Dear Sir,

Renal vein thrombosis (RVT) is one of the most serious complications of the nephrotic syndrome, and occurs in 5-62% of cases with an overall incidence of 20% [1, 2]. Its pathophysiology is poorly understood, even though various coagulation abnormalities have been described in the nephrotic syndrome. Two RVT patient subgroups have been recognised. Those with acute RVT have clinical symptoms, e.g. flank pain and macroscopic hematuria, whereas those with chronic RVT can only be characterized by their nephrotic state and absence of acute symptoms; asymptomatic cases far outnumber symptomatic cases [1,2].

The diagnosis of RVT has traditionally relied on invasive methods such as renal venography and venous phase of renal arteriography [3, 4]; computed tomography (CT) has also been recommended recently [5, 6]. Urinary fibrin-fibrinogen products above 5 µg/min and renal vein Doppler ultrasonography have also been proposed for identifying nephrotic patients who require renal vein examination [7].

The new noninvasive method magnetic resonance imaging (MRI) can be used to assess vascular patency and to visualize thrombi without the need of contrast agent administration. MRI is routinely used to diagnose abdominal venous thrombosis [8], and to detect and assess the extent of neoplastic RVT in preoperative staging of renal cell carcinoma [9].

A retrospective study of 20 proteinuric patients by Tempany et al. [10] showed that renal vein assessments by MRI were comparable to those using Doppler-ultrasonography or renal venography; this suggests that MRI may be a valuable noninvasive solution to the problems of diagnosing RVT.
We evaluated renal vein MRI for the diagnosis of RVT in a prospective study of adult patients with the nephrotic syndrome. We also investigated technical problems and potential artefacts in visualizing renal veins.

The study included 23 adult nephrotic patients (plasma albumin < 30 g/l, protein-uria > 3 g/day) who had been referred to our institution; they were aged between 17 and 70, and had urinary protein output from 5 to 25 g/day and plasma creatinine concentrations from 80 to 500 µmol/l. Histological diagnosis showed that 9 had idiopathic nephrotic syndrome (minimal change disease + segmental glomerulosclerosis); 7 had membranous glomerulonephritis; 2 had membranoproliferative glomerulonephritis; and 5 had other types of glomerulonephritis. None had symptoms or signs of RVT. Informed consent to the study was obtained in every case.

Computed tomography (CT), carried out less than 48 h before MRI, was used as the reference method; the MRI radiologist (A.R.) was not informed of the results of CT.

The same MRI protocol was used to evaluate renal vein patency in all the patients. MRI was performed on a 1.5 Tesla machine (Magnetom SR Siemens, Erlangen, FRG); the following settings were used for axial and sagittal gradient refocused (Flash) images: repetition time (TR), 30 ms; echo time (TE), 10 ms; flip angle, 30°; matrix size, 256 per 256; field of view (FOV), 350 mm (axial views) or 400 mm (sagittal views). These settings allowed images to be obtained in 8 s; consecutive breath-held images were taken to reduce movement artefacts. Slice thickness was 8 mm.

Gradient echo sequences rely on the properties of flowing blood to visualize the vessel lumen, which appears hyperintense. The high signal intensity of the vessels is due mainly to inflow of unsaturated spins into every section. Both renal veins were located on the axial plane. Sagittal images were then acquired from the renal hilum to the inferior vena cava. In axial images, the protons of blood flowing through the renal vein remain within the imaging plane longer than in sagittal images, giving lower signal intensities. Furthermore, the partial volume effect of structures adjacent to the renal veins is more pronounced in axial images.

Obstruction produces low signal intensities in the vein lumen [11, 12], and this is exploited in the diagnosis of abdominal and peripheral vein thrombosis. We therefore diagnosed renal vein thrombosis on the basis of low signal intensities in the lumen on axial and sagittal views. Renal vein patency was deduced from hyperdense lumen signals in either the sagittal views or both the axial and sagittal views. The CT and MRI scans could be interpreted in all the patients studied. Both CT and MRI revealed bilateral vein thrombosis, associated with a thrombus of the inferior vena cava in a patient with membranous glomerulonephritis. CT and MRI both showed renal vein patency in 19 patients. Reliance on MRI axial images alone would have resulted in a false diagnosis of RVT in 3 patients; however, sagittal images clearly showed both renal veins as hyperintense areas.

Our findings, along with those in a previous report [10], suggest that MRI is a valuable noninvasive technique for assessing renal vein patency in patients with nephrotic syndrome. They also show that it is necessary to analyse both axial and sagittal views, as low signals within renal veins can be confused with RVT on axial images alone.

References


