Nephrol Dial Transplant (2000) 15: 415–418

Nephrology
Dialysis
Transplantation

Case Report

Unilateral acute renal cortical necrosis (ACN) following skipping with a rope

David Tovbin[1], Sophie Lantsberg[2], Leonid Feldman[1], Irina Rachinsky[2], Liliana Lupu[3] and Yancu Hertzanu[3]

Division of [1]Nephrology, [2]Nuclear Medicine and [3]Diagnostic Radiology, Soroka Medical Center, Beer-Sheva, Israel

Keywords: cortical necrosis; rim sign; exercise; vasoconstriction; renal failure

Introduction

Unilateral renal cortical necrosis is a rare disorder. The following is a case report of unilateral renal cortical necrosis in an 18-year-old normally healthy woman following prolonged jumping over a rope. Extensive work up did not demonstrate any pathology in the large renal arteries or veins and there was no evidence of any systemic disease predisposing to thrombosis or disseminated intravascular coagulation. Though there have been previous reports of exercise-related unilateral renal infarction associated with renal artery dissection [1] or thrombosis [2], to the best of our knowledge this is the first report of unilateral cortical necrosis following physical exercise.

Case

An 18-year-old female was admitted to Soroka Medical Center due to acute right flank and lower quadrant abdominal pain in the previous few hours. Patient described the onset of acute pain 30 min after skipping with a rope, with two subsequent episodes of vomiting. There was no history of any previous illness and patient denied any medication including oral contraceptives. On physical examination patient appeared ill and suffering. Temperature was 37.8°C PR, blood pressure was 125/67 mm Hg, heart rate was 87 beats/min and there was tenderness over right flank and in the right lower abdominal quadrant with normal peristalsis. Complete blood count showed leucocytosis of 16.2×10^9/l with 90% neutrophyles, thrombocytosis of 749×10^9/l, haematocrit of 42.6% and haemoglobin of 14.2 g/dl. PT and PTT were 12 and 35 s respectively. Urinalysis revealed 10 RBC/HPF. Fractional excretion of sodium was 2.3%. Initial serum biochemistry results were urea 5.16 mmol/l, creatinine 124 μmol/l, creatinine phosphokinase of 150 IU/l, aspartate aminotransferase 105 IU/l, alanine aminotransferase 111 IU/l, lactate dehydrogenase 2260 IU/l. After an emergent ultrasound without evidence of renal abscess or hydronephrosis, explorative laparotomy and appendectomy were performed due to the clinical presentation of peritoneal irritation and suspicion of acute appendicitis, with evidence for mesenteric lymphadenitis. Pain persisted and repeated renal ultrasound demonstrated hyperechogenic right kidney (not shown). Colour Doppler showed reduced perfusion and decreased demonstration of intra-renal blood vessels in the right kidney in the presence of patent renal artery and vein, and normal left kidney. A Tc-99m-DTPA (diethylenetriamine pentaacetic acid) renal scan (Figure 1) demonstrated almost complete absence of perfusion and poor function of the right kidney, with normal perfusion, function and excretion in the left

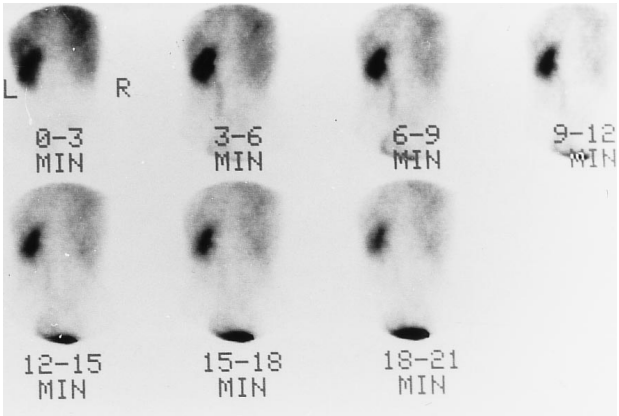

Fig. 1. DTPA renal study. Posterior view: each image represents the sum of 3 min of dynamic sequential images. The study showed an absence of perfusion and function of the right kidney. Perfusion, function and excretion in the left kidney are normal.

Correspondence and offprint requests to: David Tovbin MD, Division of Nephrology, Soroka Medical Center, Beer Sheva 84101, Israel.

kidney. Renal artery or venous thrombosis were strongly suspected and a subsequent renal angiography demonstrated patent main and segmental renal arteries with capsular arterial supply of the upper pole but no appearance of the interlobular or arcuate arteries. The renal parenchyma appeared as a mottled nephrogramme and there was no early filling of the renal veins. Angiography (Figure 2) and venography (not shown) which were completely normal did not show renal artery or venous thrombosis and since the diagnosis still remained unclear, an enhanced helical computerized tomography scan was performed. A region of low attenuation of the right kidney (representing the non-enhancing necrotic cortex), a normal signal from the medulla, a thin rim of enhanced subcapsular region (a rim sign) and no excretion of the contrast medium were found (Figure 3). These results were consistent with acute unilateral renal cortical necrosis. Tc-99m-DMSA (dimercaptosuccinic acid) scan was performed to confirm the diagnosis of cortical necrosis and to assess the extent of the damage. This scan demonstrated a small section of viable cortex of the upper pole surrounded by a wide-rimmed photopenic area indicative of cortical loss which correlated well with the angiographic findings. Protein S was 74.1% (normal 70–140), anti thrombin III 99.7% (normal 82–122), protein C 117.7% (normal 70–130), APC resistance V 2.28 (normal 2–5), C3 96 mg% (normal 80–200), C4 24 mg% (normal 16–47), anti-cardiolipin antibodies levels IgM of 1.1 U/ml (normal 0–7) and IgG of 2.9 U/ml (normal 0–10). Rheumatoid factor and anti-nuclear antibody were negative. Cardiac echocardiography and ventilation–perfusion lung scan were normal. After 36 h serum creatinine level reached 132.6 mmol/l but at 72 h decreased to 97.2 mmol/l and after 5 weeks to 88.4 mmol/l. Since Mag-3 (mercaptoacetylglycine) is actively excreted through the proximal renal tubules and thus superior to DTPA for imaging of kidneys with impaired function, a Mag-3 renal scan was performed 10 days later as a follow-up procedure. This scan demonstrated a small shrunken right kidney with very delayed perfusion and 17% of differential function. An enhanced CT scan 5 weeks later demonstrated a small shrunken right kidney with a preserved nephrographic cortical effect and a clear cortico-medullary differentiation with a slight increase in size of the contra lateral kidney. On ultrasound the right kidney was 7.9 cm and the left kidney was 11.9 cm. In summary, this patient had a unique clinical course of unilateral cortical necrosis following physical exercise.

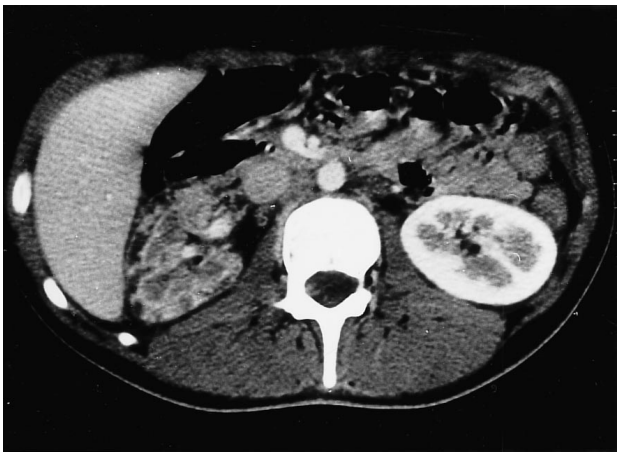

Fig. 3. Early spiral enhanced CT after bolus injection of contrast media shows right kidney with enhancement of the medulla, non-enhancement of the renal cortex, a thin rim of enhanced subcapsular tissue ('rim sign') and lack of excretion of the contrast material into the collecting system. The left kidney is normal.

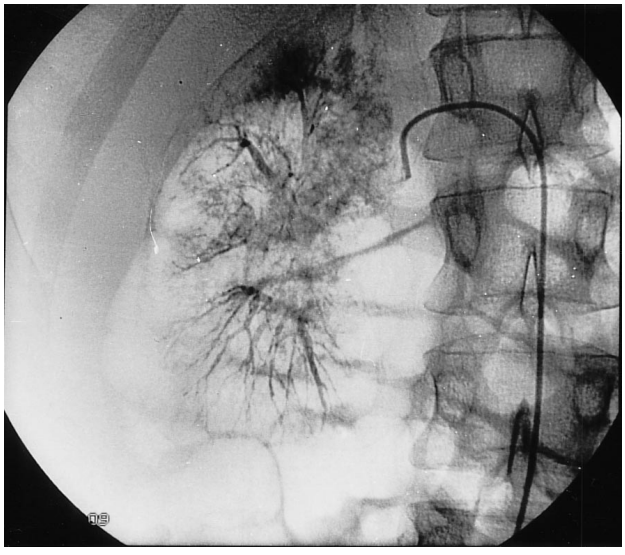

Fig. 2. Selective right renal angiography shows a disorganized intrarenal vasculature, with no filling of the interlobular and arcuate arteries and the presence of capsular vessels of upper renal pole.

Discussion

In this paper we describe an 18-year-old woman with acute unilateral cortical necrosis following physical exercise (rope skipping). Acute appendicitis was suggested by the clinical picture of peritoneal irritation but was excluded by explorative laparotomy, similar to a previous report of a polar infarct of kidney transplant [3]. The differential diagnosis of acute unilateral renal-related pain, reduction in renal function and fever, includes infectious and obstructive processes, but ultrasound did not demonstrate hydronephrosis or abscess. Thus, the differential diagnosis narrowed down to acute unilateral renal vascular events associated with renal necrosis, which was suggested in our patient by the clinical picture of flank pain and increase in serum levels of LDH, AST and alkaline phosphatase. Acute renal vein thrombosis which has been described in an adult male with vomiting induced volume depletion [5], and associated with medullary necrosis in a renal allograft [6], was excluded by renal venography. Renal infarction is usually induced by interference of arterial flow in large and medium renal arteries due to

direct trauma [4] or embolic disease [7]. Unilateral renal infarction post exercise has been described related to arterial thrombosis [2], and renal artery dissection [1], which were excluded in our patient by renal arteriography. Renal artery torsion has been previously described in kidney transplants [8–10], one of which was associated also with lack of abdominal support due to prune belly syndrome [10]. Renal artery spasms have been reported following renal angioplasty [11]. However, in our patient the eventuality of transient arterial disturbances seemed remote, since there was no direct stimulation of renal artery and the only putative aetiology for renal artery torsion could be a change in position induced by the act of skipping. The imaging studies were pathognomic for unilateral ACN with unaffected contra-lateral kidney [12–19]. Acute cortical necrosis is a rare cause of acute renal failure, related to constriction of small intracortical blood vessels with disturbed blood flow to interlobular and afferent arterioles but usually sparing the arcuate arteries. The subsapsular rim of cortex, a thicker layer under the corticomedullary junction, and some parts of the cortex may be spared due to collateral flow through multiple capsular anastomosa via the lumbal, intercostal, inferior adrenal and middle capsular arteries [12]. The appearance of enhanced CT has been shown to be pathognomic and diagnostic for ACN. The four features on CT that typify ACN include: enhancement of the medulla, non-enhancement of the renal cortex, a thin rim of enhanced subcapsular tissue ('rim sign') explained by presence of collateral blood supply, and lack of excretion of the contrast material into the collecting system. This disorder is usually associated with states of endothelial damage such as complications of pregnancy, sepsis, disseminated intravascular coagulation or haemolytic-uraemic syndrome [20]. Unilateral cortical necrosis has been previously described in renal transplantation [21], and in the contra-lateral kidney of kidneys with ureteral obstruction or malignancy [22], contrary to our patient in whom the contra-lateral kidney was intact. In addition to renal infarction, there have been reports of exercise-induced acute renal failure related to severe vasoconstriction and acute tubular necrosis [23]. However, to the best of our knowledge this is the first account of unilateral cortical necrosis following exercise without damage to main renal vessels. The aetiology for unilateral cortical necrosis in this patient is not clear and may include a rare combination of predisposing factors, each of them in itself not sufficient to induce this extremely rare pathologic process. Physical exercise has been demonstrated to be associated with renal vasoconstriction, reduction in renal cortical flow and increase in renal cortical resistance, probably due to immediate activation of α-adrenergic receptors by the renal nerves and subsequently by humural mediators [24], that theoretically might be augmented in the presence of oestrogen in a female patient [25]. Exercise-induced thrombocytosis and later reactive thrombocytosis may be associated potentially with release of serotonin which might have contributed to renal vasoconstriction [26]. However, there is still no clue to a main predisposing factor which might have induced unilateral endothelial damage and cortical necrosis. As pain was described to be extremely acute at the onset, renal artery torsion due to position changes could be a remote hypothetical possibility. In conclusion, we have presented here a unique case of unilateral renal cortical necrosis following the act of skipping with a rope.

References

1. Alamir A, Middendorf DF, Baker P, Nahman NS, Fontain AB and Hebert LA. Renal artery dissection causing renal infarction in otherwise healthy men. *Am J Kidney Dis* 1997; 30: 851–855
2. Momtegomery JH, Moinuddin M, Buchianan JS *et al*. Renal infarction after aerobics. *Clin Nucl Med* 1984; 9: 664
3. Matas AJ *et al*. Polar infarct of a kidney transplant simulating appendicitis. *Am J Surg* 1976; 131: 383–385
4. Frassinelli P, Paquale MD, Reckard C, Goodreau J, Sherween G. Bilateral renal artery thrombosis secondary to blunt trauma: case report and review of the literature. *J Trauma* 1997; 42: 330–333
5. Morrisey EC, McDonald BR, Rabetoy GM. Resolution of proteinuria secondary to bilateral renal vein thrombosis after treatment with systemic thrombolytic therapy. *Am J Kidney Dis* 1997; 29: 615–619
6. Keen ME. Renal vein thrombosis and massive medullary necrosis in a renal allograft. *Arch Pathol Lab Med* 1984; 108: 610–611
7. Lessman R *et al*. Renal artery embolism: Clinical features and Long term follow up of 17 cases. *Ann Intern Med* 1978; 89: 477
8. West MS, Stevens RB, Metrakos P, Foshager MC, Jessurun J, Sutherland FE, Gruessner RW. Renal pedicle torsion after simultaneous kidney-pancreas transplantation. *J Am Coll Surg* 1980; 187: 80–87
9. Abbit PL, Chevalier RL, Rodgers BM, Howard SS. Acute torsion of a renal transplant: cause of organ loss. *Pediatr Nephrol* 1990; 4: 174–175
10. Marvin RG, Halff GA, Elishiabi I. Renal allograft torsion associated with prune belly syndrome. *Pediatr Nephrol* 1995; 9: 81–82
11. Ino T, Shimazaki S, Kaneko K, Yamaguchi H, Kaneko K. Multiple spasms of renal arteries following percutaneous transluminal renal angioplasty in children. *Pediatr Nephrol* 1994; 8: 129–130
12. Hann L, Ptisfer RC. Renal subcapsular rim sign: new etiologies. *AJR Am J Roengenol* 1982; 138: 51–54
13. Georgen TG, Lindstorm RR, Tan H *et al*. CT appearance of acute cortical necrosis. *AJR Am J Roentgenol* 1981; 137: 176–177
14. Papo J, Aviram A, Peer G *et al*. Acute renal cortical necrosis as revealed by computerized tomography. *Isr J Med Sci* 1985; 21: 862–863
15. Jordan J, Low R, Brooke Jeffrey C. CT findings in acute renal cortical necrosis. *J Comput Assis Tom* 1990; 14: 155–156
16. Sallomi DF, Yaqoob M, White E *et al*. Case report: The diagnostic value of contrast-enhanced computer tomography in acute bilateral renal cortical necrosis. *Clin Radiol* 1995; 50: 126–127
17. Sefczek RJ, Beckman I, Lupetin AR, Dash N. Sonography of acute cortical necrosis. *AJR Am J Roentgenol* 1983; 142: 553–554
18. Kim HJ, Cho OK. CT Scan as an important tool in the initial phase of diffuse bilateral renal cortical necrosis. *Clin Nephrol* 1996; 45: 125–130
19. Badiola-Varela CM. Acute renal cortical necrosis: contrast-enhanced CT and pathologic correlation. *Urol Radiol* 1992; 14: 159–160
20. Chugh KS, Jha V, Sakhuja V, Joshi K. Acute renal cortical necrosis—a study of 113 patients. *Ren Fail* 1994; 16: 37–47
21. Blumhardt R, Growcock G, Lasher JC. Cortical necrosis in a renal transplant. *AJR Am J Roentgenol* 1982; 141: 95–96

22. Blute ML, Tempelton AC. Unilateral renal cortical necrosis. *Br J Urol* 1985; 57: 243–244
23. Izumi M, Yokoyama K, Yamauchi A, Horio M, Imai E. A young man with acute renal failure and severe loin pain. *Nephron* 1997; 76: 215–217
24. Mueller PJ, O'Hagan KP, Skoggy KA, Buckwalter JB, Clifford PS. Renal hemodynamic responses to dynamic exercise in rabbits. *J Appl Physiol* 1998; 85: 1605–1614
25. Ferrer M, Osol G. Estrogen replacement modulates resistance of artery smooth muscle and endothelial alpha2-adrenoreceptor reactivity. *Endothelium* 1998; 6: 133–141
26. Blackshear JL, Orlandi C, Hollenberg NK. Constrictive effect of serotonin on visible renal arteries; a pharmacoangiographic study in anesthetized dogs. *J Cardiovasc Pharmacol* 1991; 17: 68–73

Received for publication: 16.8.99
Accepted in revised form: 17.11.99