Thottapalayam virus (TPMV) is the only indigenous hantavirus documented so far in India, being isolated in 1964 from a shrew near Vellore in Southern India [7]. To date, no human pathogenicity for TPMV has been shown [7], but it is noticeable that shrews are insectivores, not rodents. So far, human hantavirus pathogenicity has been linked to rodents only [2]. Although we found evidence of a SEOV-like infection in 12% of the Indian ‘leptospirosis-like’ cases, another 5%, with all the more severe cases, looked surprisingly like PUUV infections. However, the worldwide geographical repartition of hantaviruses is well defined by a close co-evolution with their respective rodent carriers [2]. But the bank vole is absent from the Indian biotope and, consequently, PUUV should also be absent or spread eventually by another arvicoline, as yet unidentified, rodent. We prefer to believe, however, that our positive PUUV (and SEOV?) results may be cross-reactions with TPMV or yet another unknown hantavirus in India. Thus, the low yield of our screening may be explained by the fact that only, or in part, cross-reacting antigens were used. Finally, severe clinical courses as depicted above are atypical for the mostly milder NE. In fact, the fatal outcome of the Cochin case is rather reminiscent of (South-) American forms of the so-called ‘hantavirus (cardio-) pulmonary syndrome’ (HPS). However, HPS can exceptionally also occur in Old World PUUV cases [8].

To elucidate this problem, we are now in the process of confirming our results using the polymerase chain reaction and with neutralization tests on the largest possible battery of known hantaviruses, preferably also including TPMV. Without this gold standard for hantavirus serology [2], no final decision can be made about the exact nature and importance of hantaviruses in India.

Acknowledgements. We wish to thank Prof. W. Terpstra and Dr H. Hariski from the Tropical Institute of Biomedical Research, Amsterdam, the Netherlands, for their help in providing us with the Indian sera.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfi334

Advance Access publication 1 November 2005

Sheehan syndrome presented with acute renal failure associated with rhabdomyolysis and hyponatraemia

Sir,

Hyponatraemia is a frequent complication of moderate to severe hypothyroidism [1]. Thus, thyroid function should be evaluated in any patient with an otherwise unexplained reduction in the plasma sodium concentration. There have been several case reports of rhabdomyolysis, which may lead to acute renal failure, associated with hypothyroidism [2]. We describe a patient with acute renal failure due to rhabdomyolysis, Sheehan syndrome and hyponatraemia.

Case. A 58-year-old woman presented with confusion, severe myalgia, lower limb weakness and oliguria for 3 days prior to admission. She denied any form of strenuous muscle exercise. Her physical findings and a history of amenorrhoea following massive postpartum haemorrhage 20 years previously suggested Sheehan’s syndrome, and the pituitary hormonal studies revealed panhypopituitarism. She had not taken any treatment for this illness. Her laboratory findings were as follows: serum sodium 94 mmol/l, chlorine 70 mmol/l, urea 32 mg/dl, creatinine 1.3 mg/dl, aspartate aminotransferase (AST) 2711 U/l, alanine aminotransferase (ALT) 2321 U/l, creatine kinase (CK) >40 000 IU/l, lactate dehydrogenase (LDH) 2396 U/l, T4 <0.3 ng/dl, thyroid-stimulating hormone (TSH) 6.1 mIU/ml, FSH 2 miU/ml, LH 1 mIU/ml, cortisol 21 µg/dl, leucocytes 30 000, C-reactive protein (CRP) 200 mg/l. Due to poor general condition of the patient, with leucocytosis and a high CRP, empiric antibiotic treatment was started for possible sepsis; however, no infection source could be found. In addition, methylprednisolone and thyroxine were started.

While the sodium level was normalizing, creatinine and CK rose abruptly and oliguria developed, leading to the start of haemodialysis. Extensive intramuscular haemorrhages developed in the scapular and gluteal muscular regions (Figures 1 and 2), with progressive decrease of haemoglobin levels and increase of INR. A total of 10 units of blood was transfused. Haemorrhages could not be stopped despite replacement with fresh frozen plasma. Although the patient was afebrile, leucocytosis, high CRP level and disseminated intravascular coagulopathy suggested sepsis. The patient ultimately died. We could not find any similar case presenting with intramuscular haemorrhages in the literature.

Rhabdomyolysis and acute renal failure due to hypothyroidism is a rare entity. There are few reported cases in the literature [3–6]. Three important features were present in our patient, first an undiagnosed and untreated Sheehan syndrome of 20 years duration, second a severe and possibly chronic hyponatraemia associated with hypothyroidism, third, the development of severe intramuscular

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haemorrhages. The rhabdomyolysis was enhanced possibly due to the severe hyponatraemia. Hypothyroidism should be in the differential diagnosis in patients with acute renal failure associated with rhabdomyolysis and hyponatraemia. Prolonged hypothyroidism may increase mortality.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfi234

Advance Access publication 1 November 2005

Lamivudine and HBV-associated nephropathy

Sir,
Tang and colleagues [1] report that lamivudine treatment improves renal outcome in HBV carriers with membranous nephropathy (MN) and evidence of liver disease. In children with HBV-associated MN, lamivudine has also been anecdotally reported to induce remission of nephrotic syndrome in two case reports [2,3]. We would like to add our experience with lamivudine in one similar case (Table 1).

Since July 2002, we have treated with lamivudine (100 mg p.o., daily) one HbsAg positive Asian patient with nephrotic syndrome, biopsy proven stage II membranous nephropathy, elevated serum alanine aminotransferase (ALT), and evidence of HBV-DNA. A liver biopsy showed focal lobular and portal inflammation and changes consistent with a mild chronic hepatitis. There was significant reduction of proteinuria from 6 months of starting lamivudine treatment and the patient went into complete remission (proteinuria <0.01 g/24 h) within 1 year. By 3 years, she continued to take lamivudine with a permanent negative proteinuria. HBV DNA levels dropped from 6750 copies/ml pre-treatment to undetectable levels during treatment, at follow-up 12 months on lamivudine. Liver function tests remain normal. Lamivudine was well tolerated and not associated with any adverse events that required dosage adjustment or drug discontinuation.

Lamivudine treatment has been successful in adults with HBV-associated polyarteritis nodosa [4], and in combination with other antiviral agents to treat HIV-associated nephrotic syndrome [5]. In HBVAN, the duration of lamivudine therapy remains an unresolved issue, notably in Asian patients, as emerging data show that disease progression can continue even after anti-HBe seroconversion in this population [6]. Indeed, one patient described by Tang et al. suffered a relapse of nephrosis after 2 years of complete remission when treatment was withdrawn [1]. Thus, it is reasonable to continue lamivudine treatment for as long as possible after initial remission.

We show that lamivudine significantly improves the clinical outcome of HBV-related MN.

Conflict of interest statement. None declared.

Fig. 1. Extensive intramuscular haemorrhages in the gluteal region.

Fig. 2. Extensive intramuscular haemorrhages in the scapular region.

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