Chronic Pyelonephritis Presenting as Multiple Tumor-Like Renal Lesions

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Abstract

We encountered a 49-year-old Japanese man in whom tumor-like renal lesions developed as a result of chronic Staphylococcus aureus pyelonephritis. The patient complained of general fatigue, weight loss, and anorexia for 6 months. Contrast-enhanced computed tomography (CT) of the abdomen revealed multiple low-density lesions in both kidneys and paraaortic lymphadenopathy. A strong uptake of Ga⁶⁷ citrate in the lesions and elevation of serum soluble interleukin-2 receptor and thymidine kinase activity were strongly suggestive of primary renal lymphoma; however, histologic examination of renal biopsy specimens revealed severe tubulointerstitial change, consistent with chronic pyelonephritis. Following systemic antibiotic treatment, multiple tumor-like lesions regressed 4 months later. This case suggested that chronic pyelonephritis could present as bilateral renal tumors.

Key words: chronic pyelonephritis, lymphoma, tubulointerstitial nephritis, soluble interleukin-2 receptor

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Introduction

Imaging techniques such as ultrasound sonography, computed tomography (CT) and magnetic resonance imaging (MRI) have been dramatically improving the accuracy of diagnosis for tumor-like lesions (1). However, the differential diagnosis of these lesions in the kidney is sometimes difficult even when such tools are employed. The diagnosis is usually made presumptively based on clinical symptoms, laboratory findings, and results of diagnostic imaging. Histologic examination is also required for definitive diagnosis in some cases.

Although multiple tumor-like renal lesions are not frequently seen, identification of such lesions raises the suspicion of malignant lymphoma. Tumor-like lesions in the kidney are also sometimes observed in xanthogranulomatous pyelonephritis. Although primary renal lymphoma (PRL) is extremely rare, accounting for only 3% of all primary renal tumors (2), the most common pattern of involvement in this disease is multiple bilateral masses (observed in about 60% of cases) (3). Diagnosis of PRL on percutaneous renal biopsy has also been reported (4). In contrast, in xanthogranulomatous pyelonephritis, involvement is generally unilateral.

We report herein a case presenting with bilateral tumor-like renal lesions. Although PRL was initially suspected, chronic pyelonephritis was clinically and histologically proven as the etiologic process.

Case Report

A 49-year-old Japanese man was admitted to Fujinomiya General Hospital on June 12, 2001. The patient had been well until six months previously, when he had developed fatigue, weight loss, and anorexia. Physical examination on admission revealed the following: blood pressure, 126/90 mmHg; pulse, 73 beats/min, regular; and temperature, 36.8
Contrast-enhanced CT of the abdomen shows low-intensity lesions (arrows) in both kidneys, and enlargement of paraaortic lymph nodes. MRI disclosed low-intensity on T1-weighted images (T1WI) and low to high-intensity on T2-weighted images (T2WI). Gadolinium-enhanced MRI showed heterogeneous and intensive enhancement of these lesions, while Ga67 citrate scintigraphy demonstrated a strong accumulation in the left renal lesion.

As PRL was strongly suspected, open biopsy of the tumor-like lesion in the left kidney was performed. However, lymphomatous changes were not observed in the biopsy specimen. Instead, severe tubulointerstitial changes consisting of tubular atrophy, numerous tubular casts, and extensive interstitial fibrosis with focal cell infiltrations were noted. A focal interstitial infiltrate of lymphocytes was observed, but lymphomatous infiltration was absent. Immunostaining using polyclonal antibodies showed that infiltrating cells expressed CD20/L26 and CD45RO/UCHL-1 as the markers of polyclonal B cells and T cells, respectively. Glomeruli appeared normal on light microscopy. These histopathological findings are consistent with chronic tubulointerstitial nephritis (Fig. 3). Intravenous urography revealed cortical scarring with calyceal dilatation and deformity in the left kidney, with no evidence of vesicoureteric reflex. On the basis of these findings, we diagnosed chronic pyelonephritis as the cause of the multiple tumor-like renal lesions.

The patient was treated with meropenem trihydrate (1 g/day, intravenously), resulting in resolution of pyuria and improvement in the patient’s general condition after one week. The antibiotic agent was subsequently changed to levofloxacin (300 mg/day). After 4 months, CT revealed significant regression of the bilateral renal tumor-like lesions and paraaortic lymphadenopathy with normal kidney function.

Discussion

In general, PRL is not undisputed as a clinical entity because the kidneys do not contain lymphatic tissue and the mechanism by which this disorder develops is unclear (5). Some authors speculate that renal lymphoma originates from the renal capsule, and infiltration to the renal parenchyma...
occurs subsequently (6, 7). A characteristic finding of renal lymphoma on CT is a homogenous solid tumor which may disrupt the renal contour and extend into the perirenal tissue planes (8). On MRI, PRL appears as lesions that are hypo-intense compared with the renal cortex on T1WI and as hypo- to iso-intensity lesions on T2WI (9). Gallium also accumulates intensively in the tumor, which often confirms the correct diagnosis, thus obviating the need for unnecessary laparotomy and/or nephrectomy (10).

In the present case, on the base of radiological findings and elevated blood biological markers, we first suspected PRL as a cause of multiple tumors in the kidney. However, renal histological examination disclosed chronic interstitial nephritis without any lymphoma cell infiltration. Ga$^{67}$ citrate scintigraphy revealed a strong accumulation in the left kidney. Antibiotic treatment successfully dissolved clinical symptoms and improved tumor-like lesions and paraaortic lymph node swelling. In addition, MRI showed low-intensity on T1WI but low to high-intensity on T2WI, a similar finding with bacterial nephritis (11). These observations convincingly suggested a close relationship between the tumor-like lesions and chronic renal infection in this case.

The underlying disease that developed to bilateral chronic pyelonephritis remains unclear in this diabetic patient. At admission, we did not detect the presence of occult infections such as sinusitis and periodontitis known to spread easily to the blood-stream. Intravenous urography also did not disclose any evidence of vesicoureteric reflex. Malacoplakia, meaning soft plaque, is a rare chronic inflammatory disease which sometimes involves bilateral renal parenchyma, and can present as a renal mass (12). Malacoplakia seems to be related to a defect in the intracellular killing of coliforms, especially *Escherichia coli*. It is characterized by large polygonal-shaped macrophages with foamy eosinophilic cytoplasm (von Hansemann cells) and strongly PAS-positive granules called Michaelis-Gutman bodies (13). Xanthogranulomatous pyelonephritis, a chronic infection of unknown cause, usually involves diffusely in one kidney, but bilateral focal solid mass formation has been reported in a patient with xanthogranulomatous pyelonephritis (14). However, in the renal tissue, we did not detect any Michaelis-Gutman body or giant cell.

In the present case, although tubular atrophy and interstitial fibrosis remained, tumor regression occurred as a result of a reduction in the number of infiltrating lymphocytes in the renal interstitium due to antibiotic administration. Some authors also have reported that tumor-like lesions of acute focal bacterial nephritis disappear within several months on administration of appropriate medical therapy (15, 16). Although the precise mechanism of lesion regression in response to antibiotics is unclear, it seems likely that remodeling of the renal vasculature might be promoted by the resolution of tissue inflammation.

In this case, the right kidney did not become positive for Ga$^{67}$ scintigraphy despite the presence of mass lesions on radiological examinations. The reasons for this discrepancy remain unclear, but since the mass lesions were smaller than in the left kidney, the severity of inflammation may be weak in the right kidney.

In summary, we present a case of chronic pyelonephritis that mimics malignant lymphoma in both kidneys. This rare case suggests that chronic pyelonephritis should be considered as one of the differential diagnoses in a patient who presents tumor-like multiple lesions in the kidney.

**References**

