Acute nephritis induced by human parvovirus B 19 infection

Sir,

The relationship of human parvovirus B 19 (HPB 19) with renal disease is rare, with only two studies describing glomerulonephritis in patients with erythema infectiosum and sickle cell disease (SCD) [1]. We describe a case of acute nephritis associated with HPB 19, which, to the best of our knowledge, is the first case reported without associated SCD.

A 54-year-old Caucasian woman was admitted to a medical ward with a 3-week history of generalized malaise, arthralgia and a widespread macular rash. She was in frequent contact with her grandson, who had been recently diagnosed with erythema infectiosum (fifth disease). She had a past history of Raynaud’s disease, migraine, sinusitis and breast carcinoma, previously treated with surgery and radiotherapy. Her only long-term medication was tamoxifen. There was no family history of autoimmune or renal disease. Although she was discharged, the patient failed to improve, and was subsequently re-admitted to our renal unit with haematuria and proteinuria.

On examination, she was apyrexial, tachycardic and hypertensive, with a blood pressure of 163/90 mmHg. There was a widespread macular rash with a mottled appearance, consistent with erythema infectiosum. Although previously normal, the patient’s serum creatinine was elevated to 195 mmol/l. There was a normochromic normocytic anaemia with haemoglobin of 9.5 g/dl. Serum ANCA, anti-GBM and ANA titres were negative, with a weakly positive dsDNA antibody. Complement C3 and C4 levels were reduced at 0.34 g/l (normal range: 0.70–1.70 g/l) and 0.10 g/l (normal range: 0.18–0.58 g/l) respectively. Other investigations showed rheumatoid factor increased at 43 IU/ml (normal range <20 IU/ml) and creatinine clearance reduced to 15 ml/min (normal range: 60–140 ml/min) with a proteinuria of 0.97 g/24 h (normal range: <0.15 g/24 h). The C-reactive protein was elevated at 30 mg/l (normal range: <5 mg/l) with an ESR of 26 mm/h (normal range: <20 mm/h). Renal ultrasonography, immunoglobulins, paraproteinaemia and cryoglobulins were normal. Subsequently, polymerase chain reaction (PCR) for HPB 19 was positive, confirming current infection.

Her condition improved with symptomatic treatment. The laboratory abnormalities, including urinary protein, microscopic haematuria and rash improved within a few days; renal functions and complement levels returned to the normal range and her dsDNA became negative.

A link between renal disease and human parvovirus was first implied by Markenson et al. [2], who described two siblings with SCD and hypoplastic crises, who developed nephrotic syndrome. Similarly, Wierenga et al. [1] described seven patients with homozygous SCD who showed proliferative segmental glomerulonephritis with proteinuria and nephritic syndrome after aplastic crisis induced by HPB 19.

There have recently been several reports indicating that HPB 19 infection may elicit symptoms and laboratory findings resembling those of systemic lupus erythematosus, such as polyarthritis, positive antinuclear antibodies and hypocomplementaemia [3]. However, these reports have not demonstrated renal abnormalities as a complication of the disease. Although HPB 19 infection has been reported to be associated with vasculitis including Henoch–Schönlein purpura [4] and necrotizing vasculitis resembling polyarteritis nodosa [5], there has been no previous accounts of involvement of the renal vasculature.

Our patient with confirmed erythema infectiosum suffered an acute nephritis manifesting as acute renal failure with an active urinary sediment. The temporal relationship between the two conditions strongly suggests causality. Nephritis with HPB 19 infection has not been previously reported in patients without SCD. As the patient’s condition improved spontaneously, renal biopsy to confirm a glomerulonephritis was not justified.

In conclusion, infection with HPB 19 should be considered as a possible cause of acute nephritis in adults. This usually occurs in patients with SCD but can also occur in its absence.

Conflict of interest statement. None declared.

Royal Liverpool and Broadgreen University Hospital NHS Trust  
Matthew Howse  
Nephrology  
Link 6C, Prescott Street  
Liverpool L7 8XP, UK  
Email: vcg_in@yahoo.com


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Six cases of successful cinacalcet cessation in haemodialysis patients treated for secondary hyperparathyroidism

Sir,

Cinacalcet acts as an allosteric activator of calcium-sensing receptor (CaR) and diminishes Parathyroid Hormone (PTH) secretion [1]. For more than 2 years, it has been successfully used for the treatment of secondary hyperparathyroidism (SHPT) [2], and has been shown to facilitate achievement of the KDOQI-recommended targets for PTH, calcium, phosphorus and Ca × P product [3], with a sustained effect lasting for >2–3 years [4]. This expensive treatment is thought to be...
for life and the possibility of complete weaning had not been reported.

Among the 59 Haemodialysis (HD) patients who have been treated with cinacalcet for SHPT since 2004, we report here six successful cases of complete weaning after 12 months: four females and two males, 63 ± 20 years old, diabetics in 2/6 cases, dialysed for 41 ± 42 months, with a mean 3 × 6 h 15 min schedule achieving a mean 2.2 ± 0.6 Kt/V, using a standard dialysate calcium of 1.5 mmol/l. Serum levels of calcium, phosphorus and iPTH (Roche Elecsys) were recorded monthly before dialysis, and bone turnover markers every 3 months: phosphorus and iPTH (Roche Elecsys). Cinacalcet, 30 mg tablets were prescribed at a dose 30 mg on alternate days for a period of 3 months. Alfacalcidol dose was tapered according to PTH, Ca × P and bone markers level. Criteria for cinacalcet weaning were iPTH < 40 pg/ml, BALP < 25 μg/l, CTX < 2 μg/l and Ca × P level < 4.0 mmol²/l achieved with the lowest dose of cinacalcet, i.e. 30 mg on alternate days for a period of 3 months.

The biological and therapeutic evolution is displayed in Table I. Six months after cinacalcet cessation, serum level of PTH, calcium, phosphate and bone markers slightly increased but remained within the desirable targets, with an increase need for phosphate binder and alfacalcidol. Dialysis prescription remained stable during the study period, especially the 1.5 mmol/l dialysate calcium.

Cinacalcet is a very efficient treatment of SHPT in dialysis patients, to a degree that allows, under specific conditions, for a complete weaning trial in certain cases. Out of the great and rapid initial biological improvement, we found no baseline characteristics predicting the possibility for tapering both alfacalcidol and cinacalcet and eventually leading to cessation of cinacalcet after 1 year. The underlying explanation for such an evolution remains to be elucidated. Parathyroid cell CaR and Vitamin D Receptor (VDR) expression would be interesting to measure, but it is difficult to obtain in clinical practice. Besides, we do not systematically follow-up ultrasound parathyroid gland size and a possible decrease in gland hyperplasia. Very close biological monitoring, including bone markers in these vitamin D-replete patients, seems a very helpful strategy. Long-term evolution is unknown, especially in case of future kidney transplantation. Due to its high cost, cinacalcet should be used under close follow-up, evaluation criteria leading, in some cases, to a complete weaning trial.

Conflict of interest statement. All the authors declare that they are the scientific consultants for Fresenius Medical Care.

Centre de Rein Artificial, Guillaume Jean
Tassin la demi-lune, France
Email: guillaume-jean-erat@wanadoo.fr

Bernard Charra


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