A male with extensive renal vein thrombosis

L. Tonneijck*, M. Schouten, M.C. Weijmer, C.E.H. Siegert

Department of Nephrology, St. Lucas Andreas Hospital, Amsterdam, the Netherlands, tel.: *corresponding author: tel.: +31(0)20 5108770, fax: +31(0)20 6838771, e-mail: l.tonneijck@gmail.com

CASE REPORT

A 48-year-old male presented with right-sided flank pain of three weeks duration, which had radiated to the abdomen and groins during the last 24 hours. He reported no haematuria. His medical history included myocardial infarction (2010) and urolithiasis. Physical examination demonstrated mild pretibial oedema. His blood pressure was 111/61 mmHg. Laboratory investigation showed leucocytes 12.9 (4-10) x 109, C-reactive protein 167 (<10) mg/l, albumin 26 (38-52) g/l and creatinine 111 (60-110) μmol/l with an estimated glomerular filtration rate (eGFR) of 62 (60-125) ml/min. Ultrasound imaging did not show any signs of urolithiasis, appendicitis or diverticulitis. Additional computed tomography (CT) scanning demonstrated a thrombus in the right renal vein extending into the inferior vena cava and both iliac veins (figures 1A and 1B), which was confirmed by Doppler ultrasonography. Anticoagulant therapy was started instantly. Additional urinalysis revealed erythrocyturia (189/µl) without dysmorphia and 14.5 grams protein in 24 hours. Clotting assays showed no abnormalities and autoimmune serology and screening for malignancies and chronic infections were negative.

WHAT IS YOUR DIAGNOSIS?

See page 484 for the answer to this photo quiz.

Figure 1. (A) Contrasted CT scan showing a hypodense mass in the right renal vein suggestive for thrombus. (B) Thrombus extending into the inferior vena cava and both iliac veins





ANSWER TO PHOTO QUIZ (PAGE 480)

A MALE WITH EXTENSIVE RENAL VEIN THROMBOSIS

DIAGNOSIS

The patient was diagnosed with nephrotic syndrome complicated by renal vein thrombosis (RVT). He commenced prednisolone 60 mg once daily for a presumed diagnosis of idiopathic membranous nephropathy (IMN) or minimal change disease (MCD). Sodium restriction and subsequent angiotensin converting enzyme inhibitor (ACE-I) treatment were installed consecutively. After ten weeks of therapy, control of proteinuria was inadequate and tacrolimus 5 mg once daily was added, since both IMN and MCD have shown favourable clinical responses to this treatment. This led to a significant reduction in the proteinuria (0.46 grams in 24 hours). After three months, CT scanning showed almost complete resolution of thrombus, after which anticoagulant treatment was stopped and a kidney biopsy was performed, whereupon the diagnosis of IMN was confirmed.

The nephrotic syndrome is defined by a urinary protein level exceeding 3.5 grams in 24 hours and is associated with oedema, hypoalbuminaemia, hyperlipidaemia, and infectious and thromboembolic complications.3 Thromboembolic disease is a serious complication in patients with nephrotic syndrome and may arise from preferential loss of proteins which inhibit systemic haemostasis, increased synthesis of prothrombic factors and local activation of glomerular haemostasis.4 Venous thromboembolic complications in nephrotic syndrome include deep venous thrombosis, pulmonary embolism and RVT. RVT may be associated with nephrotic syndrome of any aetiology but most commonly occurs in patients with membranous nephropathy, with a prevalence of 37%.4 Symptoms of RVT can include acute flank pain, gross haematuria, and deterioration of renal function but is usually asymptomatic.⁴⁻⁶ Doppler ultrasonography is the primary modality for detection of RVT, alternatives being contrast-enhanced CT and magnetic resonance imaging.⁶ Anticoagulant therapy is the treatment of choice⁴ in addition to treatment of the proteinuria (ACE inhibition, immunomodulation) and of the underlying disease if present.

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