Clin Kidney J (2012) 5: 168–169
doi: 10.1093/ckj/sfs011

CKJ

Special Article

Long-term haemodialysis survival

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Abstract
Haemodialysis (HD) treatment for end-stage renal disease bears a poor prognosis. We present a case of a patient who, apart from two transplant periods lasting 8 months in all, was treated with conventional in-centre HD three times a week and who survived for 41 years. Patients should be aware that there is no theoretical upper limit for patient survival on HD.

Keywords: haemodialysis; survival

Background

Haemodialysis (HD) treatment for end-stage renal disease (ESRD) bears a poor prognosis. There is much confusion [1] in the public perception of longevity on HD, with many expressing surprise that survival beyond 20 years is possible. However, long-term survival does occur and several patients have been described with survival periods >35 years. We present a case of a patient treated with conventional in-centre HD three times a week who survived for over 40 years.

Case history

The patient was a general practitioner born in 1943. In 1967, he developed haematuria and proteinuria. A renal biopsy in July 1970 revealed proliferative glomerulonephritis. Despite treatment with cyclophosphamide and steroids, his renal function deteriorated and he started HD in September 1970 at the age of 27 years, using a left forearm arteriovenous fistula. This functioned for 26 years, requiring only one surgical revision after 25 years. After this, he used a permanent jugular catheter for access (Quinton Permcath; Covidien, Mansfield, MA). This was renewed in 2005 after 9 years of use. His in-centre dialysis regime throughout was 4–5 h three times a week.

He received three cadaver renal transplantations. The first, in 1972, was rejected after 6 months. At this time, bilateral nephrectomy was performed due to surgical problems. The second, in 1975, functioned for only 2 months before vascular rejection. A third transplant in 1984 never functioned, after which he was removed from the transplant list.

Alfacalcidol treatment for hyperparathyroidism was initiated in 1982 and bicarbonate dialysis was introduced in 1983. Despite treatment, his parathyroid hormone (PTH) rose to 3200 ng/L (normal 10–50), he developed hypercalcaemia [ionized calcium of 1.41 mmol/L (1.15–1.35)] and hyperphosphataemia [2.10 mmol/L (0.8–1.5)]. A subtotal parathyroidectomy was performed in 1984, after which his calcium normalized and PTH fell to 171 ng/L, rising slowly to 523 ng/L (14–72) at death. A coincidental thyroid cancer was discovered at operation, resulting in total thyroidectomy. The operation was not radical and he received postoperative radioactive iodine therapy. Erythropoietin (EPO) treatment started in 1989, after which his blood haemoglobin was 7.0–7.5 mmol/L. Prior to this, his spontaneous blood haemoglobin was 5.0–5.4 mmol/L and caused angina. He had received 132 blood transfusions, and EPO treatment was initially combined with repeated phlebotomy to treat complicating haemochromatosis (*p*-ferritin 5940 μg/L). In 1990, he developed bilateral hip arthrosis, which was ascribed to β2-microglobulin-associated amyloidosis. Right and left carpal tunnel syndrome operations were performed in 1993 and 1995, with good effect upon his symptoms. He suffered a minor posterior myocardial infarct in 1980. In 1992, he developed angina pectoris, and a coronary angioplasty operation was done in 1993, with excellent effect on his symptoms. He sold his practice at this time but continued to do part-time medical work for some years.

His joint and bone pains increased and opioid therapy was required. Dual energy X-ray absorptiometry in 1996 showed severe osteopoenia (bone mass 71% of normal, Z-score −4.4). A desferrioxamine test was diagnostic of aluminium osteomalacia [2], which was treated repeatedly with desferrioxamine infusions.

His health deteriorated after 1997 due to arteriosclerotic complications. A left crural amputation was performed in 1997. In 1999, he started to develop chronic ulcers on his fingers infected with *Staphylococcus aureus*, which eventually required the amputation of four fingers. Anterior ischaemic optic neuropathy developed in his right and left eye in 2001 and 2003, respectively, after which his eyesight was poor. An attempt to start treatment with high-flux dialysis failed due to psychological side effects, possibly due to increased elimination of opioid metabolites. At no time had the patient needed anti-hypertensive therapy, indeed, during the last 16 years of his life, he was hypotensive, his systolic blood pressure rarely exceeding 100 mmHg and often being as low as 70 mmHg. An echocardiography in 1998 was normal.

In July 2011, he was treated for a pseudomonas catheter sepsis (his second). He became comatose in September 2011, active treatment was stopped and he died after 41 years with ESRD, of which 40 years and 4 months had been treated with HD. He had been treated continuously with HD for 35 years and 10 months. Apart from admissions for transplantation, he was hospitalized on average for 5 days/year during the last 35 years of life.

His medical notes before 1976 have been discarded; after this, his medical notes filled 35 cm of shelf space.

Discussion

Long-term survival on HD has repeatedly been claimed for individual patients. On closer perusal, most of these patients have had long interspersed periods with a functioning transplant. Only two parameters are relevant to this discussion: the longest total period on HD and the longest continuous period on dialysis. This patient is a candidate for the first feat, but not the second.

The question of long-term survival in HD has received little scientific interest. Kurkus *et al.* [3] described two patients who had been dialysed for 35 years, interrupted by short transplant periods. Other series describe patients with shorter survival [4–8]. Factors associated with long survival are young age at start, normotension and absence of diabetes. Indeed, he developed non-symptomatic hypotension in later years, which is otherwise generally associated with a poor prognosis [9]. Psychological factors such as good compliance and a 'will to live' also seem to be important. This patient was typical in both these respects.

More information is available from the media. Ed Strudwick died recently after $39^{3}/_{4}$ years of dialysis [10]. Richard Faber [11], also from the USA, died of pneumonia in 2011 after 43 years and 2 months HD (personal communication), while Patricia Black from London was in her 39th year of dialysis in 2006 [12]. Brian Tocher [13] was also treated for 40 years, but it is unclear how much of this was transplant time. Many other patients have survived $>$30 years. It may be that these records will not be beaten in the future: while improvements in dialysis treatment (*vide infra*) may be expected to improve the prognosis of permanent dialysis, most of these patients had the modality forced upon them either due to repeated failed transplantations or multiple antibodies after blood transfusions. These problems, and therefore potential patients, are already much rarer than in the seventies.

Long-term HD survivors suffer from comorbidity, in particular arteriosclerosis, uraemic osteodystrophy and β2-microglobulin-associated amyloidosis with complicating spinal stenosis, carpal tunnel syndrome, bone cysts and arthropathy [14]. Arteriosclerotic eye disease with complicating blindness is a special problem affecting a fifth of patients [6]. Again, this patient was typical in most of these respects but avoided spinal stenosis and other complications, such as left ventricular hypertrophy and valvular calcification. His access record was impressive: one arteriovenous fistula lasting 26 years and two Quinton Permcath catheters, with two episodes of catheter sepsis, during the last 15 years.

The patient suffered from two historical diseases that can now be avoided, haemochromatosis and aluminium osteomalacia. An untransplantable young patient would today be encouraged to switch to daily home HD using high-flux filters; this would theoretically have reduced his risk of amyloidosis and may offer survival effects similar to transplantation [15]. However, accelerated arteriosclerosis [16] remains the major barrier to HD patients achieving the same life expectancy as healthy subjects.

Conflict of interest statement. None declared.

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Received for publication: 9.12.11; Accepted in revised form: 24.1.12