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INTERESTING UROGRAPHIC CHANGE OF THE URETER IN UPPER URINARY TRACT TUBERCULOSIS

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Urinary tuberculosis is one of the diseases continuously decreasing in number with the progress of chemotherapy and improvement in the living environment. In recent years the proportion of patients with this disease has been less than 0.5 percent of the out-patients.

We encountered ureteral tuberculosis showing a pattern of diverticulum-like cystic extension arising from tuberculous ulcer, which is very interesting from a roentgenologic viewpoint.

In this paper we studied on specially the mode of development of ureteral tuberculosis and its characteristic roentgenograms.

INTRODUCTION

Urinary tuberculosis is a urinary infection with mycobacterium tuberculosis.

It includes few cases of primary infection, being of the hematogenous infection mainly to the kidney and prostate gland from other lesions, predominately pulmonary tuberculosis. Ureteral tuberculosis is mainly of the descending infection from renal tuberculosis but also includes cases of ascending infection from prostatic or vesical tuberculosis. It could also be caused by lymphogenous infections from the ureter or tissues around the ureter on rare occasions.

Generally, roentgenologic changes of urinary tuberculosis are primarily accounted for by the patterns of renal tuberculosis, and roentgenologic changes of ureteral tuberculosis are secondary in many cases, consisting mainly of stricture, dilatation, shortening, straightening, irregularity and vesico-ureteral-reflux.

We have recently encountered a case of tuberculosis of the upper urinary tract showing ureteral patterns which are of great interest from a roentgenologic viewpoint.

In this paper we report the case with some discussion in reference to literature.

CASE REPORT

On November 17, 1978, a 43-year-old man hospitalized for tuberculous subcutaneous abscess in the left precordial region at the Department of Surgery was transferred to the Department of Urology with abnormality in left kidney revealed by excretory urography and urinary turbidity as the chief complaint.

The past history and family history were not remarkable.

Laboratory examinations revealed WBC 7300, erythrocyte sedimentation rate 18mm at 1 hour and 34mm at 2 hours. Urinalysis showed RBC 0-2/hpf, WBC>100/hpf, albumin +, acid-fast bacillus negative on smear, but positive on culture. Endoscopy revealed vesical mucosa and both ureteral orifices to be normal. Indigo-carmine test was normal on the right but no excretion of dye was observed even at 10 minutes on the left. KUB did not show any abnormal findings such as calcification.

Drip infusion pyelograms were normal on the right side, but dilated or rat-bitten patterns of calyces, cavitation and irregularities were observed in the left kidney. The middle calyx and ureter were not visualized (Fig. 1). Retrograde pyelography confirmed lesions in the left kidney and showed patterns of cystic extension suggestive of the diverticulum at three places in the left ureter (Fig. 2).
Fig. 1. Excretory urogram. The right kidney and ureter are normal. The left kidney shows hydrouphrotic, atypical irregularity, cavitation, poor visualization of contrast medium of the upper and lower calyces. The middle calyx and left ureter are not visible.

Fig. 2. Left retrograde pyelogram shows, in addition to characteristic involvement of left kidney, three diverticulum-like-cavity of left ureter due to tuberculous ulcerations.

Fig. 3. Cut surface of removed ureter shows cavitation due to tuberculous ureteritis.

Fig. 4. The ureteral epithelium has disappeared. In the mucosa, there are several follicules with a Langhan’s giant cells, epitheloid cells, lymphocytes (H.E. stain ×40).
On the basis of the results above, the patient was diagnosed as tuberculosis of the kidney and ureter on the left, and underwent nephroureterectomy, removal of abscess in the right precordial region and partial resection of the 5th rib on the right on December 11, 1978.

The removed kidney showed pyonephrosis containing plenty of caseous substances.

The ureter had a prominently hypertrophied wall and showed a marked defect corresponding to the region showing roentgenographic changes (Fig. 3).

Histologic findings: The kidneys showed a characteristic tuberculous lesion. The same was true of the ureter, there being tuberculous nodulations with epitheloid cells, Langhan’s giant cells and lymphocytes in the ureteral mucosa.

Partially, marked ulcerous lesions such as defects of the mucous epithelium and tunica muscularis were also observed (Fig. 4).

Judging from the above, it appears that the contrast medium was retained in the defective areas of the mucous membrane and tunica muscularis arising from tuberculous ulcer resulting in an interesting finding, a pattern of cystic extension suggestive of the diverticulum on the retrograde pyelographic examination.

**DISCUSSION**

In Japan the case of tuberculosis has markedly decreased in number with the progress of chemotherapy and improvement in the living standard.

In 1952 the number of cases of pulmonary tuberculosis decreased to half and subsequently the case of genitourinary tuberculosis also decreased in number.

With 1950~1960 as the peak, the case of urinary tuberculosis decreased and at present accounts for less than 0.5% of the out-patients1-5.

As for the route of infection, cases of primary urinary tuberculosis are rare and urinary tuberculosis is mostly of the secondary infection from pulmonary tuberculosis, showing the hematogenous spread mostly to the kidney. However, a direct hematogenous spread to the prostate gland and epididymis is also noted in some cases.

**Urinary tuberculosis is mainly of the descending infection from the kidney; from the kidney to the ureter, bladder and prostate gland; from the prostate gland to the spermatic cord and epididymis. The ascending infection, from the prostate gland to the bladder and ureter, is also observed on rare occasions.**

While ureteral tuberculosis is mainly of the descending infection from the kidney, there develops ureteral tuberculosis lymphogenously on rare occasions.

As early symptoms of urinary tuberculosis, bladder-irritating symptoms is often observed and these symptoms often lead to detection of urinary tuberculosis5).

Renal tuberculosis shows a variety of x-ray patterns according to the degree of advance of lesions6-12).

These patterns are classified into moth-eaten, ragged, rat-bitten calyces, stenosis of the calyceal neck and dilation of calyces, calyceal obstruction, small or large cavitation, hydronephrotic changes, irregular shadows, no visualization of kidney and calcification (chalk or mortar kidney).

These changes are brought about by a mixture of various lesions coupled with destruction of the kidneys, cicatrical stenosis and ureteral changes, being findings characteristic of tuberculosis.

Roentgenograms of ureteral tuberculosis also have distinctive features, being often taken as secondary changes of renal tuberculosis10-13,16,17).

Ureteral tuberculosis begins with inflammation and edema of the mucous membrane and subsequently there develops submucosal infiltration.

Tuberculous follicles are produced and tuberculous nodules are formed on the surface. With the advance of the disease, these follicles get united to cause caseation and eventually form a typical tuberculous ulcer. When the inflammation advances more deeply, there develop nodulation and ulceration, and inflammation spreads to the adventitial sheath of the ureter and tissues around the ureter.

Protracted tuberculosis shows chronic findings with granulation tissue in the submucous layer. Fibrosis arises from this
granulation tissue. As these changes occur frequently, the ureteral wall becomes hypertrophied, loses normal elasticity and preristalsis, develops fibrosis, cicatrization, stenosis and causes shortening and rigidity of the ureter, resulting in dilatation of the upper urinary tract \(^{10,12-17}\).

Such changes, single or multiple, short or long, can be seen anywhere in the ureter but occur mainly at the end of the ureter.

This is related to the fact that there are generally more cases of vesical tuberculosis than those of ureteral tuberculosis in the descending infections from renal tuberculosis, meaning that inflammation spreads from the urinary bladder to intramural ureter and the terminal portion of the ureter in many cases.

This chronic inflammation leads to dysfunction of the ureterovesical valve. Further, cicatricial contracture gives rise to vesico-ureteral reflux \(^{10,15}\).

Besides, there also are fibrosis and contracture as the healing process of the lesion, which cause ureteral stenosis and shortening of the ureter.

The ureter shows characteristic findings roentgenographically such as stricture, rigidity, shortening, straightening, defect, reflux and kink depending on the process of inflammation and is described as beaded ureter, corkscrew ureter and pipestem ureter according to the case \(^{10,12,17}\). However, calcification is rare (Fig. 5).

Our case was very unique in roentgenograms revealing hypertrophied ureteral wall resulting from chronic inflammation, narrowing of the ureteral lumen, and cystic extension suggestive of the diverticulum at three places due to ulcer.

In other regions of the ureter, fibrosis was conspicuous and peristalsis was absent.

REFERENCES


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