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Unveiling Renal Tuberculosis: Common Indicators in the Mastic Kidney - A Case Report

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ABSTRACT

Tuberculosis remains a significant public health concern according to the World Health Organization (WHO). The challenges associated with delayed diagnosis and the severity of lesions caused by Mycobacterium tuberculosis (BK), even post-anti-bacillary treatment, underscore the gravity of the disease, often necessitating nephrectomies.

The clinical presentation can be deceptive, emphasizing the importance of early diagnosis preceding recurrent urological symptoms. In our case study, the diagnosis was prompted by the emergence of the "mastic kidney," signifying an advanced stage of the pathology. Following anti-bacillary treatment, ablative surgical interventions are employed to address potentially serious consequences.

Keywords: Dengue; Serotype; Co-infection; Pathogenesis; Mosquito-borne; NS3; Therapeutic.

INTRODUCTION

Tuberculosis remains a pressing global public health concern, with nearly 10 million new cases reported

annually worldwide, prompting sustained attention from the World Health Organization (WHO) [1]. The intersection with the human immunodeficiency virus (HIV) pandemic and the emergence of multidrug-resistant Mycobacterium tuberculosis strains further complicates the landscape [2]. Urogenital tuberculosis (UGT) holds the fifth position among various localizations in Morocco, following pulmonary tuberculosis, lymph nodes, osteoarticular, and digestive manifestations [3]. UGT constitutes 15 to 30% of all extrapulmonary tuberculosis cases globally [3].

Late diagnosis often poses a challenge, and the severity of Mycobacterium tuberculosis (BK) lesions, persisting even after anti-bacillary treatment, underscores the gravity of the disease, frequently leading to nephrectomies and renal insufficiency [4,5]. Diagnostic imaging plays a pivotal role in identifying urogenital tuberculosis, with intravenous urography and computed tomography serving as fundamental diagnostic tools. Tuberculosis typically involves parenchymal excavation and stenosis of the excretory tract [4]. This report details the case of a 45-year-old patient presenting with bilateral low back pain, revealing characteristic renal putty lesions highly suggestive of urogenital tuberculosis through urinary tract imaging.

CASE REPORT

Mrs. LA, a 46-year-old individual with a history of pulmonary tuberculosis treated a decade ago, presented with bilateral low back pain, accompanied by a decline in general health, anorexia, and a notable weight loss of 10 kg over four months. No hematuria or calculi emission was reported. Clinical examination revealed an apyretic patient with no lumbar tenderness but slight sensitivity in the lumbar fossa. Laboratory results indicated renal dysfunction with a creatinine level of 45 mg/l and a clearance of 13.55 ml/min/1.72 m^2 according to MDRD. Elevated inflammatory markers, including SV, CRP, and leukocytosis (15,000), were observed. Initial radiological assessment via an AUSP revealed a coralliform calculus projecting onto the left renal region, indicative of a complex lithiasic obstruction (Figure 1). A subsequent uro CT scan unveiled extensive parenchymal calcifications in a non-functional kidney, characteristic of an advanced stage of renal tuberculosis, commonly known as a "mastic kidney" (Figure 2). The left kidney showed dilation and multiple staged ureteral stenoses.

A three-day BK urine test confirmed genitourinary tuberculosis. The patient underwent urgent drainage of the right kidney using a double JJ probe, resulting in a slight improvement in renal function (creatinine reduced to 30 mg/l). A cystoscopy with biopsy revealed no anomalies. Therapeutically, the patient received a nine-month anti-bacillary treatment comprising rifampicin, isoniazid, and pyrazinamide. Post-treatment creatinine levels remained stable at 30 mg/l, and radiological follow-up indicated a non-functional putty kidney. Subsequently, a nephrectomy was performed due to the irreparable kidney damage. Macroscopic examination revealed pyelonephritic destruction and histological analysis confirmed a mononuclear inflammatory infiltrate, lymphoplasmacytic filtrate, histiocytes, and granulomatous inflammatory reactions with characteristic giant cells typical of urogenital tuberculosis (Figure 3).

Postoperative monitoring revealed a stable renal function with creatinine levels maintained at 30 mg/L, and satisfactory morphological evaluations were observed.

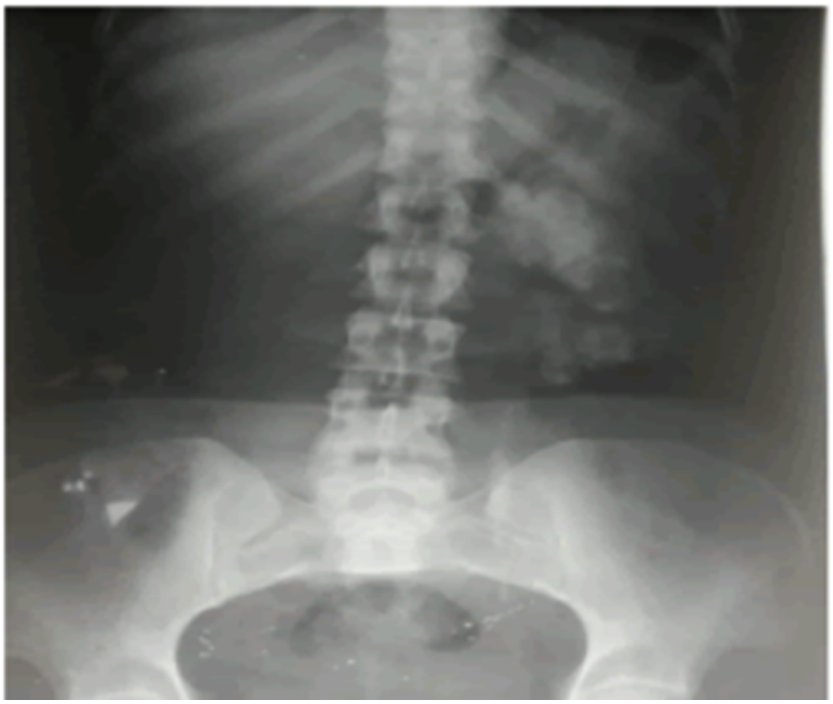

Figure 1: The image of the AUSP shows an opacity of calcium density in the left renal region.

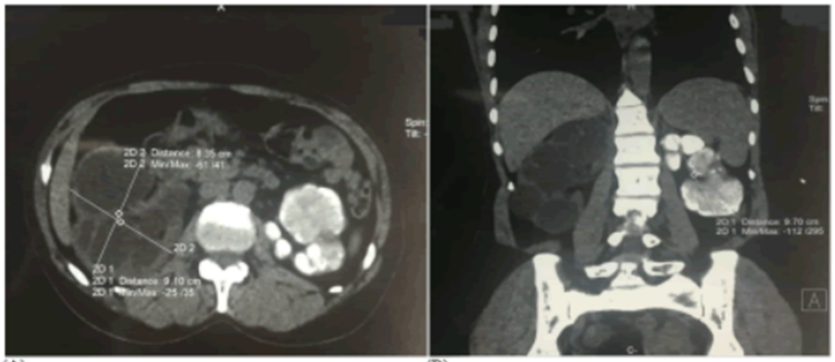

Figure 2: Figure 2: (a) Abdominal CT in axial section: several lobar parenchymal calcifications of all the calyces of the left kidney "matic kidney", (b) Abdominal CT in coronal reconstruction: left kidney completely calcified putty".

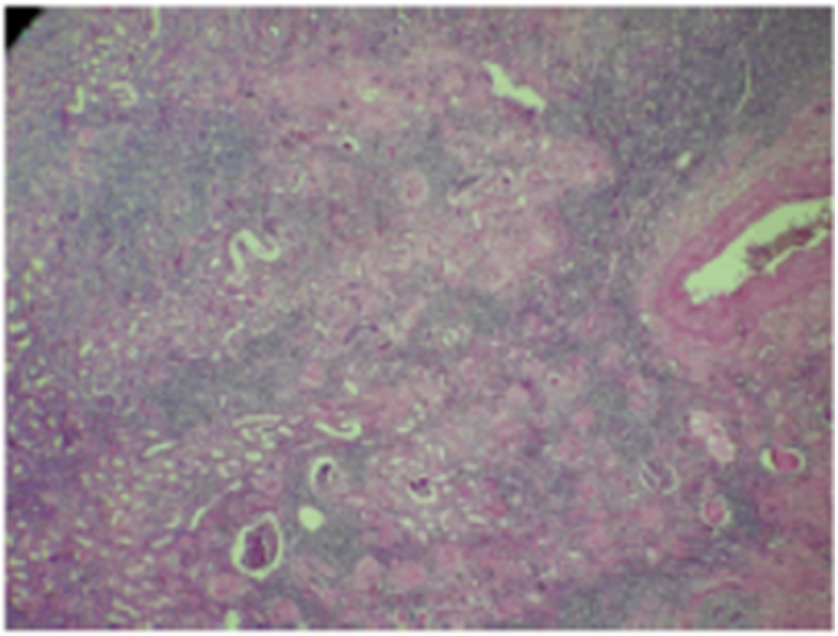

Figure 3: Mononuclear inflammatory infiltrate of the renal parenchyma (HGx100).

DISCUSSION

The epidemiology of renal tuberculosis exhibits significant variability in frequency and geographic distribution. Its exact prevalence is challenging to ascertain due to screening method dependencies. Renal (genitourinary) involvement constitutes approximately 30% of extrapulmonary tuberculosis cases [6]. Pulmonary localization prevails as the most common, followed by lymph nodes, with renal involvement following closely. Tuberculosis affecting the kidneys typically occurs bilaterally through the hematogenous route, resulting in granuloma formation. While these lesions usually heal without causing kidney disease, they can become caseous, leading to tubular lumen rupture years after primary pulmonary infection. In most cases, renal function remains unaffected, except for rare instances where postrenal acute renal failure (ARF) manifests due to granulomatous ureteric stenosis [7].

Hematogenous kidney damage produces granulomas in the glomeruli, evident as white nodules, particularly in the cortex, measuring 1 to 3 mm in diameter. Histologically, these are epithelioid granulomas, often containing giant Langhans-type cells, with or without caseous necrosis [8]. Radiologically, renal calcifications, a classic manifestation of tuberculosis, are observed in 24 to 44% of cases [5,9]. Extensive parenchymal calcifications resulting in a mastic kidney appearance are pathognomonic of a terminal stage of urogenital tuberculosis, best studied through computed tomography.

Pathological lesions of renal tuberculosis can be suggestive but non-specific, varying based on lesion location, spread route, germ virulence, and immune defenses. Initial lesions resemble nonspecific chronic interstitial nephritis. In the state phase, hematogenous renal involvement is characterized by white nodules, cortical in nature, and corresponding to epithelioid and intercellular granulomas. These granulomas can lead to the destruction of renal parenchyma with areas of scar fibrosis. Cortical granulomas may rupture into the tubular and/or pyelic lumen, resulting in tuberculous pyelonephritis. Tuberculosis-related kidney damage can take various forms, such as cavitation or pronephroses, referred

to as the "mastic kidney," often associated with calcifications.

Therapeutically, the recommended approach involves Quadri therapy for anti-tuberculosis treatment, followed by therapy for a total duration of six months. The management of a caseous lesion in a partially functional kidney remains a subject of debate. However, it is advisable to address ureteral obstruction through interventions like a JJ catheter or subcutaneous nephrostomy if the kidney maintains functionality with adequate cortical thickness and creatinine clearance above 15 ml/min. In the presented case, emergency drainage was performed on the right kidney using a double J probe, followed by a left nephrectomy due to persisting radiological calcifications and the nonfunctional nature of the kidney, posing a source of infection or potential secondary arterial hypertension.

CONCLUSION

The urinary and genital forms of tuberculosis share an insidious and destructive progression, often resulting in irreversible sequelae, with impaired renal function being a prominent concern. Late diagnosis, owing to nonspecific clinical signs, underscores the importance of considering tuberculosis in the face of atypical and recurrent symptoms. Treatment encompasses medical and surgical interventions for sequelae, aligning with the regimen used for pulmonary tuberculosis. However, renal insufficiency correction might be incomplete, leading to potential severe sequelae, including the need for dialysis.

CONCLUSION

The authors declare no conflicts of interest concerning the publication of this article.

REFERENCES

1. World Health Organization. Global tuberculosis report 2015. Geneva: WHO 2015.
2. Aït Belghiti F, Che D. L’épidémiologie de la tuberculose en France en 2013. Bull Epidemiol Hebdo 2015;(9-10):175-271.
3. Chaulet P, Khaled NA, Amrane R. Problèmes posés par la tuberculose dans le monde en 1982. Rev Fr Mal Respir 1983;11(2):79-110.
4. Di Stefano-Louineau D, Faure F, Moulin G, Chagnaud C, Kasbarian M, Helenon O. Tuberculose rénale et urétérale. EMC Radiodiagn Urol Gynecol 1994:34-230.
5. Gibson MS, Puckett ML, Shelly ME. Renal tuberculosis. Radiographics 2004;24:251-256.
6. Eastwood M, Kennedy DH. Extrapulmonary tuberculosis. In : The biology of Mycobacteria, vol III, edited by Ratledge C., Stanford JL, Grange JM. New-York : Academic Press 1989;245-284.
7. Conte G, Iavarone M, Santorelli V, De Nicola L. Acute renal failure of unknown origin: Don't forget renal tuberculosis. Nephrol Dial Transplant 1997;12:1260-1261.
8. Mallinson WJW, Fuller RW, Levinson DA. Diffuse interstitial renal tuberculosis- an unusual cause of renal failure. Q J Med 1981;50:137-148.
9. Watfa J, Michel F. Uro-genital tuberculosis. Prog Urol 2005;15:602-603.