At renal sonography (US), the presence of a perirenal hypoechoic rim (the ‘kidney sweat’ sign) was first described by Yassa et al. [1] as a new sonographic finding in renal parenchymal disease and renal failure. We describe similar findings in nine patients with renal parenchymal disease with or without renal failure, and elaborate further on the clinical, radiological, pathological and biochemical analysis of the perirenal fluid.

MATERIALS AND METHODS

Over a 5-year period, nine patients were found to have an anechoic rim of fluid surrounding the renal parenchyma at sonographic examination performed for various clinical indications. Sonograms were obtained on SSD 2000 (Aloka, Tokyo) or P 700 (Philips Medical Systems, Netherlands) ultrasound equipment using a convex 3.5 MHz transducer.

The clinical and laboratory data are summarized in Table 1. Six patients (cases 2–4, 7–9) had additional duplex Doppler US examinations using a 3.5 MHz Doppler probe (SSD 2000, Aloka, Tokyo). Duplex Doppler examinations consisted of sampling the intrarenal segmental and interlobar arteries, and calculations of the resistive index (RI) values from three to five spectral waveforms using the Aloka computer measurement; RI was defined as the ratio

$$RI = \frac{\text{psv} - \text{medv}}{\text{psv}}$$

RI = (peak systolic velocity—minimum end diastolic velocity)/(peak systolic velocity)

Five patients (cases 1, 2, 7–9) had unenhanced CT of the kidneys followed by contrast-enhanced CT in two patients (cases 1, 2) with normal serum creatinine level, performed on whole body scanner (Philips CX/Q, Netherlands) with a data acquisition time of 2.8 sec. (120 Kv, 110 mA) and contiguous 10 mm sections through the region of interest. Dynamic CT was performed immediately after an intravenous injection of 100 ml of non-ionic contrast medium containing 300 mg iodine per ml (iobitridol 300 mgI/ml;
Table 1 – Summary of clinical and laboratory data, histopathological diagnosis, fluid biochemistry and clinical course

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/sex</th>
<th>Clinical and laboratory findings</th>
<th>Biopsy</th>
<th>Fluid analysis</th>
<th>Clinical course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>23/F</td>
<td>Right flank pain, history of arthritis, high ESR, positive ANA and anti ds DNA</td>
<td>Kidney biopsy: lupus nephritis</td>
<td>( – )</td>
<td>Right renal vein thrombosis</td>
</tr>
<tr>
<td>2</td>
<td>12/F</td>
<td>Increase in abdominal girth</td>
<td>Kidney biopsy: focal and segmental glomerulosclerosis</td>
<td>Creatinine 0.7 mg/dl Osmolality 255 mosmol/l Protein 3 g/l Glucose 66 mg/dl</td>
<td>Progressive renal failure</td>
</tr>
<tr>
<td>3</td>
<td>62/M</td>
<td>Weight loss, fatigue, Bence Jones proteins in urine, concentric hypertrophy of LV on echocardiography</td>
<td>Skin and bone marrow biopsies: multiple myeloma with secondary amyloidosis</td>
<td>( – )</td>
<td>Passed away</td>
</tr>
<tr>
<td>4</td>
<td>67/M</td>
<td>Bilateral lower extremity oedema, anaemia, low platelets, diffuse thickening of LV wall on echocardiography</td>
<td>( – )</td>
<td>( – )</td>
<td>Shrunken small kidneys/passed away</td>
</tr>
<tr>
<td>5</td>
<td>50/M</td>
<td>Nausea and vomiting, hypertension, anaemia</td>
<td>( – )</td>
<td>( – )</td>
<td>Chronic renal failure on dialysis</td>
</tr>
<tr>
<td>6</td>
<td>71/M</td>
<td>Abdominal pain, vomiting, fever, anaemia, known multiple myeloma</td>
<td>Bone marrow: multiple myeloma</td>
<td>( – )</td>
<td>Passed away</td>
</tr>
<tr>
<td>7</td>
<td>58/M</td>
<td>Diabetes, sepsis, hypertension, generalized oedema</td>
<td>( – )</td>
<td>( – )</td>
<td>Passed away</td>
</tr>
<tr>
<td>8</td>
<td>20/F</td>
<td>Leg oedema, eye puffiness</td>
<td>Refused kidney biopsy</td>
<td>( – )</td>
<td>Improved on salt restriction diet</td>
</tr>
<tr>
<td>9</td>
<td>23/F</td>
<td>History of pre-eclampsia, leg oedema, anaemia</td>
<td>Kidney biopsy: non-diagnostic</td>
<td>Glucose 96 mg/dl BUN 14 mg/dl Creatinine 0.6 mg/dl LDH 12 IU/L Protein 14 g/l Na⁺ 140 K⁺ 3.6 Cl⁻ 111 CO₂ 28</td>
<td>Lost to follow-up</td>
</tr>
</tbody>
</table>

ESR, erythrocyte sedimentation rate; LV, left ventricle; ANA, antinuclear antibodies; anti ds DNA, anti double stranded DNA; (–), not performed.
Xenetix, Guerbet, France) administered as a bolus. One patient (case 9) had MRI performed on a 0.5 T unit (Signa, General Electric Medical Systems, Milwaukee, WI, U.S.A.). A spin echo pulse sequence was used to obtain T1-weighted images with a repetition time (TR) of 740 ms and echo delay time (TE) of 11 ms and T2-weighted images were obtained by fast spin echo TE/TR 90/6666. Three patients (cases 1, 2, 9) had a kidney biopsy, two patients (cases 3, 6) had skin and bone marrow biopsies. Aspiration of the perirenal fluid was performed in two patients (cases 2, 9) after informed consent. In case 2 the perirenal fluid was misinterpreted as a renal cortical cyst for which the patient underwent a surgical exploration, but the fluid reaccumulated in the subcapsular space after surgery, while in case 9 the perirenal fluid was aspirated under CT guidance. Biochemical analysis of the aspirated fluid samples was obtained.

RESULTS

The histopathology, biochemical analysis of the fluid, diagnosis and clinical course are summarized in Table 1. Five patients (cases 1–4, 7) presented with a nephrotic syndrome. Ascites was present in four patients (cases 2, 4, 6, 9). Renal failure (i.e. a serum creatinine level greater than 2 mg/dl) was present in four patients (cases 3–6), who had a poor outcome and clinical course. Two patients (cases 2, 7) had a serum creatinine level less than 2 mg/dl but low creatinine clearance compatible with decreased renal function. Five patients (cases 1, 2, 7–9) had a normal serum creatinine level; one of them (case 2) later developed progressive renal failure. Proteinuria was present in eight patients (cases 1–4, 6–9). All patients had increased cortical echogenicity compared with liver tissue at sonography, compatible with renal parenchymal medical disease. Six patients (cases 1, 2, 4, 7–9) had enlarged kidneys greater than 13 cm in length, while three patients (cases 3, 5, 6) had normal size kidneys. Two patients with nephrotic syndrome (cases 1, 4) developed renal vein thrombosis; in case 1 the renal vein thrombosis was unilateral, while case 4 had bilateral renal vein thrombosis that had led to small shrunken kidneys measuring less than 9 cm in length. The corticomedullary differentiation (CMD) on sonography, was preserved but with poor definition in six patients (cases 1, 4, 6–9), and three patients (cases 2, 3, 5) had loss of CMD. Of the six patients investigated by duplex Doppler examinations, three patients (cases 2, 8, 9) had normal mean RI values less than 0.70 (range 0.54–0.70; mean 0.65), while the other three patients (cases 3, 4, 7) had markedly elevated RI values (range 0.81–0.92; mean 0.86), indicating severe impedance to blood flow from underlying parenchymal disease. Patients with normal RI values were young, with no renal failure and had a better prognosis. Patients with elevated RI values were older, had renal failure and a poor prognosis.

Three sonographic patterns or grades of perirenal fluid were observed:

(1) Grade 1 consisted of a small rim or thin layer of fluid ranging from 3 to 10 mm in thickness (Fig. 1) surrounding part of the renal parenchyma, mainly the anterior aspect of the kidney and its lower pole (cases 1, 3, 5–7), or completely surrounding the kidney in a circumferential pattern (case 4).

(2) Grade 2 consisted of a moderate amount of fluid ranging from 1 to 5 cm in thickness (Fig. 2) with evidence of strands and indentations of the renal parenchyma (cases 8, 9).

(3) Grade 3 consisted of a large fluid collection greater than 5 cm in thickness surrounding the kidney (Fig. 3) (case 2), simulating a cyst.

The perirenal fluid was bilateral in five patients (cases 3, 4, 6–9), unilateral in one patient (case 1), while two patients (cases 2, 5) had solitary kidneys surrounded by fluid. Case 1 had nephrotic syndrome, developed right renal vein thrombosis without evidence of perirenal fluid around the right kidney, but had unilateral left perirenal fluid. The fluid was invariably subcapsular in all patients, subcapsular and extracapsular in three patients (cases 2, 8, 9), and associated with ascites in four patients (cases 2, 4, 6, 9). In patients with grade 1 sonographic fluid collection pattern, the perirenal fluid was less conspicuous on CT than at US. However, CT demonstrated to better advantage than US: the separation of the capsule from the renal tissues (cases 2, 8, 9) (Fig. 3c); the presence of fluid across the midline in the retroperitoneum surrounding the aorta and inferior vena cava (cases 2, 7–9) (Fig. 2b); generalized body oedema (case 7); and thickening of the renal fascia (cases 8, 9). Two patients (cases 3, 7) had bilateral pleural effusions. The attenuation values of the perirenal fluid on CT ranged from −14 to +10.6 Hounsfield Units (mean, 4.6 HU). One patient (case 9) had an MR examination which showed low signal intensity fluid on T1-weighted images, and high signal intensity fluid on T2-weighted images isointense with cerebrospinal fluid and bile. No correlation was found between the degree of perirenal fluid and renal failure; indeed, grade 1 perirenal fluid was observed in older
Fig. 2 – Case 8: 20-year-old girl with leg oedema and proteinuria. (a) Longitudinal sonogram of the right kidney showing a grade 2 perirenal fluid surrounding an enlarged kidney with increased cortical echogenicity and poor corticomedullary differentiation compatible with renal parenchymal medical disease. Note the stranding of the fluid (white arrows) and indentations of the renal parenchyma (arrowheads) by the subcapsular fluid. (b) Unenhanced CT showing bilateral subcapsular perirenal fluid crossing the midline in the retroperitoneum and surrounding the major vessels (white small arrows).

Fig. 3 – Case 2: 12-year-old girl with focal glomerulosclerosis. (a) Longitudinal sonogram of the left kidney showing a grade 3 perirenal fluid collection surrounding a poorly differentiated kidney. (b) Contrast-enhanced CT demonstrating fluid around left kidney. (c) Follow-up contrast-enhanced CT obtained after surgery, showing a functioning enlarged left kidney with residual or recurrent perirenal fluid in the subcapsular space separating the capsule (small white arrows) from the renal tissues.
patients with renal failure and poor outcome, while grades 2 and 3 were observed in young patients with no renal failure and better outcome. Biochemical analysis of the aspirated fluid (cases 2, 9) revealed a transudate with characteristic values of its constituents similar to blood serum.

**DISCUSSION**

Fluid in the perirenal space may be infrequently seen at cross-sectional imaging of patients with abnormalities that affect the kidneys or the adjacent retroperitoneal structures and organs. The most common fluid accumulations are blood, pus and urine, depending on the causative renal or extrarenal disease. The most common fluid accumulations are blood, pus and urine, depending on the causative renal or extrarenal disease process [2]. Perirenal fluid secondary to renal parenchymal disease was recently described by Yassa *et al.* [1], and a case report by Orofino *et al.* [3] described the presence of unilateral perirenal fluid collection in a patient with membranous nephropathy, nephrotic syndrome and ipsilateral renal vein thrombosis. The authors attributed the development of unilateral perirenal fluid collection to ‘the less direct drainage of the ureteral and capsular veins into the vena cava secondary to renal vein thrombosis, an increase in the intravascular hydrostatic capillary pressure within an exuberant collateral circulation and the decreased oncotic pressure which could have led to fluid extravasation resulting in transudate accumulation in the subcapsular space’ [3]. Yassa *et al.* [1] suggested that the hypoechoic rim was perirenal extracapsular oedema, though this was not proved pathologically. Like Yassa, we have no proven explanation for the development of the perirenal fluid, but we tend to agree with Orofino because biochemical analysis of the fluid revealed a transudate and CT confirmed the presence of subcapsular fluid. We postulate that the perirenal fluid transudate is secondary to a haemodynamic abnormality such as venous obstruction associated with thrombosis of the main renal vein, or veno-occlusive disease with thrombosis of the small intrarenal veins. However, in case 1, unilateral perirenal fluid accumulated around the left kidney with a normal renal vein while the right kidney, with main renal vein thrombosis, had no perirenal fluid. In addition, we performed a histopathologic analysis of the renal biopsy specimens (cases 1, 2) and found no evidence of small intrarenal venous thrombosis. We believe that the perirenal fluid is a transudate that may occur in patients with a sodium retaining state and oedema resulting from a nephropathy. We did not observe this phenomenon in patients suffering from a sodium retention state without nephropathy such as heart failure, cirrhosis or constrictive pericarditis.

At ultrasound, the perirenal fluid appears as anechoic rim. This should not be confused with the hypoechoic rim of solid tissue that may surround the kidneys in patients with thickened renal fascia, perirenal lymphoma, acute cortical necrosis, amyloid deposition and retroperitoneal fibrosis [2,4–8]. The fluid is readily demonstrated by US and can be easily differentiated by US and CT from other fluid collections and lesions in the appropriate clinical scenario. The presence of perirenal fluid is usually bilateral, but may be unilateral in a few cases. The fluid may extend across the midline in the retroperitoneum, at the level of the renal hila through the anatomical communications of the perirenal spaces [9]. There was poor correlation between the three grades of fluid collection and the severity of the underlying renal disease. Poor prognostic factors were elevated RI value, the presence of a grade 1 pattern, the age of the patient, and the associated degree of renal failure.

Yassa *et al.* [1] described this new sonographic finding of perirenal fluid in patients with parenchymal disease and called it ‘kidney sweat’, a new sign of renal failure that was found in 14% of patients with this condition. We found it an infrequent sign correlating with the presence of a sodium retaining state and oedema in patients with intrinsic renal parenchymal disease, with or without renal failure, and not associated with a specific nephropathy. The only common clinical finding between our patients appeared to be the presence of the sodium retaining state. The nephropathies represented in our patients included lupus nephritis, focal and segmental glomerulosclerosis, amyloidosis, renal lymphangiomatosis and diabetic nephropathy. Others [1,3] have reported perirenal fluid in hypertensive nephropathy, heart failure, glomerulonephritis, acute tubular necrosis, drug-induced nephropathy and membranous nephropathy. We do not agree with Yassa *et al.* [1] that it is necessarily a sign of renal failure since it was present in patients with renal parenchymal disease without renal failure.

**REFERENCES**
