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**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 grandmother, who had been recently diagnosed with erythema infectiosum (fifth disease). She had a past history of Raynaud’s disease, migraines, 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 afebrile, 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 anaemia with haemoglobin of 9.5 g/dl. Serum phosphorus and Ca product [3], with a sustained effect lasting 2–3 years [4]. This expensive treatment is thought to be beneficial in patients with SCD but can also occur in its absence.

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
Cinacalcet (mg/d) 0 45
Sevelamer (tab/d) 5.8
Alfacalcidol (µg/l) 58 ± 25
CTX (µg/l) 4.4 ± 1.6
Alfacalcidol (µg/w) 3.1 ± 2.5
Sevelamer (tab/d) 5.8 ± 4
Cinacalcet (mg/d) 0

<table>
<thead>
<tr>
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<th>Baseline</th>
<th>3 months</th>
<th>6 months</th>
<th>9 months</th>
<th>Weaning at 1 year</th>
<th>After 6 months of weaning</th>
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<tbody>
<tr>
<td>PTH (pg/ml)</td>
<td>1039 ± 753</td>
<td>346 ± 158</td>
<td>198 ± 108</td>
<td>142 ± 70</td>
<td>60 ± 28</td>
<td>143 ± 96</td>
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<tr>
<td>Ca (mmol/l)</td>
<td>2.5 ± 0.1</td>
<td>2.2 ± 0.07</td>
<td>2.16 ± 0.07</td>
<td>2.16 ± 0.07</td>
<td>2.15 ± 0.04</td>
<td>2.28 ± 0.27</td>
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<td>(2.4–2.6)</td>
<td>(2.1–2.4)</td>
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<td>Phosphataemia</td>
<td>1.65 ± 0.2</td>
<td>1.38 ± 0.3</td>
<td>1.46 ± 0.2</td>
<td>1.26 ± 0.18</td>
<td>1.4 ± 0.2</td>
<td>1.48 ± 0.2</td>
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<tr>
<td>BALP (µg/l)</td>
<td>34 ± 18</td>
<td>25 ± 15</td>
<td>19 ± 12</td>
<td>14 ± 9</td>
<td>18 ± 4</td>
<td>2 ± 1.0</td>
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<tr>
<td>CTX (µg/l)</td>
<td>4.4 ± 1.6</td>
<td>2.9 ± 1.8</td>
<td>2.35 ± 1.2</td>
<td>1.75 ± 0.7</td>
<td>1.26 ± 0.6</td>
<td>2 ± 0.9</td>
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<td>(4.0–2.6)</td>
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<td>Sevelamer (µg/l)</td>
<td>45 ± 0.5</td>
<td>25 ± 0.5</td>
<td>25 ± 0.5</td>
<td>15 ± 0</td>
<td>0</td>
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<tr>
<td>(40–50)</td>
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</table>

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 PTH (Roche Elecsys) were recorded monthly before dialysis, and bone turnover markers every 3 months: phosphorus and iPTH (25-OH vitamin D) was prescribed at a dose 40–166 nmol/l, range 40–166 nmol/l, 100 µg. Serum levels of 25-OH vitamin D remained stable before dialysis and on alternate days when prescribed at a dose <30 µg/day. All patients received daily oral cholecalciferol in order to correct 25-OH vitamin D deficiency. Serum levels of 25-OH vitamin D remained stable at baseline and after 12 months (81 ± 50, range 40–166 nmol/l vs 108 ± 52, range 40–176 nmol/l). The standard phosphate binder was sevelamer and no oral calcium was given. Alfacalcidol dose was tapered according to PTH, Ca × P and bone markers level. Criteria for cinacalcet weaning were PTH <100 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 1. 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.

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

Centre de Rein Artificiel, Guillaume Jean Tassin la demi-lune, France Charles Chazot Email: guillaume-jean-erat@wanadoo.fr Bernard Charra

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