In all, 192 prospective kidney donors underwent evaluation at our center between February 2002 and May 2005 that included renal imaging. Seventy-six were eventually excluded for medical or surgical reasons. Out of those excluded, eight were found to have one or more kidney stones by ultrasound (two patients) or computed tomography (CT) scan (six patients). No patient gave a previous history of hypertension, nephrolithiasis or visible hematuria. Kidney size was 11±0.7 cm, with average asymmetry 0.3 cm. Stone size varied from 1 to 7 mm in diameter with up to three in one kidney; two patients had bilateral stones. Ureteric stones, bladder stones, or renal cysts were not detected in any patient. Their age was 43±13 years, three were male, four were white, three Asian, and one Native American and BMI was 27.1±3. Their fasting and two-hour post 75 gram glucose load blood sugar levels and hemoglobin were normal, but one patient had hypercholesterolemia. All patients had a normal 24-hour urine protein excretion rate or random urine microalbumin-to-creatinine ratio, as well as a normal 24-hour urine creatinine clearance (120±21 ml/min). Although the average blood pressure was normal (124/70 mm Hg by 24-hour ambulatory BP monitoring), seven were nocturnal nondippers. Four out of these eight patients had persistent microscopic hematuria.

Seven patients provided a 24-hour urine collection to evaluate for metabolic abnormalities favorable to stone formation. Three had significant hypercalciuria (9.2–16.3 mmol/d, N 0–7.5), three had hypocalciuria (0.4–1.0 mmol/d, N >1.6), two had hyperuricosuria (5.5–5.9 mmol/d, N 2–4), and two had hyperoxaluria (508–814 μmol/d, N 190–480). Three patients had more than one metabolic abnormality.

Nephrolithiasis can cause microscopic hematuria without other clinical manifestations. In one study (2), 195 of 906 subjects with asymptomatic hematuria were found to have renal calculi by ultrasound. Twenty-four of these subsequently required urological management (2). Hypercalciuria and hyperuricosuria may also cause microscopic hematuria in adults, which can resolve with appropriate therapy such as thiazides and allopurinol (3). In addition, hypercalciuria, hyperuricosuria, and nephrolithiasis are associated with thin basement membrane nephropathy, a glomerular disease (4). Although we typically decline these individuals as kidney donors, we agree with Koushik et al. that clear guidelines for managing young and healthy adults with microscopic hematuria are lacking.

In summary, in addition to the list provided by Koushik et al., occult nephrolithiasis should be considered as a cause for microscopic hematuria in the prospective kidney donor population. This will enable appropriate diagnosis, counseling, referral, and management of these individuals independent of the transplantation process.

Dengue Shock Syndrome in a Liver Transplant Recipient

Dengue fever is the world’s most important viral hemorrhagic fever disease. It is a major cause of mortality and morbidity in tropical areas (1). Its clinical outcome ranges from asymptomatic cases through dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). We report the first case of DSS in a liver transplant recipient.

A 66-year-old man had been the recipient of a cadaveric ABO compatible donor liver allograft. He was diagnosed with hepatitis C cirrhosis and classified Child Pugh B9. The postoperative immunosuppressive treatment consisted of prednisone and tacrolimus. Three weeks after transplant, he developed diarrhea associated with fever and abdominal pain. On the physical examination, he had jaundice. On the admission day, investigations revealed a platelet count of 103,000/mm³, a creatinine of 1.5 mg/dL, a hemoglobin of 13 g/dL (packed cell volume, 38.6), a serum aspartate amino-transferase of 931 U/L, and a serum alanine aminotransferase of 427 U/L. At admission, an abdominal ultrasonography (US) revealed ascites and hepatomegaly. Doppler US was normal. The patient’s blood and urine culture were sterile. Antibiotic therapy with vancomycin, ganciclovir and cefepime was initiated. Twelve hours after admission, the patient became anuric. He deteriorated into a stage of profound shock. Dopamine was initiated. On the day after admission, the patient’s platelet count, hemoglobin, serum aspartate aminotransferase, alanine aminotransferase, and prothrombin time (International Normalized Ratio) were 30,000/mm³, 14.2 g/dL (cell packed volume, 42.3), 14,096 U/L, 2,717 U/L and 2.4, respectively. The patient’s general condition became worse. The patient died within two days of admission. Serology and polymerase chain reaction (PCR) for cytomegalovirus were negative. Serum quantitative PCR and liver tissue immunohistochemistry and PCR confirmed the dengue diagnosis of type 3 dengue virus. The patient had no documented previous history of dengue infection. He fulfilled the World Health Organization criteria for grade IV DSS.

Immune response plays an important role in the pathophysiology of DHF-DSS. Sequential infection with different dengue virus genotypes is related to the development of DHF-DSS. After

REFERENCES

the first infection, antidengue antibod-
ies against the specific genotype respon-
sible for this infection will be present in
the patient’s blood. These antibodies are
not effective against others genotypes,
but they are responsible for a robust im-
uminologic responsiveness in a second
infection. Activated T-cell and cytokine
mediators initiate hemorrhagic mani-
festations and capillary leakage of plasma
in DSS (2). Cytokines, mainly IL-6, IL-10,
and macrophage migration factor (MIF)
are believed to be involved in the patho-
gensis of dengue infection and serve as a
predictor of its outcome (3).

Liver transplant recipients are im-
umosuppressed in order to avoid re-
jection. Their T-cell responsiveness is
diminished, which may explain the lack
of cases of DSS in transplanted patients.
The case that we reported in an area that
is endemic for dengue hemorrhagic fe-
ver is extremely important to alert phy-
sicians for its diagnosis.

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REFERENCES
Invasion and maintenance of dengue virus
type 2 and type 4 in the Americas. J Virol
2. Burke DS, Monath TP. Flaviviruses. In:
Knipe DM, Howley PM, eds. Fields virology.
of serum levels of macrophage migration in-
hibitory factor with disease severity and clin-
ical outcome in dengue patients. Am J Trop

KSHV/HHV-8 Infection of Tubular Epithelial Cells in Transplantation Kidney

Epidemiological studies have con-
sistently shown that posttransplant KS
(PT-KS) may be either due to the reactiva-
tion of Kaposi’s sarcoma associated her-
pesvirus (KSHV)/human herpesvirus-8
(HHV-8) infection in solid organ recip-
ients, or to the primary infection with
the virus transmitted from the donors
(1). In the latter cases, the source of viral
transmission remains unknown. KSHV/
HHV-8 is known to infect endothelial
and lymphoid cells, but the viral tropism
is likely to be broader than initially sus-
pected. Relevant to this, in a case of dis-
seminated acute primary KSHV/HHV-8
infection affecting a newborn, the virus
could be found by in situ hybridization
in the kidney, both in the glomerulo-
endothelial and in the tubular epithelial
cells (2). Furthermore, experiments in a
transgenic mouse model have demon-
strated that the kidney tubular epithe-
lium is permissive to the KSHV/HHV-8
latency associated nuclear antigen (LANA)
promoter activity and consequently to
KSHV/HHV-8 latency (3).

We have previously reported one
case of primary infection with KSHV/
HHV-8 in a renal recipient followed by the
development of a disseminated PT-KS
and related hemophagocytic syndrome. In
the present study we used a combination of
immunohistochemical and molecular
methods to test the hypothesis that the do-
nor’s kidney could represent a site of viral
infection (4). Archival specimens from the
formalin-fixed and paraffin embedded tis-

FIGURE 1. Micromanipulation of a LANA-positive cell from tubular epithelium
of donor’s kidney biopsy from patient one. Sections of paraffin-embedded renal
tissue were stained with a monoclonal antibody to the KSHV latency-associated
nuclear antigen, LANA-1 (Novocastra Laboratories Ltd, Newcastle, UK), as de-
scribed previously (1). LANA-1 positive cells were then isolated by microdissec-
tion. The tissue section is shown before (A) and after (B) the isolation of the cell.
PCR detection of KSHV orf26 sequence in 2 of 12 samples of microdissected
LANA positive cells from patient (C). MK, molecular weight marker IX; NC,
negative control; PC, positive control, represented by the DNA extracted from
the PT-KS.

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