

3. Johnson RJ, Segal MS, Srinivas T *et al.* Essential hypertension, progressive renal disease, and uric acid: a pathogenetic link? *J Am Soc Nephrol* 2005; 16: 1909–1919

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### Association of Henoch–Schönlein purpura and Graves' disease

Sir,

We read with interest the article 'Henoch–Schönlein purpura associated with propylthiouracil overdose' by Lee *et al.* [1]. They reported the first case of a patient with Graves' disease who developed Henoch–Schönlein purpura (HSP) after a propylthiouracil overdose. We wondered whether HSP would have occurred if she had maintained the prescribed dosage. Although a causal relationship between HSP and Graves' disease or propylthiouracil use is unclear, we recently reported the case of a patient who developed Graves' disease during ciclosporin treatment for severe HSP nephritis [2], and speculated that these two diseases might be related. In the past 10 years, we have experienced two additional cases of HSP associated with Graves' disease.

#### Patient 1

A 28-year-old previously healthy woman presented with a skin eruption starting at 12 weeks gestation. Physical examination revealed palpable purpura of the lower extremities extending to the knees, but otherwise was unremarkable. Her past medical history revealed that she had been diagnosed with Graves' disease 4 years previously and had been treated with propylthiouracil for 3 years. One year ago, she came to maintain euthyroid state without propylthiouracil treatment. Family history revealed that her mother and younger sister had hypothyroidism. Her blood pressure was normal, and laboratory tests, including complete blood count, BUN, creatinine, aminotransferases, alkaline phosphatase, urinalysis, hepatitis B and C and human immunodeficiency virus serologies, were negative or normal. Erythrocyte sedimentation rate (ESR) was 59 mm/h and serum IgA level 429 mg/dl (normal reference values <300). About 1 month later, she experienced a cutaneous flare, which persisted several weeks, without evidence of renal disease or hypertension.

#### Patient 2

A 12-year-old girl was admitted to the hospital with a history of fatigue, abdominal pain, vomiting and dark urine. On admission, she had arthralgia, oedema and a palpable purpuric rash on the lower extremities. Past medical history revealed that a tonsillectomy had been performed at the age of 9 years, and Graves' disease was diagnosed at the age of 10 years. She was commenced on propylthiouracil and subsequently remained euthyroid state on a stable dose of propylthiouracil. She was hypertensive, and laboratory investigations were: ESR 59 mm/h, BUN 74 mg/dl, serum creatinine 4.8, serum albumin 3.2 g/dl, cholesterol 177 mg/dl, serum IgA 291 mg/dl, creatinine clearance 13 ml/min/1.73 m<sup>2</sup>,

antinuclear antibody negative and anti-ds DNA antibody negative. Urinalysis showed many RBCs/HPF and proteinuria 30 mg/dl. She was diagnosed as having acute nephritic type of HSP nephritis, and treated with methylprednisolone pulse therapy followed by prednisolone, calcium-channel blocker (Madipine) and propylthiouracil. She achieved complete remission of HSP nephritis including resolution of haematuria and proteinuria at 1.4 years of follow-up. About 3 years after the development of HSP, the patient is still in complete remission without recurrence of vasculitis.

Our cases suggest that HSP may develop in a patient with Graves' disease receiving the usual dose of propylthiouracil or no treatment. Therefore, it is likely that not only propylthiouracil use but also Graves' disease itself may be a triggering factor for the development of HSP in susceptible individuals. However, a large prospective study may further elucidate the clear relationship between HSP and Graves' disease or propylthiouracil use.

*Conflict of interest statement.* None declared.

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1. Lee JE, Jeon CH, Chang DK, Lee MS, Oh HY. Henoch–Schönlein purpura associated with propylthiouracil overdose. *Nephrol Dial Transplant* 2006; [Epub ahead of print]
2. Shin JI, Park JM, Lee JS, Kim DH, Jeong HJ. Development of Graves' disease during cyclosporin treatment for severe Henoch–Schönlein nephritis. *Nephrol Dial Transplant* 2005; 20: 2014–2015

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### Cloudy dialysate due to lercanidipine

Sir,

Cloudy dialysate in a patient on continuous ambulatory peritoneal dialysis (CAPD) results from a diverse group of aetiologies. Most commonly, it reflects an increased number of leucocytes secondary to bacterial peritonitis. However, an occasional patient presents with leucocyte-negative, cloudy dialysate. Dihydropyridine type calcium channel blockers would cause turbid peritoneal dialysate [1]. In our study, we investigated whether patients on CAPD having lercanidipine (dihydropyridine type calcium channel blocker) treatment had turbidity of peritoneal dialysate. We evaluated 23 patients with end stage renal disease on CAPD, 14 female and nine male, with the mean age of 45.3 ± 17.6 years. The mean time on CAPD was 15.9 ± 11.6 months. The patients who enrolled in this study were given lercanidipine 5 mg/day. Peritoneal dialysate was evaluated in terms of turbidity of dialysate on the first day. Our diagnostic criteria for drug-induced turbidity of dialysate as its development within the first day after the treatment, absence of clinical symptoms of peritoneal inflammation and the peritoneal dialysate containing normal