showed unspecific ileitis and colitis. Abdominal CT scan showed portal vein thrombosis with progression to superior and inferior mesenteric vein and splenic vein (Figure 1). Anti-thrombin III, protein C and S were normal; lupus anticoagulant $1.19$ ($<1.2$); anticardiolipin antibody was positive (IgG 30.5/IgM 50: $<10$).

Our patient meets the criteria for SLE and for APS [1,4]. APS is characterized by lupus anticoagulant, anticardiolipin antibodies, vascular thrombosis, thrombocytopenia and recurrent foetal losses [4]. APS occurs frequently in patients with SLE [4]. Hirohata et al. [5] described a patient with portal vein thrombosis associated with APS. In our patient, the loss of protein may be explained by the increased intestinal congestion caused by portal vein thrombosis. The increase of the portal vein pressure causes increased intestinal congestion and lymph production and subsequent protein leakage [6]. A normal lymphocyte count, elevated serum cholesterol and absence of lymphangiectasia on intestinal biopsy help distinguish lupus-associated PLE from PLE due to direct or indirect lymphatic obstruction [3]. Normal endoscopy and mucosal biopsy can rule out protein loss due to mucosal disruption [3].

Corticosteroids have been demonstrated to dramatically improve the course of PLE, particularly when signs of an inflammatory disease are present [3,6]. Another therapeutic option is octreotide, which reduces hepatic and splanchnic blood flow, lowering portal pressure [6,7]. The patient’s symptoms improved with daily corticosteroid, octreotide and anticoagulation with warfarin.

Conflict of interest statement. None declared.

Is salt-wasting the long awaited answer to the hyperuricaemia seen in uromodulin storage diseases?

Sir,

Recent articles have revealed that familial juvenile hyperuricaemic nephropathy (FJHN) and medullary cystic kidney disease (MCKD) are caused by mutations in uromodulin, which is also known as Tamm–Horsfall protein [1–3]. These uromodulin storage diseases present with varying degrees of chronic interstitial renal disease, hyperuricaemia, a reduced fractional excretion of uric acid (FeUA) and renal salt-wasting [4,5]. However, the aetiology of the hyperuricaemia seen in these diseases remains a mystery. How can a mutation in THP, a protein produced exclusively in the thick ascending limb (TAL), result in hyperuricaemia when all the uric acid transport is believed to occur only in the proximal tubule? One explanation is that salt-wasting causes a compensatory upregulation of both sodium and uric acid transport in the proximal tubule.

Recently, the THP knockout mouse was found to have increased expression of distal tubule sodium transporters [6]. We further examined salt and water balance in these animals (Table 1). These animals have normal renal histology and do not have an elevated serum uric acid level, likely secondary to the presence of the enzyme uricase. We found a striking difference in the ratio of urinary sodium to urinary uric acid (wildtype $n = 7$, $1.98 \pm 0.19$ vs knockout $n = 14$, $0.53 \pm 0.03$, $P = 0.002$) (Figure 1). Thus, for a given amount of salt excretion, the knockout animals are excreting less uric acid. To account for this finding, kidney mRNA levels of uric acid transporters were evaluated by real-time RT-PCR and Western blot, but no significant differences were detected.

None of the described uric acid transporters are directly coupled to sodium transport. One possible explanation for this data is the existence of an as yet undescribed sodium linked uric acid transporter. Another explanation is that uric acid transport is upregulated along with sodium transport in the proximal tubule. To further investigate this hypothesis, more formal studies of uric acid transport will be needed. However, the data from our experiment are consistent with the theory that the decreased FeUA seen in FJHN may result from distal salt-wasting and a compensatory upregulation of proximal tubule resorption of sodium and uric acid.

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doi:10.1093/ndt/gfl043

Advance Access publication 18 January 2006
Table 1. Serum and urine chemistries

<table>
<thead>
<tr>
<th>Chemistry</th>
<th>+/+</th>
<th>−/−</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine sodium mmol/l</td>
<td>69±18.72</td>
<td>79±24.46</td>
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<tr>
<td>Urine creatinine mg/dl</td>
<td>37.3±14.37</td>
<td>32.3±12.01</td>
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<tr>
<td>Urine uric acid mg/dl</td>
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<td>16.9±6.36</td>
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<td>Serum sodium mmol/l</td>
<td>156±5.91</td>
<td>154±2.62</td>
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<tr>
<td>Serum creatinine mg/dl</td>
<td>0.2±0.05</td>
<td>0.2±0.06</td>
<td>0.78</td>
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<tr>
<td>Serum uric acid mg/dl</td>
<td>0.8±0.95</td>
<td>0.8±0.55</td>
<td>0.91</td>
</tr>
<tr>
<td>FeNa</td>
<td>0.30±0.12</td>
<td>0.43±0.24</td>
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<tr>
<td>FeUA</td>
<td>35.22±19.5</td>
<td>23.77±13.7</td>
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<tr>
<td>FeNa/FeUA</td>
<td>150.2±26.6</td>
<td>64.2±31.3</td>
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<tr>
<td>Urine sodium/urine creatinine</td>
<td>1.98±0.19</td>
<td>0.53±0.03</td>
<td>0.002*</td>
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</tbody>
</table>

*P < 0.05.

Fig. 1. Ratio of urine sodium to urine uric acid.

Conflict of interest statement. The authors have no conflict of interest to declare.


doi:10.1093/ndt/gfk081

Advance Access publication 31 January 2006

Tests for latent tuberculosis

Sir,

In their article, Shankar and colleagues underline the significant burden of tuberculosis within their population and the importance of identifying latent infection in those with end stage renal disease (ESRD) [1]. They found significant rates of anergy to cutaneous tuberculin skin testing in those with ESRD (44% vs 16% in control group). The study adds to the evidence in other populations and supports the notion that cutaneous anergy limits the value of this test [2]. We have previously reported the use of molecular biological techniques to help improve the diagnostic certainty of clinical infection with tuberculosis in this patient group [3]. Similarly, novel molecular techniques have recently been developed to detect the presence of latent tuberculosis infection. Immunoassays based on the detection of interferon-γ to specific Mycobacterium tuberculosis antigens ESAT6 and CFP10, appear to be specific and sensitive for the diagnosis of latent tuberculosis [4]. These techniques can detect latent infection in immunosuppressed patients and can differentiate between those previously vaccinated with the Bacillus Calmette Guérin (BCG) strain, a known confounding factor with tuberculin skin testing [5]. Importantly, the detection of latent disease using these assays seems to correlate with patients who will develop clinical infection [6]. We, therefore, recommend the consideration of these techniques to improve the management of this complex group of patients.

Conflict of interest statement. None declared.

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