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**Dialysis: when to start or when to stop?**

Sir,

There have been several recent discussions around the timing of initiation of dialysis [1,2]. There remains little scientific consensus on when to start and on which clinical and biochemical parameters we base this decision. In the broadest sense, dialysis should serve to decrease morbidity and/or mortality whilst maintaining or improving quality of life. It is almost assumed that renal replacement therapy will offer this to all. However, an incident dialysis population is heterogeneous and failure to take this into account leads to non-individualized generic care. We must therefore recognize that the optimal time to start dialysis may differ for varied patient sub-groups. Additionally, in those individuals who fail to thrive on dialysis, then withdrawal of therapy has to be considered. This group may further provide insight into those individuals for whom dialysis is altogether inappropriate. These questions are perhaps posed most often when we consider an elderly co-morbid patient with end-stage renal failure (ESRF). Following the introduction of dialysis it has been demonstrated that age correlates with symptom burden on renal replacement therapy [3]. UK Registry data reports a 50% mortality at 1 year in incident dialysis patients over the age of 85 years, with the greatest attrition rate being in the first 3 months [4]. This early mortality may negate any subsequent benefits from an early start. Furthermore, Williams et al. [5] examined 24 consecutive cases in which dialysis was felt to be inappropriate. It was found that even when a conservative approach is taken functional status could be maintained until death is imminent. The following short cases highlight the points that an ‘early start’ may not lead to maintenance, but to deterioration in quality of life. Also, that dialysis can be withdrawn in some with the realistic expectation of an improvement in symptoms and with the preservation of independence making it a viable therapeutic option.

**Case 1.** An 83-year-old female with ESRF (calculated GFR of 7 ml/min) secondary to renal limited vasculitis commenced haemodialysis (HD) via a native fistula. She achieved a urea reduction ratio (URR) in excess of 65%. Although no haemodynamic compromise occurred, HD left her severely fatigued. Consequently she spent the inter-dialytic interval hospitalized and nursing dependent. Following discussions with the patient, HD was discontinued 5 months after initiation. All other medical therapies were continued. She remains at home 1 year after discontinuation.

**Case 2.** An 88-year-old male with ESRF (calculated GFR of 13 ml/min) secondary to diabetic nephropathy commenced HD via a native fistula. Despite several modifications to his HD prescription he suffered recurrent haemodynamic collapse and paroxysms of atrial fibrillation. Following discussion, treatment was withdrawn 10 months after initiation. With adherence to fluid and dietary restrictions he has required no hospitalizations in the following 7 months.

**Case 3.** A 78-year-old female with severe cardiac compromise and ESRF (calculated GFR of 8 ml/min) secondary to diabetic nephropathy commenced HD via a cuffed catheter. She achieved a URR of >65%. Again, HD left her severely fatigued and following 2 months of hospitalization she discontinued dialysis. She required no further hospital admissions before her death 6 months after discontinuation of HD.

Treatment with renal replacement therapy is no longer restricted to certain populations. We may be guilty of extrapolating benefit into individuals where this may not be the case. Indeed it is conceivable that in some we may cause harm. Any further work into the timing of initiation of dialysis should take into account the heterogeneous nature of the ESRF population and should be extended to cover the other issues raised here, as they are relevant to daily clinical practice.

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**Severe ascites following renal transplant biopsy caused by a rupture of a subcapsular lymphocele: treated successfully by retroperitonealization**

Sir,

We would like to share our experience of a patient who developed subcapsular lymphocele post cadaver transplant from a paediatric patient. Lymphoceles complicate 18% of renal transplants [1]. They usually occur in the first 6 months following transplant [2]. Lymphoceles are usually diagnosed because of pain over the transplanted kidney, or are found incidentally during investigation of renal failure. They may cause ureteric obstruction [3]. Many treatment regimes have
been reported including puncture drainage, povidone iodine, ethanol or tetracycline sclerotherapy and internal drainage by surgical or laparoscopic marsupalization. Many times the patient may end up finally losing the transplanted kidney.

A 29-year-old female patient underwent cadaveric transplant from a paediatric donor age 6 years in October 2001 for end-stage renal failure due to hypertensive renal disease. The procedure was uneventful and the kidney functioned well (baseline serum creatinine of 90 μmol/l at 2 weeks). The maintenance immunosuppression was tacrolimus and prednisolone.

In August 2002 the patient presented to Accident and Emergency with abdominal pain and an increased serum creatinine (179 μmol/l) raising the suspicion of rejection. She had an ultrasound scan of the abdomen, which showed a large kidney with a small loculated perinephric fluid collection. A renal biopsy showed thrombotic microangiopathy, which was felt to be secondary to tacrolimus. Hence rapamycin was substituted for tacrolimus. Two weeks after the biopsy she was admitted again with grossly distended abdomen. Repeat ultrasound scan showed gross ascites with a normal transplanted kidney. Diagnostic paracentesis was performed and ~11 of fluid was drained. The ascitic fluid re-accumulated rapidly and repeated paracentesis of 6–8 l were performed over a 2-month period to relieve symptoms of severe abdominal distension. The ascitic fluid showed 1355 WBCs with 95% lymphocytes and TB. PCR (polymerase chain reaction) of the fluid was positive and hence she was started on anti-TB treatment. Whole body CT scanning and the gallium scanning revealed no abnormality other than the ascitic fluid. Lymphangiogram was performed and demonstrated that the lymph appeared to be from the transplanted kidney.

As the ascites persisted she required twice weekly paracentesis for symptoms of paracentesis secondary to distension. The patient underwent laparotomy. She was found to have an enlarged kidney with fluid seeping out through the opening in the capsule (Figure 1). On opening the capsule, fluid gushed out. It became apparent that the patient had a subcapsular lymphocele, which was draining into the peritoneum through a rent in the capsule probably caused at the time of the renal biopsy some months previously. Retroperitonealization of the capsular opening by sewing the ascending colon to the edge as well as continuous intra-abdominal and subcapsular drainage helped in decreasing the drainage over a period of 6 weeks. The patient has made an excellent recovery.

Our case of subcapsular lymphocele is the first of its kind to be reported. The delayed presentation is unusual and may have been precipitated by the renal biopsy. Initial treatment with drainage over a prolonged period of time was unsuccessful. The subcapsular lymphocele became evident because of the biopsy, which opened the window to the peritoneum. The technique of retroperitonealizing the lymphocele with colon sutured to the edge of the rent may have helped in absorption of the lymph through the retroperitoneum and thus gradually decreasing the ascites.

Rapamycin has been blamed for an apparent increased incidence of lymphocele in Germany where transplanted kidney of patients treated with rapamycin developed lymphoceles. The authors have been unable to explain these findings [4].

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Fig. 1. Rupture site of the subcapsular lymphocele.


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