LETTER / Genito-urinary imaging

Unilateral renal cortical necrosis: Report of a case

R. Quin a, S. Moliere a, E. Rust b, M. Ohana a, C. Roy a,∗

a Radiology B Department, Strasbourg University Hospitals, Nouvel Hôpital Civil, 1, place de l’Hôpital, BP 426, 67091 Strasbourg, France
b Department of Biophysics and Nuclear Medicine, Strasbourg University Hospitals, hôpital de Hautpierre, 1, avenue Molière, 67098 Strasbourg cedex, France

KEYWORDS
Cortical necrosis; Renal insufficiency; Computed tomography

Bilateral renal cortical necrosis is a rare but classic cause of acute renal failure occurring following a state of shock. It results in major irreversible renal impairment. A unilateral form is unusual, so that a particular mechanism is suspected that allows renal function to be preserved. The aim of this original observation is to discuss this pathogenic hypothesis and present a recently observed case.

Observation

A 38-year-old woman with no medical history was admitted to the emergency department with left renal colic which had developed over the previous 72 hours. She had a marked inflammatory reaction.

A CT scan without contrast injection performed 48 hours previously had shown a 4 mm calculus wedged in the right ureteral meatus responsible for right ureteral hydronephrosis. Twenty-four hours after admission, following insertion of a JJ stent, septic shock occurred requiring immediate intensive care, with the appearance of disseminated intravascular coagulation (DIC).

On a CT scan performed on the same day, first without, then with injection of contrast agent (creatinine clearance 55 ml/min), there was no enhancement, in the late venous phase, of the left internal cortex, pathognomonic of necrosis (Figs. 1 and 2). In the delayed phase, there was no opacification of the left excretory system. The right system was normal, with the right JJ stent correctly positioned (Fig. 3).

Two months later, DMSA (99mTc dimercaptosuccinate) renal scintigraphy showed an under-functioning left kidney, providing 16% of total renal function.

∗ Corresponding author.
E-mail address: catherine.roy@chru-strasbourg.fr (C. Roy).

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http://dx.doi.org/10.1016/j.diii.2013.01.026
Three months later, renal function was still moderately impaired. Renal MRI was performed (Figs. 4 and 5) which showed partial recovery of the damaged kidney.

Discussion

Acute bilateral cortical necrosis occurs in certain contexts of multiple organ failure. It complicates DIC in pathological pregnancies with or without sepsis, in isolated septic shock or intoxication. Cortical necrosis in renal grafts has also been described.

The necrosis is ischaemic, secondary to vascular spasm and/or diffuse thrombotic microangiopathy lesions promoted by DIC. It causes destruction of the renal cortex, without affecting the medulla, the juxta-medullary cortex and a subcapsular cortical border. In sepsis, endotoxin-mediated endothelial lesions are added to this.

It has a poor prognosis with irreversible renal sequelae requiring permanent extra-renal dialysis.

Nowadays, imaging provides an early diagnosis and renal biopsy is no longer essential. While an ultrasound appearance with a hyperechoic cortex highlighted by normal echogenicity of the subcapsular (Fig. 1) and juxta-medullary region suggests the condition, a CT examination is pathognomonic [1] when the cortex is hypodense after injection of a contrast agent. This is justified, despite the renal impairment, given the seriousness of the picture and the fact that extra-renal dialysis will have to be continually performed.

The unilateral form is unusual. To our knowledge, there are seven, very old, isolated cases reported in the literature [2–8]. In addition to shock, they mention particular circumstances: six had contralateral hydronephrosis [2–7] and one occurred after intense physical effort [8]. Our case is the...
Unilateral hydronephrosis: associated of renal shock, 

There are several factors that may explain the protective role of hydronephrosis:

- a mechanical factor: the high pressure exerted on the parenchyma reduces the number of microthrombi in the glomerular arterioles. It also induces changes in the glomerular haemodynamics by opening anastomoses from the capsular arteries [9];
- biochemical factors: endotoxins are involved in cortical necrosis due to a lytic effect on the vascular endothelium [10] and in the event of hydrenephrosis, their effect on the glomeruli is reduced. The presence of hydrenephrosis also causes protective agents such as insulin-like growth factor and prostaglandin E2 to be produced [3]. These agents are thought to neutralise the vasoconstriction of the afferent arterioles by modulating the action of angiotensin II, providing protection against renal ischaemia.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References