliorated the dyspnoea at the expense of recurrent hypotension and syncope. It has been suggested that peritoneal dialysis is beneficial in such patients [2,3] although the need for randomized trials has been emphasized [4,5]. Some have even recommended peritoneal dialysis for patients with intractable heart failure and normal renal function [6,7].

Second, it is useful to remember the role of the kidney in congestive heart failure [8]. In retrospect, functional renal failure played a role in our patient, since renal function recovered and dialysis could be stopped. Our patient is, therefore, not quite comparable with other patients with true end-stage failure and congestive heart failure [9]. In normal individuals, low cardiac output stimulates the renin–angiotensin–aldosterone axis, leading to sodium and water retention in the distal tubule and collecting duct. Retention of sodium and water leads to enhanced venous preload and improved cardiac contractility by the Frank–Starling mechanism [10]. In the failing heart, left ventricular performance does not improve and then deteriorates with increasing filling pressures. Non-osmotic stimulation of the vasopressin system provides more water retention, while activation of the sympathetic nervous system increases peripheral vascular resistance and after-load. Treatment with diuretics improves the situation in that a decrease in filling pressures improves left-ventricular performance. Further interventions may change the individual Frank–Starling curve and lead to re-compensation. A severe drop in cardiac output also prompts renal vasoconstriction and a decline of the glomerular filtration rate. Patients who already have markedly impaired renal function may end up in a situation where diuretics do not provide sufficient excretion of sodium and water. Our patient was in such a vicious circle, from which only peritoneal dialysis could provide escape.

Finally, the case presented here must prompt some thoughts on the ethical aspect of the situation, not least because the number of octogenarians with renal failure is rising. From a medical point of view, starting peritoneal dialysis in a very elderly patient with end-stage renal failure and congestive heart failure may be a straightforward decision. In the real world, home therapy is often impossible due to age, co-morbidity and the absence of a suitable caregiver. In this scenario, in-centre IPD offers the only chance of survival. An average patient may spend the equivalent of a full-time job on or with dialysis and still fail recent



Fig. 2. The patient during her appointment in our outpatient department on 30 November 2006.

standards of weekly Kt/V [11]. Hence in-centre IPD is regarded as less than ideal by many nephrologists and some feel it should not be offered at all. We, too, were very sceptical as to whether dialysis was appropriate in this bedridden 81-year-old lady with seemingly intractable congestive heart failure. Attempts to involve the family led to nothing and the lack of an advanced directive prompted us to embark on a trial of dialysis. We were surprised to see the patient, in whom we had contemplated withdrawal of dialysis, improve so remarkably. This patient taught us to be careful in judging treatment as futile. Ethicists have emphasized that the term ‘futility’ is indeed misleading [12]. Along the same line, Andrews [13] described a case that taught him to be more careful in a seemingly hopeless situation. He concludes that we should not ‘play God’. Another dangerous train of thought is to decline treatment on the basis that quality of life will be poor. Perceptions of life and its quality differ remarkably [14]. The quality of life associated with centre-based IPD may seem unacceptable to a healthy physician but perfectly acceptable to a chronically ill patient with no alternative. A trial of dialysis may enable the patient to decide whether the life on IPD is acceptable or not.

Teaching points

- (i) End-stage renal failure in a very elderly patient with advanced congestive heart failure is a complex medical situation; many of these patients cannot tolerate haemodialysis due to valvular disease or impaired left-ventricular function. Patients with severe aortic stenosis are particularly affected.
- (ii) Low cardiac output stimulates neuro-humoral mechanisms, leading to inappropriate retention of sodium and water. In the failing heart, volume expansion leads to increasing preload but worsening left-ventricular performance. Impaired renal function may preclude removal of sodium and water with diuretics. In such patients, peritoneal dialysis may provide unique haemodynamic stability and slow removal of sodium and water.
- (iii) The decision not to initiate or to withdraw dialysis in the face of severe comorbidity and advanced age is a complex ethical dilemma. Our ability to delineate a prognosis is fraught with uncertainties and a seemingly hopeless case may recover. All we can do is offer information, advice and an opinion.
- (iv) We should be particularly reluctant to judge a prognosticated quality of life as unacceptable. A trial of dialysis may be a reasonable option and the patient may judge the quality of life as acceptable or not.

Conflict of interest statement. None declared.

References

1. Mark AL. The Bezold-Jarisch reflex revisited: clinical implications of inhibitory reflexes originating in the heart. *J Am Coll Cardiol* 1983; 1: 90–102
2. Khalifeh N, Vychytil A, Horl WH. The role of peritoneal dialysis in the management of treatment-resistant congestive heart failure: a European perspective. *Kidney Int* [Suppl] 2006; S72–75
3. Gotloib L, Fudin R, Yakubovich M, Vienken J. Peritoneal dialysis in refractory end-stage congestive heart failure: a challenge facing a no-win situation. *Nephrol Dial Transplant* 2005; 20 [Suppl 7]: vii32–vii36
4. Mehrotra R, Khanna R. Peritoneal ultrafiltration for chronic congestive heart failure: rationale, evidence and future. *Cardiology* 2001; 96: 177–182
5. Cnossen N, Kooman JP, Konings CJ, van Dantzig JM, van der Sande FM, Leunissen K. Peritoneal dialysis in patients with congestive heart failure. *Nephrol Dial Transplant* 2006; 21 [Suppl 2]: ii63–ii66
6. Bertoli SV, Ciurlino D, Maccario M *et al.* Home peritoneal ultrafiltration in patients with severe congestive heart failure without end-stage renal disease. *Adv Perit Dial* 2005; 21: 123–127
7. Alkan T, Akcevin A, Turkoglu H *et al.* Postoperative Prophylactic Peritoneal Dialysis in Neonates and Infants After Complex Congenital Cardiac Surgery. *ASAIO J* 2006; 52: 693–697

8. Schrier RW. Body fluid volume regulation in health and disease: a unifying hypothesis. *Ann Intern Med* 1990; 113: 155–159
9. Segall L, Covic A. Haemodialysis or peritoneal dialysis—which is the best option for end-stage renal disease patients with congestive heart failure? *Nephrol Dial Transplant* 2006. Published online Nov 11
10. Colucci WS and Braunwald E. Pathophysiology of heart failure. In: Braunwald E, ed. *Heart Disease. A Textbook on Cardiovascular Medicine*. Saunders, Philadelphia: W.B., 1997: p. 394
11. Dombros N, Dratwa M, Feriani M *et al.* European best practice guidelines for peritoneal dialysis. 7 Adequacy of peritoneal dialysis. *Nephrol Dial Transplant* 2005; 20 [Suppl 9]: ix24–ix27
12. Weijer C, Elliott C. Pulling the plug on futility. *Brit Med J* 1995; 310: 683–684
13. Andrews PA. A patient who made me think: We should not play God. *Brit Med J* 2003; 326: 634
14. Thomasma DC. Quality-of-life judgments, treatment decisions, and medical ethics. *Clin Geriatr Med* 1986; 2: 17–27

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