Mucosal or submucosal ureteral wall thickening at CT? the clue of the diagnosis.

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Learning objectives

- to illustrate the differences in appearances of ureteral thickening: mucosal or submucosal thickening
- to discuss the different etiologies of ureter thickening correlated with clinical evolution, analytical results and pathology

Background

The clinical presentation of the ureter thickening is highly variable. It may be asymptomatic; alternatively it may cause colicky pain or haematuria or it may present clinical and analytical signs of sepsis.

Treatment options vary depending on the aetiology which may be A) inflammatory (pyelitis or pyeloureteritis related or not to kidney stones), B) catheter-related or idiopathic; C) infectious (related to tuberculosis or bacterial infection); or D) neoplastic (related to urothelial carcinoma or lymphoma).

Knowledge of the different radiological manifestations of the etiologies of thickening of the ureter is the key to correct diagnosis and to the selection of optimal treatment.

Findings and procedure details

The radiological appearances of mucosal o submucosal ureter wall thickening are different, correlated by pathology.

The ureteral wall is thin and is composed by mucosa, lamina propria, muscle. (Fig. 1 on page 5)

Usually, mucosal thickening is caused by cell proliferation at the mucosal and lamina propria (Fig. 2 on page 5).

Submucosal thickening is caused by cell proliferation at the lamina propria (Fig. 3 on page 6).
We studied different patients with radiological alterations of the ureter wall on CT and correlated them with the clinical evolution and/or pathology. There are some diseases that causes usually mucosal thickening like urothelial neoplasm or pyeloureteritis and other that causes usually submucosal thickening like chronic inflammatory catheter-related or lymphoma (Table 1 on page 7).

**INFLAMMATORY AETIOLOGY**

- Chronic inflammatory catheter-related

Some patients with chronic double J catheter in the urinary system, develops ureteral thickness because of chronic inflammatory (Fig. 3 on page 6).

**INFECTIOUS AETIOLOGY**

- Pyeloureteritis

Alone pyeloureteritis is a rare entity that can simulates an urothelial neoplasm. The clinical symptoms and laboratory items as the clinical evolution can help to make the correct diagnosis. After medical treatment of antibiotics, the symptoms disappeared. (Fig. 4 on page 8 and Fig. 5 on page 9).

**NEOPLASM AETIOLOGY**

- urothelial neoplasms

Transitional cell carcinoma (TCC) is commonly encountered in the urinary bladder and is usually diagnosed at cystoscopy. Five percent of urothelial tumors arise from the ureter or the renal pelvis or calices, accounting for approximately 10% of upper tract neoplasms. Patients with TCC typically present with hematuria, which may be frank or microscopic. Up to one-third of patients present with flank pain or acute renal colic, symptoms more typically associated with calculi. Occasionally, tumors may manifest with distant metastases or be discovered incidentally at radiologic examination.

Renal TCC most frequently arises in the extrarenal part of the pelvis, followed by the infundibulocaliceal region. The distribution is equal between the left and right kidneys, with 2%-4% of cases occurring bilaterally. Twenty-five percent of upper tract tumors occur
in the ureter (Fig. 2 on page 5), where 60%-75% of cases are found in the lower third, with no side predominance. Tumor spread occurs by mucosal extension or local, hematogenous, or lymphatic invasion. The most common sites for metastases are the liver, bone, and lungs. The tumor stage at diagnosis influences the development of local recurrence and metastases and hence overall survival. Multicentric TCC is common and is associated with poor survival. Synchronous or metachronous tumor of the ipsilateral or contralateral collecting system is also common, necessitating vigilant urologic and radiologic follow-up.

Upper tract TCC typically occurs in the 6th and 7th decades of life, with males affected three times more often than females. Besides increasing age and male gender, the most important risk factor is smoking, with smokers being two to three times more likely to develop TCC than nonsmokers. Chemical carcinogens (aniline, benzidine, aromatic amine, azo dyes), cyclo-phosphamide therapy, and heavy caffeine consumption are also associated with TCC, and all predispose to synchronous and metachronous tumor development. These substances are metabolized and excreted in the urine as carcinogenic substances that act locally on the urothelium. Stasis of urine and structural abnormalities such as horseshoe kidney are also associated with increased prevalence.

These tumors are usually small at diagnosis, grow slowly, and follow a relatively benign course. Pedunculated or diffusely infiltrating tumor is less common, accounting for approximately 15% of upper tract TCCs, but tends to behave more aggressively and be more advanced at diagnosis. Infiltrating tumors are characterized by thickening and induration of the ureteric or renal pelvic wall. If the renal pelvis is involved, there is often invasion into the renal parenchyma. However, this infiltrative growth pattern preserves renal contour and differs from renal cell carcinoma, which is typically expansile.

- Lymphoma

Renal lymphoma has a wide variety of CT appearances. In many cases, diagnosis is not difficult because patients present with a known lymphoma at the time of imaging. Lymphoma typically involves the kidney in one of several recognizable patterns. These patterns include multiple renal masses, solitary masses, renal invasion from contiguous retroperitoneal disease, perirenal disease, renal sinus involvement and diffuse renal infiltration. No specific correlation has been found between the exact type of lymphomatous involvement and the pattern or prevalence of renal involvement. Direct renal invasion from contiguous retroperitoneal disease is another common pattern of involvement in renal lymphoma and is seen in approximately 25%-30% of patients with documented disease. These patients typically present with a large, bulky retroperitoneal mass that envelopes the renal vasculature and invades the renal hilum. Lymphoma can preferentially affect the renal sinus, although this is an uncommon occurrence. In most patients, the renal arteries and veins remain patent despite tumor encasement, a finding
that is characteristic for lymphoma. However, contiguous extension of retroperitoneal involvement in the renal collecting system can often cause obstruction, and affected patients will commonly present with hydronephrosis (Fig. 6 on page 10). Transitional cell carcinoma is usually associated with a greater degree of obstruction of the collecting system (Fig. 7 on page 11). Displacement of the kidney can also be seen. Following treatment of larger masses, residual fibrosis is often seen and can be mistaken for recurrent or residual disease.

Images for this section:

Fig. 1
**Fig. 2:** Ureteral wall thickening because of neoplastic cells proliferation in mucosa and lamina propria. A. pathology specimen (orange line) correlated by coronal nephrographic phase CT B (white arrow).
Fig. 3: Ureteral wall thickening because of inflammatory cells and vessels in lamina propria. A. Pathology specimen (red line) correlated by coronal nephrographic phase CT B (white arrow).
<table>
<thead>
<tr>
<th>Condition</th>
<th>Mucosal Thickening</th>
<th>Submucosal Thickening</th>
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</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Yes</td>
<td>Sometimes-usually</td>
</tr>
<tr>
<td>Inflammatory-idiopathic</td>
<td>Sometimes-usually</td>
<td>Yes</td>
</tr>
<tr>
<td>Stone</td>
<td>Sometimes</td>
<td>Never</td>
</tr>
<tr>
<td>Urothelial neoplasm</td>
<td>Yes</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>Never</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Table 1
**Fig. 4:** Male of 57 years old with left colic pain, fever and leukocytosis. A Axial unenhanced CT, B Axial nephrographic phase CT and C Axial pyelographic phase CT, demonstrates wall pelvis renal thickness (white arrow). One month after medical treatment, renal pelvis wall was normally (black arrow), D Axial nephrographic phase and E Axial pyelographic phase.
Fig. 5: Urethral thickness (white arrow) also was present in the same case as figure 5. A Coronal unenhanced CT, B Coronal pyelographic phase CT, demonstrates urethral wall thickness with periureteral fat stranding. C Coronal pyelographic CT shows resolution after 1 month of medical treatment.
Fig. 6: 80-year-old woman with right colic pain. A axial unenhanced CT, B axial nephographic CT and C axial pyelographic CT demonstrates marked thickness of the renal pelvis wall, associated with retroperitoneal bulky mass; right renal function is preserved, with correct captation and elimination of the endogenous contrast agent. The diagnosis was made by percutaneous core needle biopsy guided for CT and the diagnosis was lymphoma non-Hodgking type B.
Fig. 7: The same patient as figure 15. A axial pyelographic CT and B coronal nephrographic CT demonstrates involved ureter (white arrow) by a bulky retroperitoneal mass, without collapsed ureter lumen and preserved elimination of endovenous contrast agent.
Conclusion

The CT appearances of the ureteral thickening helps to reach the correct diagnosis, helped by clinical and analytical findings.

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References


