sieving coefficient of Cys C. Thus, in our view, the presented data do not support a tubular secretion of Cys C.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfl658

Advance Access publication 30 November 2006

Ulcereative tuberculin skin test in a dialysis patient

Sir,
A 53-year-old lady, hypertensive since 2003, was diagnosed with end-stage renal disease (ESRD) in January 2006 and initiated on continuous ambulatory peritoneal dialysis (CAPD). She has been on three exchanges per day with 2.5% dextrose solution. She used to achieve 1.5 L of ultrafiltration per day. Prior to this presentation, she had never suffered any mechanical or metabolic complications. Her peritoneal equilibration test revealed her to be a high average transporter. She presented with complaints of breathlessness, cough with no expectoration and anorexia. She had no peripheral oedema and fever. Her blood pressure was under control and echocardiography was normal. A chest radiograph revealed distention of pulmonary veins. Suspecting congestive heart failure, she was initiated on continuous cyclic peritoneal dialysis. There was an improvement in the breathlessness and cough, but her anorexia and fatigue had worsened. A tuberculin skin test (TST), was done with 5TU which ulcerated within 24 h, suggesting an infection due to Mycobacterium tuberculosis (Figure 1). The patient was treated with chemotherapy, and a Kappa light chain immunoperoxidase stain was positive for light chain deposition disease (LCDD), monoclonal immunoglobulin (Ig) light chain deposition usually involves the kidney [1]. End stage renal failure (ESRF) occurs in 70% of patients [2]. Outcomes of kidney transplantation are poor, due to recurrent allograft disease or progression of the underlying plasma cell dyscrasia [1,3]. Thus, renal transplant strategies must address the underlying disease. We describe the first reported case of LCDD treated with sequential autologous peripheral blood stem cell (PBSC) transplantation and kidney transplantation.

Sir,
In light chain deposition disease (LCDD), monoclonal immunoglobulin (Ig) light chain deposition usually involves the kidney [1]. End stage renal failure (ESRF) occurs in 70% of patients [2]. Outcomes of kidney transplantation are poor, due to recurrent allograft disease or progression of the underlying plasma cell dyscrasia [1,3]. Thus, renal transplant strategies must address the underlying disease. We describe the first reported case of LCDD treated with sequential autologous peripheral blood stem cell (PBSC) transplantation and kidney transplantation.

A 58-year-old male presented with mild anaemia and renal impairment (serum creatinine 2.8 mg/dl). Urinary protein excretion was 0.71 g/day. Serum protein electrophoresis revealed an IgG kappa paraprotein level of 1000 mg/dl, with depressed IgA and IgM. Bone marrow examination showed 5–6% plasma cells. Renal biopsy showed granular tubular protein deposits, thickening of the tubular basement membrane and peri-tubular sclerosis, in keeping with LCDD. Kappa light chain immunoperoxidase stain was positive (Figure 1). The patient was treated with chemotherapy,
underlying disease, despite no evidence of recurrent light
incomplete suppression and subsequent progression of the
was still significant treatment-related toxicity with
dose was modified because of renal impairment. There
post-renal transplant immunosuppression. Chemotherapy
based on concerns about additive risks of chemotherapy and
PBSC transplantation. Kidney transplantation was performed
first because of the limitations on drug dosing imposed by
ESRF. One patient died, but the rest remained well without
recurrence of light chain deposition.

Twenty-six months post-renal transplant, IgG level was
500 mg/dl, with serum kappa free light chains of 83.2 mg/l (3.3–19.4).
Severe neutropenic sepsis precluded a planned second
PBSC transplant. A year later, the patient underwent
HLA-identical living donor kidney transplantation.
Post-operatively there was delayed graft function. Serum
creatinine improved to a baseline of 2 mg/dl.

Our patient is the first case of LCDD treated with
sequential transplantation. Leung
[4] described seven patients with the related disorder primary amyloidosis who
underwent sequential kidney transplantation and autologous
PBSC transplantation. Kidney transplantation was performed
first because of the limitations on drug dosing imposed by
ESRF. One patient died, but the rest remained well without
recurrent amyloidosis (follow-up period 0.7–4.1 years).

Our decision to perform PBSC transplantation first was
based on concerns about additive risks of chemotherapy and
post-renal transplant immunosuppression. Chemotherapy
dose was modified because of renal impairment. There
was still significant treatment-related toxicity with
incomplete suppression and subsequent progression of the
underlying disease, despite no evidence of recurrent light
chain deposition in the renal allograft.

Conflict of interest statement. The content presented in this article
has not previously been published in whole or in part. There was no
conflict of interest for any of the authors of the article. We have had
no involvements that might raise the question of bias in the work
reported, or in the conclusions, implications or opinions stated.

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doi:10.1093/ndt/gfl669

Advance Access publication 28 November 2006

Human Urotensin II in the plasma of anephric subjects

Sir,

Human urotensin II (UII), perhaps the most potent
mammalian vasoconstrictor known, is thought to be
produced by the kidneys. The urotensins are a family of
vasoactive peptides first isolated from various fish species
over 20 years ago. Homologous peptides have been isolated in
numerous species including frogs, rodents, pigs, primates and
humans. It has been demonstrated in-vitro that human UII is
between 8- and 110-fold more potent than endothelin-1 as
a vasoconstrictor, and is the most potent mammalian
vasoconstrictor known. The precise metabolic pathway(s)
of urotensin metabolism is unknown, though high density of
UII and its receptor have been demonstrated in mouse
and monkey kidneys [1] and human kidneys, [2] and is thus
thought to be synthesized, secreted and cleared by the
kidneys [3,4,5]. Also, some researchers have reported higher
blood values of UII in patients with kidney disease as well as
with hypertension. These reports have stimulated interest in
a possible aetiological role of UII in kidney disease and
hypertension in people with kidney disease; consequently
we sought to determine whether plasma concentrations of
UII are detectable in subjects with end-stage renal disease
(ESRD) on dialysis, and in particular in surgically anephric
subjects.

Thirty-one ESRD subjects undergoing routine haemodia-
lysis were enrolled, including two surgically anephric subjects
(i.e. both patients had bilateral nephrectomy at least 6
months prior to enrolment). Blood samples were obtained for
UII before initiation of a mid-week dialysis session.
UII concentrations were measured in unextracted plasma
by radioimmunoassay using human UII-specific monoclonal
antibody at GlaxoSmithKline laboratories (NA). The
mean UII was compared between anephric and non-anephric
subjects with a paired Students’ t-test, using JMP IN
statistical software version 4 (SAS Institute, Carey,
NC, USA).

Fig. 1. Pre-transplant kidney biopsy showing moderate widespread
deposition of kappa light chains within and surrounding many
tubular basement membranes. Immunoperoxidase staining for
kappa light chain; original magnification ×400. Bar = 100 μm.