Idiopathic retroperitoneal fibrosis: a rare onset of the illness caused by haemorrhagic fever with renal syndrome

Sir,

Idiopathic retroperitoneal fibrosis (IRF) is a collagen vascular disease of unknown aetiology. It is characterized by chronic, non-specific retroperitoneal inflammation, which may cause ureteric obstruction. Many authors believe that it is a type of immune disorder [1]. Bosnia and Herzegovina is known as a region where Hanta virus infection has been endemic for >50 years [2]. A case of IRF associated with haemorrhagic fever with renal syndrome (HFRS) has not been reported up to now.

A 44-year-old, previously healthy man was hospitalized with acute renal failure. He was febrile 2 days before admission, had dull abdominal pain, decreased urine output, shortness of breath, diarrhoea and arterial hypertension (190/120 mmHg). Blood tests showed metabolic acidosis (HCO₃⁻: 15.9 mmol/l) and increased C-reactive protein (11.26 mg/l), potassium (7.9 mmol/l), serum creatinine (884 µmol/l), blood urea nitrogen (17.6 mmol/l) and globulins (49.1 g/l) and decreased haemoglobin (7.4 mmol/l). Urinalysis showed proteinuria and leukocyturia. Urine culture was negative. Indirect immunofluorescence tests for Hanta viruses were positive for Puumala virus. Ultrasound showed acute renal parenchymal lesions with bilateral hydronephrosis, grades I–II, and widening of the wall of the abdominal aorta. The presence of a great number of rodents in the forest where the patient was working has been reported by the epidemiology service.

After supportive, antihypertensive and diuretic therapy, the patient’s renal function stabilized, with serum creatinine at 187 µmol/l and potassium at 4.5 mmol/l. Intravenous urography showed a functioning left kidney, with a suspected retrocaeval ureter on the right side and dilation of the channels of the right kidney. A computed tomography contrast scan showed a solid retroperitoneal mass, in the form of thick plate of high density, extending from the level of the renal hilum down caudally to the bifurcation of the aorta (compatible with retroperitoneal fibrosis). A double-J stent was applied and steroids and androgens were administered (pronison 60 mg plus tamoxifen 20 mg x 2). After 3 months, the stents were removed and medications were continued. After 6 months, the patient’s total DTPA clearance was 61.1 ml/min (11.8 ml/min in the left kidney and 49.3 ml/min in the right kidney), measured by technetium 99m marked by diethylaminoacid. Steroids and androgens were withdrawn after 12 months. The patient has normal blood pressure and stable renal function, with serum creatinine at 125 µmol/l.

It remains a mystery whether HFRS triggered an immune abnormality and acceleration of the symptoms of a latent IRF or whether the two diseases merely coincided. Adequate treatment of HFRS was certainly the reason that renal function recovered and the progression of the disorder caused by the chronic disease, IRF, was hampered.

Conflict of interest statement. None declared.

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Haemodialysis and thermoregulation

Sir,

We read with great interest the editorial comment of Drs Passlick-Deetjen and Bedenbender-Stoll [1]. This editorial rightly stresses the importance of thermal factors in the pathogenesis and prevention of intra-dialytic hypotension (IDH). Thermal factors are primarily responsible for the inappropriate cutaneous vasodilation observed during dialysis with standard dialysate temperatures (37–37.5 °C) and are the explanation for differences in the haemodynamic response between haemodialysis on the one hand and isolated ultrafiltration and haemo(dia)filtration on the other hand [2–4].

However, we would like to comment on two issues addressed in this review. The first concerns the pathophysiology of the increase in core temperature during haemodialysis, which even occurs if no energy is added from the extracorporeal circuit to the patient (so-called thermoneutral treatments). The authors state that this phenomenon is primarily due to initial cutaneous vasoconstriction caused by ultrafiltration, which prevents the dissipation of heat from the body (Gotch hypothesis). This hypothesis appeared to be confirmed by the significant relation between the ultrafiltration rate and the energy removal needed to prevent the increase in core temperature during so-called isothermic dialysis [5]. However, in collaboration with the group of Nathan Levin, we performed a falsification study of this hypothesis and found no differences in core temperature changes between thermoneutral dialysis treatments either with or without ultrafiltration. Moreover, no differences in extracorporeal energy flow rate were observed between isothermic treatments respectively performed with and without ultrafiltration [6]. In addition, no study yet demonstrated initial vasoconstriction followed by vasodilation during haemodialysis. In contrast, cutaneous vasodilation is already observed shortly after the start of dialysis [7]. Therefore, although we were not able to elucidate the primary responsible factors for the increase in core temperature during dialysis in this study, the Gotch hypothesis clearly did not pass the falsification process.

Our second comment concerns the use of cool (temperature) dialysis. The reader of the editorial might feel somewhat discouraged to lower dialysate temperatures because cool dialysis may lead to shivering. However, as the