physical examination, her abdomen was tender in the left iliac fossa with no signs of peritonism. The abdominal film was unremarkable. Abdominal ultrasound showed an ileocolic intussusception (Figure 1).

She remained clinically and biochemically nephrotic at this time. She entered the remission phase of nephrotic syndrome between Days 7 and 10 after the initiation of therapy which coincided with the complete resolution of her abdominal pain.

Gastrointestinal disturbances are frequently encountered in the course of nephrotic syndrome. The differential diagnosis considered included renal vein thrombosis, peptic ulcer disease and subacute bowel obstruction.

Fortuitously, at the time of ultrasonography, the patient developed an episode of colicky abdominal pain, and the intussusception could be demonstrated.

Ultrasonography is the diagnostic tool of choice to detect intussusception, although it can be operator dependent or limited by body habitus.

Intussusception causes 'telescoping' of the bowel due to a lead point in the bowel, which in this case is due to incoordinate gut motility and bowel wall oedema.

Intussusception is not infrequently described in the paediatric literature, but the usual cause in adults is secondary to a bowel tumour, which acts as a lead point for the invagination of the bowel [2]. Treatment of the underlying nephrotic syndrome resulted in resolution of the intussusception without the need for any intervention [3,4]. Infusions of albumin have also been described [5].

We conclude that nephrologists should consider intussusception in the differential diagnosis of abdominal pain in the setting of nephrotic syndrome as early recognition may improve prognosis.

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1. Palmer SC, Nand K, Strippoli GF. Interventions for minimal change nephropathy in adults with nephrotic syndrome. *Cochrane Database Syst Rev* 2008; 23
2. Asai K, Tanaka N, Tsumura K *et al*. Intussusception of the small bowel associated with nephrotic syndrome. *Pediatr Nephrol*. 2005; 20: 1818–1820
3. Del-Pozo G, Albillos JC, Tejedor D *et al*. Intussusception in children: current concepts in diagnosis and enema reduction. *Radiographics* 1999; 19: 299
4. Ko HS, Schenk JP, Troger J *et al*. Current radiological management of intussusception in children. *Eur Radiol* 2007; 17: 2411
5. Cho MH, Hwang HH, Coe BH *et al*. The reversal of intussusception associated with nephrotic syndrome by infusion of albumin. *Pediatr Nephrol* 2009; 24: 421–422

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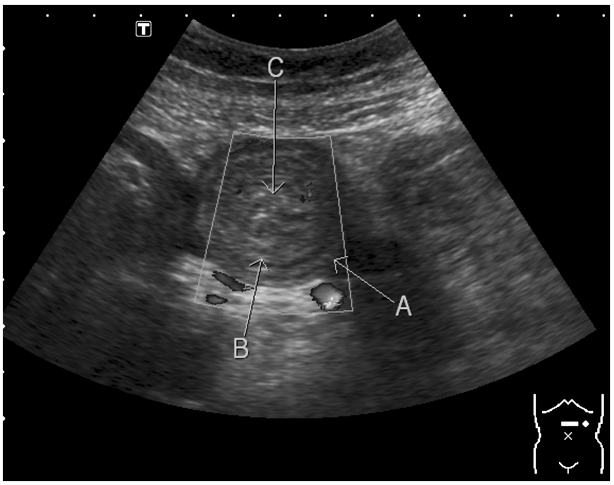

Fig. 1. Transverse section of intussuception. Bull's-eye sign/target/crescent-in-doughnut. **A**. Intussicepiens. Concentric rings of alternating hypoechoic and hyperechoic layers. **B**. Returning limb of intussuceptum. **C**. Mesentery of intussuceptum. Central hyperechoic portion.

Advance Access publication 24 June 2010

Obstructive uropathy due to inflammatory abdominal aortic aneurysm occurring 18 years after surgical repair of an atherosclerotic aneurysm

Sir,

Chronic periaortitis has a wide range of causes [1]. Obstructive uropathy associated with chronic periaortitis complicating abdominal aortic aneurysm (AAA) repair is well recognized and tends to occur early within 12 months of AAA repair. It is unusual for this to occur many years after surgery.

A 71-year-old male was referred to the renal team after an emergency hospital admission by the medical team with acute kidney injury. Serum creatinine was 774 μmol/L (eGFR 7 mL/min/L), having been 140 μmol/L (eGFR 47 mL/min/L) 4 months previously.

There was history of an atherosclerotic AAA open repair with an aortic graft in 1990. In 1994, the patient had a surgical exploration and evacuation for retroperitoneal haematoma.

In 1995, the patient underwent surgical repair for leaking AAA. The proximal half of the graft was resected and replaced with a second Gelsoft graft (rifampicin soaked).

In September 2008, the patient was admitted to our hospital with malaise, anorexia and oliguria.

On examination, there was a pulsatile abdominal mass just lateral (left) to his midline laparotomy scar. An abdominal CT scan showed a large infrarenal AAA measuring 9.2 cm. The right kidney was of normal size but severely hydronephrotic with a hydroureter extending down to the level of the aortic bifurcation. The left kidney was small and also hydronephrotic (Figure 1). There was extensive fibrosis noted at the level of the aortic bifurcation associated with obstructive uropathy (Figure 2).

The patient remained hypotensive with a poor urine output. He subsequently deteriorated and died 4 days after admission.

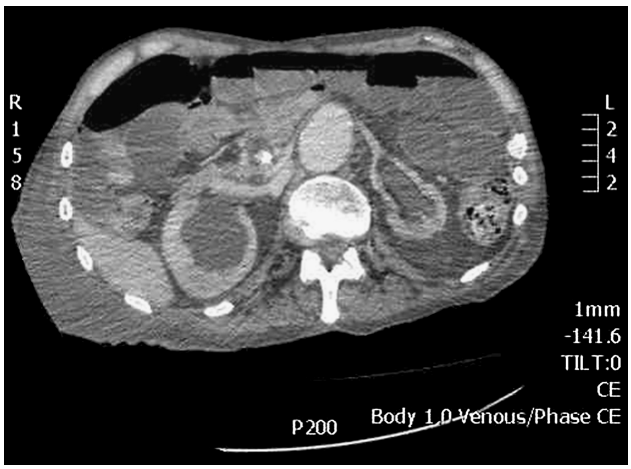

Fig. 1. Bilateral hydronephrotic kidneys.

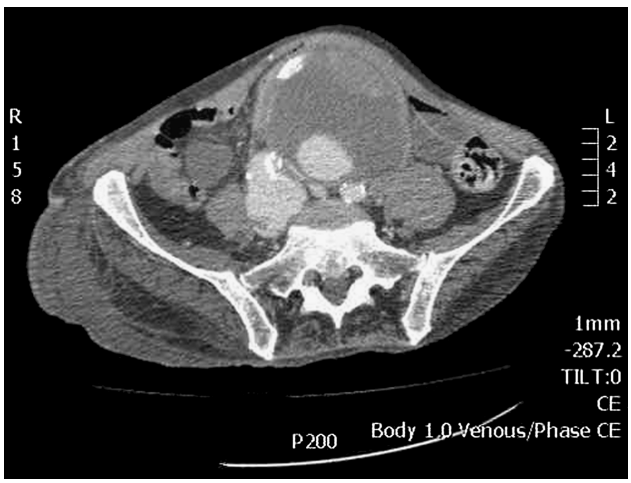

Fig. 2. A large infrarenal abdominal aortic aneurysm with extensive fibrosis.

Early hydronephrosis developing within the first year occurs in 10–20% of patients following surgical graft repair of AAA. The most probable cause for this is mechanical due to the compression of the ureter against the native iliac artery from the anteriorly placed graft [2].

In a prospective study of 101 patients who underwent aortofemoral and aortoiliac reconstructive surgery, 12% of patients developed mild to moderate hydronephrosis. All patients were asymptomatic, and the obstruction resolved spontaneously in 10 of 11 patients within 3 months of onset.

The incidence of delayed hydronephrosis occurring 1 year or more post surgical repair is unknown. This complication may be relatively common after reconstructive vascular surgery as in our patient, especially in association with infected grafts [3]. The mechanism for the development is not fully understood. The most widely held view is that this complication develops secondary to an inflammatory process causing fibrosis [4].

The long time interval is unusual and suggests perhaps a different mode of inflammatory pathways compared to what is commonly seen in early obstructive uropathy associated with surgical graft repair of AAA. More research is needed to elucidate the mechanisms underlying chronic periaortitis [5].

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1. Parums DV. The spectrum of chronic periaortitis. *Histopathology* 1990; 16: 423–431
2. Schein M, Saadia R. Ureteral obstruction after abdominal aortic surgery. *Am J Surg* 1991; 162: 86–89
3. Goldenberg SL, Gordon PB, Cooperberg PL *et al*. Early hydronephrosis following aortic bifurcation graft surgery: a prospective study. *J Urol* 1988; 140: 1367–1369
4. Frusha JD, Porter JA, Batson RC. Hydronephrosis following aortofemoral bypass grafts. *J Cardiovasc Surg (Torino)* 1982; 23: 371–377
5. Alessandra P, Augusto V. Chronic periaortitis: a fibro-inflammatory disorder. *Best Pract Res Clin Rheumatol* 2009; 23: 339–353

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Validity of haemoglobin A1c and glycoalbumin for an appropriate evaluation of glycaemic control in Japanese diabetic patients with chronic renal failure

Sir,

Although the validity of glycoalbumin (GA) instead of haemoglobin A1c (HbA1c) measurement in patients on haemodialysis (HD) has recently been discussed by some investigators [1,2], an appropriate indicator for glycaemic control in patients with pre-dialysis chronic renal failure (CRF) has only rarely been reported [1]. The application of erythropoietin (EPO) for the treatment of renal anaemia increases the proportion of young erythrocytes over old erythrocytes in peripheral blood [3], and HD procedure per se causes the mechanical destruction of red blood cells (RBC). These conditions may reduce the half-life of HbA1c. On the other hand, GA is affected by an accelerated turnover of albumin in the case of nephrotic syndrome frequently observed in pre-dialysis patients due to a massive loss of protein into the urine [2]. The aim of the present study is to evaluate the validity of both indicators in Japanese patients with diabetes separately according to their CRF stage, either undergoing HD or not and either being treated with EPO or not.

Methods

Four hundred and seventy-five patients with diabetes (279 males, 33 type 1 diabetes, 63 ± 13 years old, mean ± SD) were enrolled from November 2007 to June 2009 at Kurume University Hospital and Ito Clinic, in which 97 were treated with maintenance HD (Group HD, no haemodialysis filtration), 112 had impaired renal function with their serum creatinine (S-Cr) levels >1.2 mg/dL in male and 0.9 mg/dL in female subjects (Group RD), and 266 had normal renal function (Group N). Estimated GFR (eGFR) was calculated according to the formula modified by the Japanese Society of Nephrology in 2008 [eGFR for male = $194 \times \text{S-Cr}^{-1.094} \times$