Case Reports

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ACUTE SEGMENTAL RENAL INFARCTION DUE TO FACTOR V LEIDEN

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Summary.- *OBJECTIVE: Renal infarction is rare and has variable clinical presentations causing diagnostic difficulties. Although most renal infarctions are caused by an obvious thromboembolic factor some are only explained by hereditary or acquired thrombophilia. The authors present a case of segmental renal infarction associated with factor V Leiden.*

METHODS/ RESULTS: A 48-year-old man presented with right flank pain that was unresponsive to analgesia for renal colic. CT scan was performed revealing a partial renal infarction. The etiologic study was only positive to factor V Leiden. In spite of the diagnosis and treatment it resulted in atrophy of the affected renal area.

CONCLUSIONS: Renal infarction can be a presentation of thrombophilia that should be searched in the absence of an obvious embolic factor. Renal CT scan is the best way to a rapid diagnosis and treatment.

Keywords: *Renal infarction. Segmental renal infarction. Prothrombotic disorders. Factor V Leiden.*

Resumen.- *OBJETIVO: El infarto renal es raro y la presentación clínica inconstante causa dificultades diagnósticas. Aunque la mayoría de los infartos renales son causados por factores tromboembólicos obvios, algunos sólo se explican por una trombofilia hereditaria o adquirida. Los autores presentan un caso de infarto renal segmentario asociado al factor V Leiden.*

MÉTODO/RESULTADOS: Hombre de 48 anos presentó con síntomas sugestivos de cólico nefrítico pero la resistencia a analgesia resultó en la realización de un TAC renal que reveló un infarte segmentario del riñón derecho. Lo estudio fue solamente positivo para a presencia de lo Factor V de Leiden. A pesar del diagnóstico y tratamiento el retraso resultó en atrofia del área renal afectada.

CONCLUSIONES: El infarto renal puede ser la presentación de una trombofilia y ésta debería ser pesquisada en la ausencia de factor embolico evidente. La utilización del TAC renal facilita lo diagnóstico y pronto tratamiento.

Palabras clave: *Infarto renal. Infarto renal segmentario. Trombofilia. Factor V Leiden.*

INTRODUCTION

Acute loin pain frequently caused by renal stone diseases can rarely be the presentation of renal vascular occlusions. Renal infarct remains a late and underdiagnosed entity in spite of the knowledge of the clinical picture and of the predisposing factors.

We report a segmental renal infarct caused by inherited prothrombotic disorder.

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CASE REPORT

A 48-year male patient presented to ED with severe acute right loin pain, nausea and vomiting. In spite of unremarkable past medical history even for stone disease a prompt clinical diagnosis of renal colic was undertaken and he started intravenous analgesia.

For persisting and aggravating pain further work-up was entertained. Blood analyses revealed left shift leucocitosis, elevation of alanine and aspartate aminotransferases (ALT and AST) and of the serum lactic desydrogenase (LDH - 3xN) and normal renal function. Facing a normal ultrasound we performed a contrast enhanced CT scan that revealed absence of perfusion territory of the posterior segmentary right renal artery (Figure 1).

He began systemic hypo coagulation with intravenous heparin and maintained warfarin for six months. After a transient hyperthermia he became totally asymptomatic on the third day. Analytical changes resolved progressively.

A thorough search for a cause of the vascular occlusion excluded all embolic risk factors compelling a screen for a thrombophylia (Table I). This was positive for a resistance to activated C protein, due to heterozygocy to factor V of Leiden.

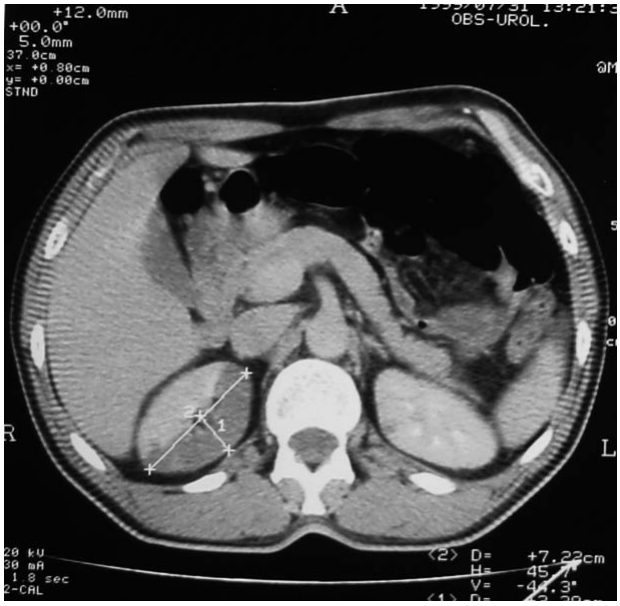

FIGURE 1. TAC renal

The patient remains asymptomatic and on ticlopidin hydrochloride. Control CT scan reveal atrophy of the posterior aspect of the right kidney.

DISCUSSION

The renal infarct is a rare event accounting for 0,004 to 0,007% of all admissions to the emergency department (1). The increasing awareness of the clinical presentation and predisposing factors and mainly by improvements in renal imaging is becoming more efficiently diagnosed. But still remains a missed diagnosis in over half of cases at initial ED visit that leads often to a extended diagnostic delay (2 to 336h) (1,2).

The intense sharp lumbar aggravating pain unresponsive to therapy is the most frequent symptom (61%), accompanied of nauseas and vomiting (3). But it can be totally asymptomatic or with atypically located pain to the superior quadrants of the abdomen, back, or thorax, thus causing diagnostic difficulties with colecistitis, pancreatitis or acute myocardial infarct (4).

The physical exam reveals a distressed patient with pain to the percussion of the flank. Analytically they present leucocytosis, elevation of the sedimentation rate and of the enzymatic values of AST, ALT, and LDH. LDH is the most sensitive marker (1,2,4). The triad of pain, elevated LDH and proteinuria and/or hematuria is present in over 80% of cases (1,5).

Although renal ultrasound can detect changes in the renal tissue perfusion with use of Doppler, the contrasted enhanced renal CT scan is the most reliable and easily obtained method of diagnosis in ED (4,5).

The heart diseases accounts for more than 90% of the cases of renal embolism. Other rarer causes include the displasia of the renal artery, aneurysms, connective tissue diseases, vasculitis, cholesterol embolism and trauma (4,5). The hipercoagulability states, typically associated with vein thrombosis are becoming more accounted by arterial vascular events (1,7,8).

The factor V of Leiden is the most common inherited thrombophilia in Caucasians (5% of population). It is characterized by a genomic substitution that maintains unaffected clothing activity but it becomes resistant to the inactivation by the activated C protein, thus favouring thrombosis.

Therapeutic guidelines have not yet been established but the aim of treatment is to preserve (re-establish renal perfusion) renal function and the treatment of the underlying cause. The therapeutic options include conservative measures with systemic anticoagulation, early use of (90

TABLE I. HEREDITARY AND ACQUIRED THROMBOPHYLIA.

Hereditary thrombophylia	**Acquired thrombophylia**
Protein C deficiency	Dislipidemia
Protein S deficiency	Antiphospholipid antibody sindrome
Antothrombin III deficiency	Sifilis
Factor V Leiden (activated protein C resistance)	Vasculitis (LES, poliartritis nodosa)
Hyperhomocysteinemia	Malignancy
Dysfibrinogenemia	Multiple mieloma
Plasminogen activator inhibitor deficiency	Lupus anticoagulant Anticardiolipin antibody
	Nephrotic syndrome

to 180 min from onset of symptoms) fibrinolitic agents (intra-arterial or in situ associated with an angioplasty), and surgical embolectomy (1,2,4,5).

The emergence of trombolytic therapy (streptokinase, urokinase or tissue plasminogen activator factor) (5) associated to intra-arterial use the balloon angioplasty, makes possible a renal salvation in the order of 70-86% with low morbidity.

As renal viability depends on the precocity of diagnosis, the collateral renal circulation and the efficacy of treatment, a high clinical suspicion is necessary for prompt diagnosis mainly in the absence of underlying heart disease. Segmental renal infarction can be the presentation of a trombofilia and should de addressed in the absence of evident embolic factor. Contrasted renal CT scan provides a fast diagnosis and enables the precocious use of the thrombolytic treatment associated with anticoagulation.

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