cardiac auscultation. Bilateral crackles were determined in lungs.

Her laboratory findings were as follows: serum sodium 112 mmol/l, chlorine 77 mmol/l, urea 442 mg/dl, creatinine 7.5 mg/dl, haemoglobin 6.5 g/dl, leucocytes 30 000, arterial blood gas pH 7.1, bicarbonate 8 mmol/l, pCO₂ 22 mmHg, C-reactive protein (CRP) 41 mg/l. Due to poor general condition of the patient, with acidosis, haemodialysis was started. Ultrasonographic examination of the abdomen revealed the situs inversus and polycystic kidney disease. The CT of the abdomen and thorax CT was performed (Figures 1 and 2).

To our knowledge, this is the first case of KS and polycystic kidney disease reported in the literature.

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A rare case of perinephric urinoma due to idiopathic retroperitoneal fibrosis

Sir,
Urinoma as a consequence of urine extravasation is a rare complication of severe urinary tract obstruction, where it is most frequently in the fetus and the newborn [1]. However, urinoma in adults is usually developed by the traumatic urinary tract injuries [2]. Retroperitoneal fibrosis is an uncommon disease that is characterized by chronic inflammatory process with progressive fibrosis of retroperitoneal tissue and entrapment of ureters and other structures located in the retroperitoneum [3,4]. We describe an uncommon case of perinephric urinoma secondary to idiopathic retroperitoneal fibrosis.

Case

A 33-year-old male was admitted to the hospital for severe back pain, general weakness and weight loss over the past 3-month duration. He had back pain continuously radiating to flanks, groins and thighs. His medical history was unremarkable. He had a family history of lymphoma. On examination, he presented with costo-vertebral angle tenderness and right upper abdominal tenderness. Laboratory findings revealed elevated erythrocyte sedimentation rate of 106 mm/h, C-reactive protein of 4.226 mg/dL and serum creatinine of 1.7 mg/dL. Urine analysis and its cytology were negative. Serologies and tumour markers were all negative. The chest X-ray and electrocardiogram were within normal. Computed tomography (CT) revealed bilateral hydronephrosis and right perinephric urinoma with retroperitoneal mass compressing both ureters and aorta (Figures 1 and 2). On the third hospital day, the patient’s serum creatinine level was increased up to 2.2 mg/dL and urine output was markedly decreased. Bilateral percutaneous nephrostomy was performed to relieve both hydronephrosis. A CT-guided needle-punctured biopsy of the retroperitoneal mass was performed to confirm the diagnosis. On the pathological examination, the tissue showed chronic inflammation with large numbers of mononuclear cells within fibroblasts and collagen bundles (3). Tissue Tb PCR examination to exclude tuberculosis revealed to be negative. There was no evidence of malignancy. The radiologic and pathological findings were consistent with idiopathic retroperitoneal fibrosis with perinephric urinoma, which were completely resolved by oral prednisolone treatment. There has been no sign of relapse during the follow-up of 2 years.

Discussion

Retroperitoneal fibrosis is a rare chronic inflammatory disease characterized by the progressive fibrosis of retroperitoneal tissue, which results in entrapment of ureters, and other retroperitoneal organs leading to an unusual back pain [3–5]. Approximately two-thirds of retroperitoneal fibrosis cases are considered to be idiopathic and to be related with...
autoimmune mechanisms, but the exact pathogenesis still remains uncertain. The other one-third is associated with many different causes such as use of medicines, malignancies, infections, radiotherapy and prior intra-abdominal surgery. Ureteral obstruction causing acute or chronic renal insufficiency is the most common complication of retroperitoneal fibrosis, which develops ureteral involvement in ∼80–100% of patients. However, this is uncommon in urinary leakage due to urinary tract obstruction of retroperitoneal fibrosis [6,7].

The patient in our case had no evidence of neoplasms, autoimmune diseases and drugs related to secondary retroperitoneal fibrosis, where we could conclude that idiopathic retroperitoneal fibrosis was the most reasonable diagnosis. The patient had bilateral ureteral involvement with hydronephrosis, which resulted in right perinephric urinoma. His major symptom was severe unusual low back pain, and it resulted from rapidly progressive hydronephrosis and urinoma. CT was the study of choice in the diagnosis of retroperitoneal fibrosis and urinoma (Figure 1). Treatment of retroperitoneal fibrosis is targeted to improve inflammatory process to slow down fibrosis and to relieve the obstruction of ureters or other retroperitoneal structures (Figure 2). Although there is no guideline about the treatment of idiopathic retroperitoneal fibrosis, there were many case reports and retrospective studies that recommend glucocorticoid as a primary treatment [3,8]. For those who are refractory to steroid therapy, immunosuppressants such as cyclophosphamide, azathioprine, methotrexate, mycophenolate mofetil and tamoxifen are recommended as an alternative [9].
In conclusion, urinoma is an uncommon complication of idiopathic retroperitoneal fibrosis, which develops acutely into unusual back pain. Either CT or magnetic resonance imaging (MRI) should be considered to define the cause of vague, non-specific and severe back pain or flank pain.

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Long-term effects of calcium antagonists on augmentation index in hypertensive patients with chronic kidney diseases

Sir,

In 2004, the Japanese Society of Hypertension recommended calcium channel blockers (CCBs) as second line drugs, with the renin–angiotensin (Ang) system (RAS) inhibitor as the first choice, for the treatment of hypertension associated with chronic kidney disease (CKD). We reported that augmentation index (AI) is related to proteinuria in CKD patients, and that RAS inhibition preserves arterial compliance in CKD [1,2]. However, the effects of CCBs on arterial stiffness remain unclear among CKD patients.

A prospective comparative study was performed between 26 non-diabetic CKD patients treated with amlodipine and 27 patients on benidipine (supplemental methods). Patient backgrounds including the prescription of the RAS inhibitor did not differ between groups (supplemental table). Brachial blood pressure was controlled equally well in both groups for a year (supplemental figure). A year later, body weight (to 59 ± 11 kg, P < 0.05) and estimated glomerular filtration rate (eGFR) were decreased, and AI was increased without changes in proteinuria (Figure 1) in the amlodipine group. However, in the benidipine group, either eGFR, body weight or AI was not altered, but proteinuria was reduced.

In renal tissue, L-type calcium channels are only found in afferent arterioles, while N-type and T-type calcium channels are localized in both afferent and efferent arterioles [3]. Amlodipine blocks L-type and N-type calcium channels and dilates afferent arterioles much more than efferent arterioles. In contrast, benidipine that inhibits L-type and T-type calcium channels, dilates both afferent and efferent arterioles and reduces glomerular pressure. We have demonstrated that efferent arteriolar constriction is mediated by inositol trisphosphate-induced calcium mobilization and calcium entry through transient receptor potential (TRP) channels [4]. T-type CCBs inhibited AngII-induced calcium mobilization rather than calcium entry in efferent arterioles [3]. TRP channels possess molecular similarity with voltage-dependent calcium channels, but they lack the structure of voltage-sensor, gating independently of voltage. It is possible that some CCBs including benidipine inhibit calcium entry through TRP channels into efferent arteriole.

Increasing AI elevates central blood pressure, worsening glomerular hypertension, proteinuria, renal and cardiovascular prognosis [1,2]. Although we would not deny the other possibilities (supplemental discussion), benidipine could reduce oxidative stress on arterial wall by decreasing proteinuria. Albumin passed through slit diaphragm is absorbed by proximal tubular cells. Although a small amount of protein is cleared by acidification [5], oxidative process is involved in dealing a large amount of protein, generating reactive oxygen species that appear to leak from the kidney. This escalation of AI should worsen glomerular hypertension further, forming a vicious circle of progressive kidney damage [1,2].

Our results provided the evidence that benidipine may be superior to amlodipine in renoprotection as the antihypertensive additional to the RAS inhibitor when similar blood pressure levels are attained. Furthermore, the present data suggest that the influence on AI differs among types of CCBs in patients with CKD.

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Supplementary data

Supplementary data is available online at http://ndt.oxfordjournals.org.

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