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Radiologic signs of genitourinary tuberculosis: An aid for earlier diagnosis

Dr Sanjay Prakash Dhangar, Dr Ibrahim H Kothawala, Dr Sachin Patil, Dr Abhay Kumar, Dr Abhijit Whatkar

Department of urology, Bharti Hospital and Research Center, Pune-Satara Road, Dhankawadi, Pune-411043, Maharashtra, India

Abstract

Objective: We herein describe the various modes of radiological signs of genitourinary tuberculosis (GUTB) and a simple diagnostic approach to it.

Materials and Methods: We made a literature search through Medline database and various other peer-reviewed online journals to study the various modes of presentation in GUTB. We reviewed over 100 articles published online, which were tracked through the key words like GUTB and extrapulmonary tuberculosis.

Results: GUTB has varied presentation and the most common way of presentation is in the form of irritative voiding symptoms, which are found in more than 50% of the patients. The usual frequency of organ involvement is: kidney, bladder, fallopian tube, and scrotum. The usual tests used to diagnose GUTB are the demonstration of mycobacterium in urine or body fluid and radiographic examination. Intravenous urography (IVU) has been considered to be one of the most useful tests for the anatomical as well as the functional details of kidneys and ureters. In cases of renal failure, MRI can be used. Newer examinations such as radiometric liquid culture systems and polymerase chain reaction (PCR) give rapid results and are highly sensitive in the identification of mycobacterium.

Conclusion: GUTB can involve any part of the genitourinary system and presentation may vary from vague urinary symptoms to chronic kidney disease. Plain X-ray and IVU provide us with important diagnostic and functional clues before and after diagnosis of GUTB.

Keywords: Diagnosis of genitourinary tuberculosis, extrapulmonary tuberculosis, GUTB, IVU, radiological signs of GUTB

1. Introduction

Tuberculosis (TB) continues to be an important public health problem in our country. The World Health Organization (WHO) estimates that the largest number of new TB cases in 2005 occurred in the South-East Asia Region [1]. In India, more than 1000 lives are lost every day due to TB despite the availability of modern diagnostic aids and treatment [2]. Genitourinary tuberculosis (GUTB) is the second most common form of extrapulmonary tuberculosis after lymph node involvement [3]. Kidney is usually the primary organ infected in urinary disease, and other parts of the urinary tract become involved by direct extension except epididymis and fallopian tubes [4]. Timely diagnosis and treatment will prevent the late sequelae of this disease, like non-functioning kidney and thimble urinary bladder. Here, we discuss the important and classic radiological signs of genitourinary tuberculosis to diagnose it at the earliest time and to prevent the dreaded complications.

2. Radiological Signs

2.1 Plain Radiograph

Radiographic identification of calcification associated with renal TB is becoming less common. It is noted on conventional radiography in 24-44% [5]. It may be the first sign that TB is present [5]. Fine calcifications that were previously unidentifiable are now much better seen with CT [6]. Although calcification is unusual in the early stages of the disease, nearly every end-stage tuberculous kidney contains calcification. Renal calcification can take a variety of patterns, varying from few minute areas of calcification to a complete cast of the kidney. Initially, the calcifications are faint and punctate, but eventually coalesce [7, 8]. Triangular ring-like calcifications that are characteristic of papillary necrosis may be noted within the collecting system [9]. A lobar pattern of calcification, with calcific rims outlining the periphery of distorted renal lobes, is pathognomonic of TB [10] (Figure 1A, 1B). When calcification extend along the ureter, it is virtually as diagnostic as the lobar pattern of calcification. In fact, the occurrence of any upper ureteral calcification along with any other renal calcification is a good pointer of renal TB.

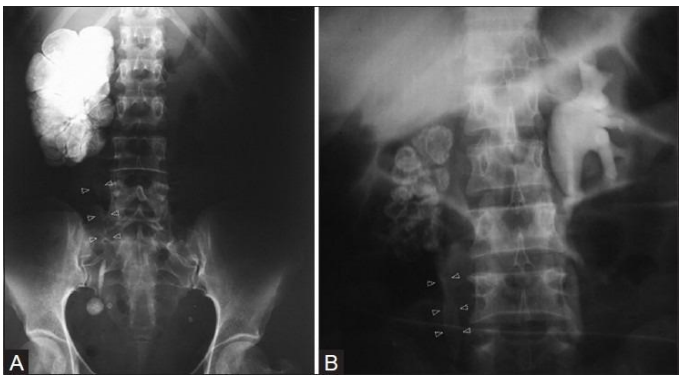

Fig 1A: Plain radiograph revealing classic lobar pattern of calcification, which is pathognomonic of end-stage renal tuberculosis. Ureteral calcification is also noted, which is fainter in upper parts (arrowheads), Fig 1B: intravenous urogram revealing the 'classic' lobar pattern of calcification in a non-functioning right kidney.

Gow believed that calcification in renal TB has an unfavorable prognosis and, if left alone, would result in an increase in the size of the calcification and deterioration of renal function [11]. Renal or ureteric calculi have been noted in up to 19% of cases

[12]. Calcified caseous tissue characteristically appears to be very homogeneous and only moderately dense, looking like ground glass; this is often referred to as 'putty kidney'.(fig 2) Premkumar *et al*. labelled calcification 'putty-like' if any area of faint calcification of uniform density was greater than 1 cm in diameter [13].

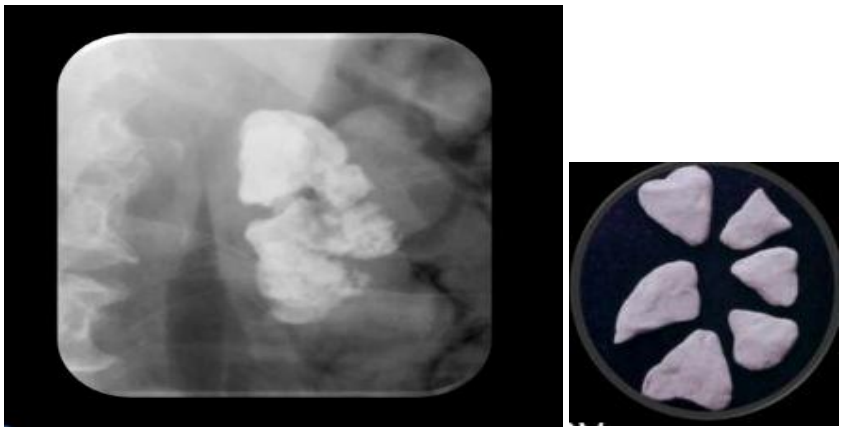

Fig 2: Diffuse uniform extensive parenchymal calcification forming a cast of the kidney with autonephrectomy

2.2 Intravenous Urography

The IVU has been considered as one of the most useful tests for obtaining anatomical and functional details of the kidneys [14]. It can show a broad range of findings, depending on the severity of infection. In a series of 45 patients, the IVU pointed to the diagnosis of urinary TB in 88% [15]. However, approximately 10-15% of patients who present with active renal TB may have normal urographic findings [16]. Isolated parenchymal miliary tubercles usually produce urographic findings only when a calyx is involved [17]. The earliest urographic change occurs in the minor calyces, with subtle initial signs such as minimal calyceal dilatation [5] and mild loss of calyceal sharpness due to mucosal edema [17] (Fig 3A,3B). As the disease progresses, the calyceal outline becomes more irregular, fuzzy, and ragged and, later, feathery and moth-eaten in appearance (Fig 4A, 4B, 4C).

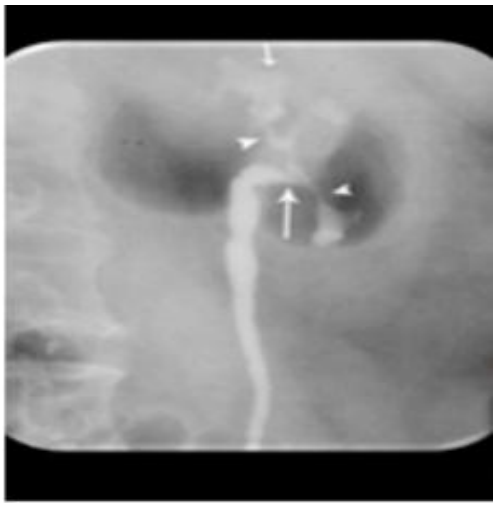

Fig 3A: showing fuzzy irregular and truncated calyces-features suggestive of papillary necrosis (white arrow in ivp film)

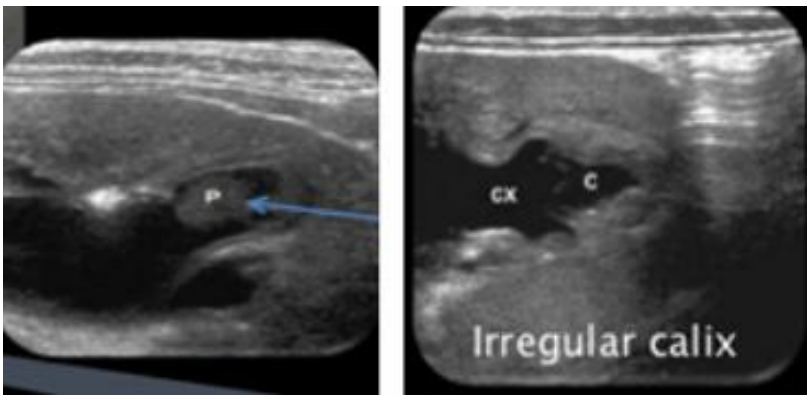

Fig 3B: Usg films showing irregular calyx and papillary necrosis (p=necrosed papilla)

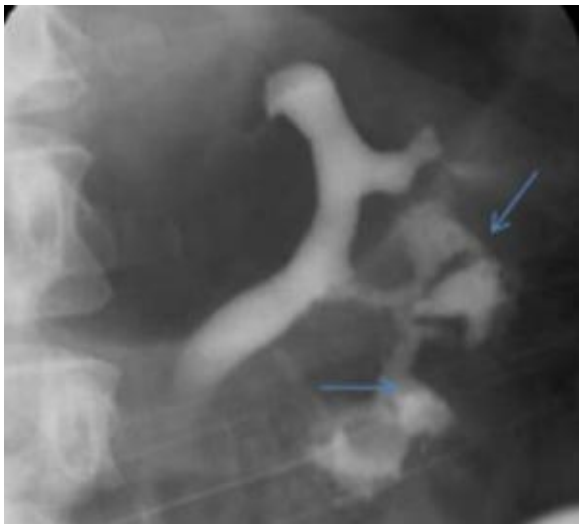

Fig 4A: showing moth eaten appearance in IVP

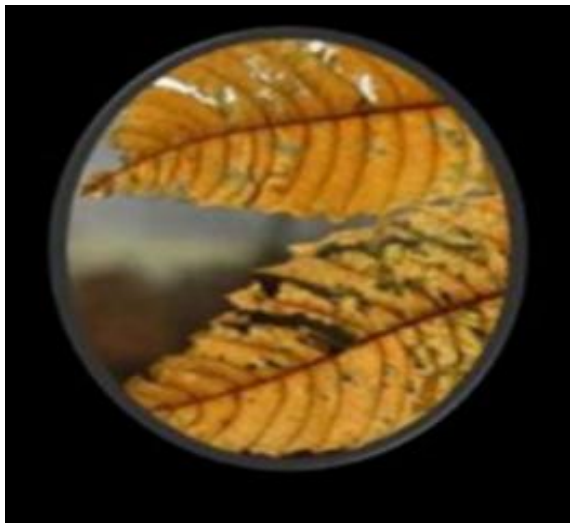

Fig 4B: illustartion of moth eaten appearance

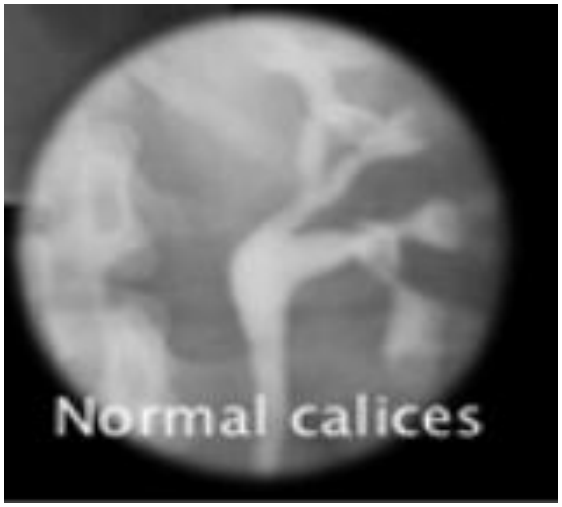

Fig 4C.

Although calyceal erosion has been described as the first IVU sign in renal TB, in practice, early papillary necrosis (Fig 3A, 3B) may be the first detectable sign. Caseating parenchymal tuberculomas may rupture into an adjacent calyx, resulting in an irregular cavity that fills with contrast [17, 5] (Fig 5A, 5B). According to Elkin, this resembles renal papillary necrosis on IVU or retrograde pyelography [17]. TB papillary necrosis results not only from ischemia, which is the basis of change in most renal papillary necrosis, but also as a result of direct tissue destruction. We have seen classic early forniceal and even central papillary necrosis (Fig 3A, 3B) in numerous proven cases of renal TB that cannot be differentiated from papillary necrosis due to other causes. (Fig 6A, 6B)

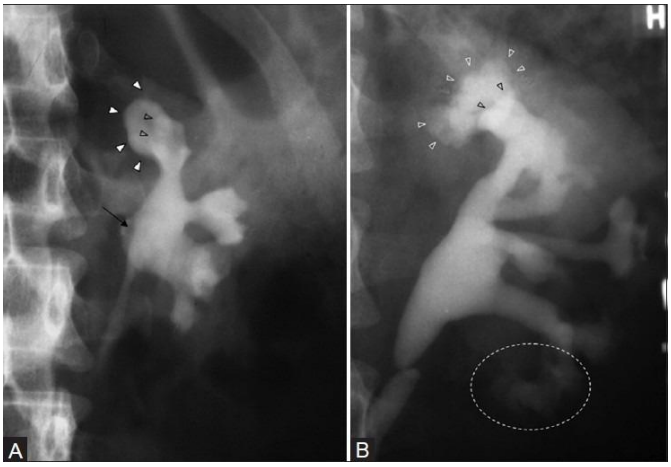

Fig 5: (A) Intravenous urogram revealing a "hiked up" renal pelvis (arrow). Tuberculosis cavity (white arrowheads) communicating with the upper group of calyces. Black arrowheads represent medial border of a compound upper calyx, (B) Intravenous urogram revealing fluffy cavities (white arrowheads) communicating with a compound upper calyx (black arrowheads). Odd shaped pockets of contrast communicating with a lower calyx (and with each other) [circled areas], represent caseated necrotic cavities

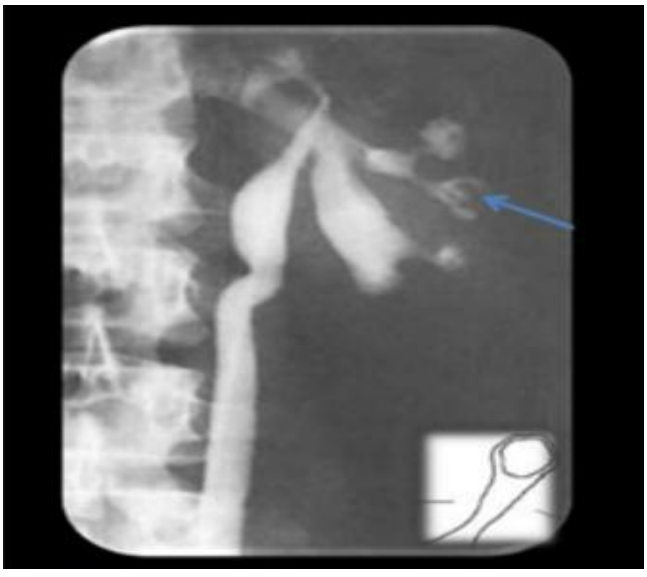

Fig 6A: Showing Forniceal excavation or the lobster claw Sign in which the necrotic papillary tip may remain within The excavated clayx, producing a signet ring when the clayx Is filled with contrast material

Fig 6B: illustrating the lobster's claw

In TB, the central type is probably due to ischemia, and the forniceal, usually due to direct erosion. Ischemic papillary necrosis in renal TB could be caused by a small granuloma eroding or impinging upon adjacent vessels [18], or be the result of TB endarteritis [19]. Medullary cavitation with communication to the collecting system has been described as a frequent finding by Kollins *et al*. [20] (Fig 7A,7B) It may be the sole radiographic abnormality and, at times, may not be differentiable from papillary necrosis due to other causes. It may involve one or more papillae, unilaterally or bilaterally, and can vary in appearance from small and smooth to large and irregular [20]. Irregular pools of contrast material may thus be seen adjacent to dilated calyces [21]. Focal or global compromise of renal function may be noted (Fig 8A, 8B)

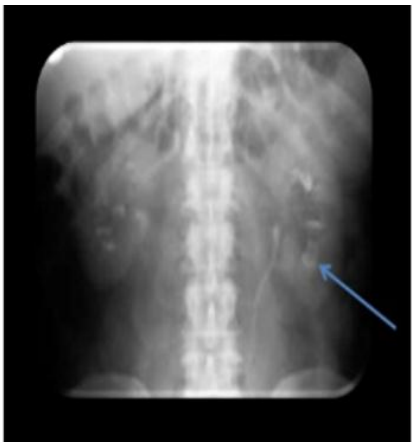

Fig 7A: Showing cavitation: egg in cup appearance

Fig 7B: illustrating egg in cup appearance

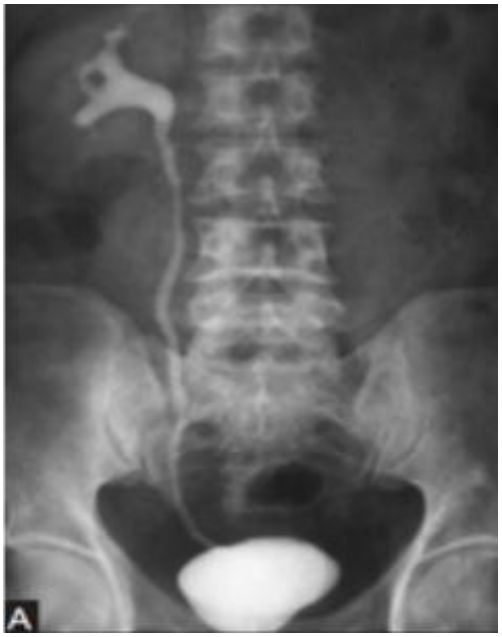

Fig 8A: intravenous urogram revealing a non –functioning left kidney and a small Capacity urinary bladder. The combination is suggestive of a tuberculosis origin for the Non- function,

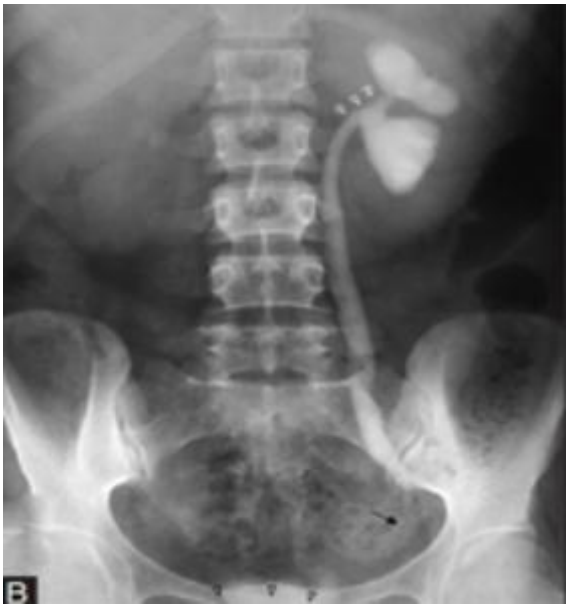

Fig 8B: ntravenous urogram revaling non-functioning right kidney. Left renal pelvic and upper infundibular scarring (white arrowheads), resulting in Uneven caliectasis. a let lower ureteric stricture (arrow) and small capacity bladder(black arrowheads) are also noted

The abnormalities outlined so far, i.e., calyceal dilatation, loss of sharpness, fuzziness, papillary necrosis, and initial cavitation, are the early changes of renal TB. The late or advanced manifestations include extensive cavitation, fibrotic strictures, cortical scars, mass lesions, calcification, autonephrectomy, perinephric abscess, and fistula formation.

Cicatricial deformity of the calyces may lead to pinching of the tips of the minor calyces. Continued destruction may transform the minor calyx into a large pocket of necrotic caseous material, with or without fibrous obstruction of the infundibulum [5]. Cavities may be differentiated as obstructive or non-obstructive [22]. In the former, contrast medium does not enter the cavity on

retrograde pyelography but vague visualization may occur on the IVU (Fig 9).

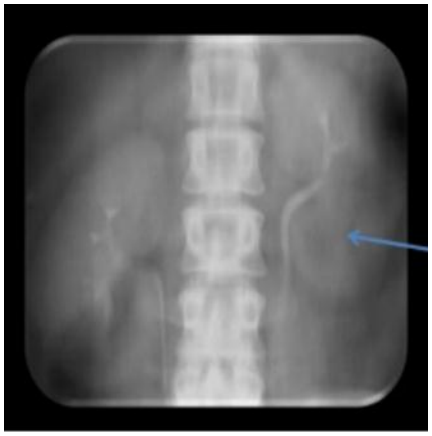

Fig 9: Showin nonfilling of the collecting system elements at the lower pole of the left kindney (arrow)

In the latter, opacification occurs only on the retrograde study (Fig 10 & 11)

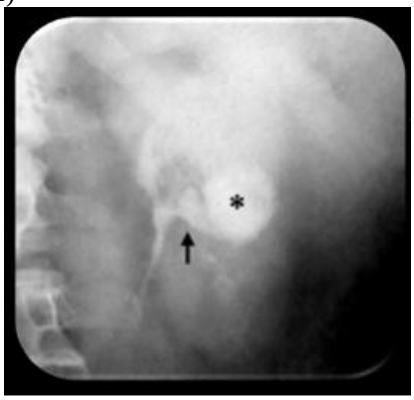

Fig 10: showing the collecting system with contrast material in large papillary cavity star showing the cavity and the arrow showing the blunted calyx

Making it difficult to differentiate a dilated, diseased calyx from a cavity that has ruptured into the calyx. The obstructive type needs close follow-up with US. Gentle balloon dilatation may be attempted if the narrowing gets worrisome. Successful antegrade balloon dilatation of tuberculous strictures of the urinary tract has been performed by Kim *et al* [23, 24]. The lipping type of cavity, projecting medially, is considered diagnostic of TB [25].

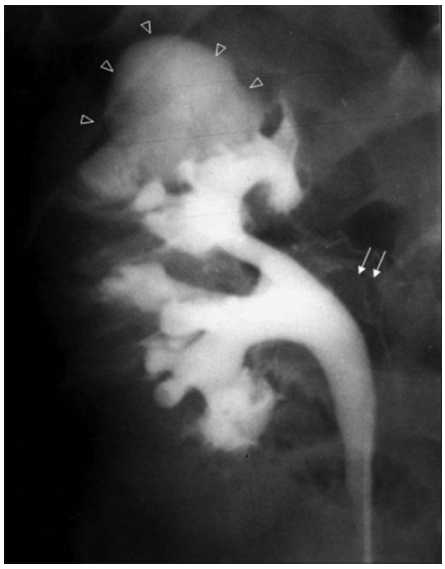

Fig 11: Pyelocavitatory (arrowheads) and pyelolymphatic reflux (arrows) noted on retrograde pyelography

Renal functional damage due to strictures is greater than that from renal parenchymal tuberculomas [17]. The three danger points of fibrosis are: The lower ureter, the pelvi-ureteric junction, and the neck of a calyx [6, 5, 26]. Fibrosis is the result of healing and may thus develop during treatment [17]. Strictures may affect the calyceal neck, infundibulum, or the renal pelvis and result in hydrocalyx, regional hydrocalicosis, or generalized dilatation of the calyces and infundibula, respectively. Commonly, a number of strictures are present, and the renal pelvis is small and contracted. Obstruction of areas not directly affected by tuberculous ulcerations and kinking of the renal pelvis can occur due to traction from a strictured infundibulum and parenchymal fibrosis. These are known as Kerr's kinks[27]. Scarring causes various kinds of calyceal deformities, some of which are probably unique to TB. A stricture of the inferior margin of the renal pelvis and its cephalic retraction, the so-called 'hiked-up pelvis,' may be seen[6] (Fig 12A,12B).

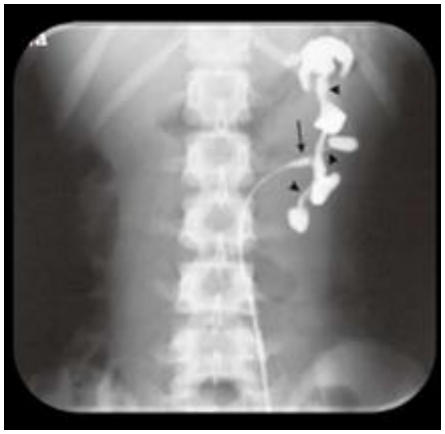

Fig 12A: Showing hiked up pelvis: cephalic retraction of the inferior medial margin of the renal pelvis at the ureteropelvic junction

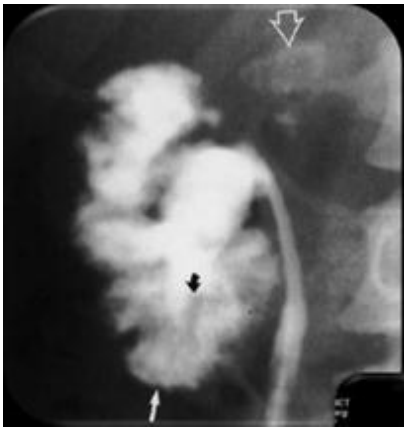

Fig 12B: Showing Kerr's kink: cortical scarring with dilatation and distortion of adjoining calyces coupled with strictures of the pelvicalyceal system

Obstruction from strictures leads to dilatation of the PCS and pressure atrophy of the renal tissue. Such hydronephrosis tends to have irregular margins and reveal filling defects, which are due to caseous debris [28] (Fig 13). TB infection in the dilated calyces results in a closed pyocalycosis and leads to caseation of the surrounding renal tissue. A completely stenosed infundibulum or calyx can cause complete failure of contrast excretion by the involved renal parenchyma ('phantom calyx') [17] (Fig 14). If such an area is small and represents the only abnormal focus within the kidney, the urogram may erroneously be interpreted as normal [29]. A tiny infundibular stump (amputated calyx) or spike may be a good clue in such instances.

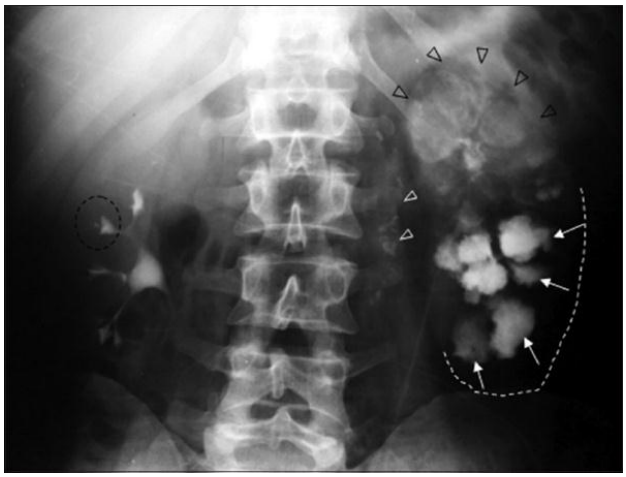

Fig 13: Delayed phase of intravenous urogram with a non-functional left kidney opacified retrogradely: Developing lobar caseation in the upper third of the left kidney (black arrowheads). Note assimilation of the dilated calyces into the renal parenchyma. Ragged hydrocalicosis (indicative of marked urothelial thickening) noted in the lower half of the left kidney (arrows). Parenchymal demarcation is still clear adjacent to the same (dotted line represents the non-visualized left renal outline). Right renal papillary necrosis is also seen (circled area) and so are calcified left paraspinal lymph nodes (white arrowheads)

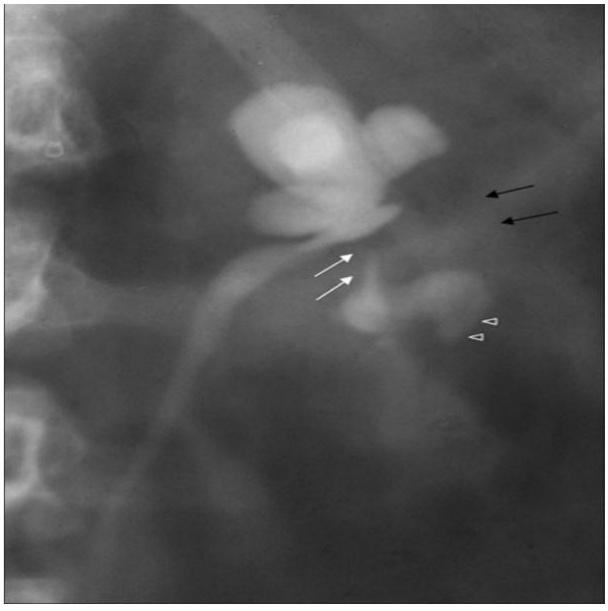

Fig 14: Intravenous urogram revealing cicatrization that has resulted in obliteration of the renal pelvis, multiple infundibular strictures (white arrows) and uneven caliectasis. Note non-visualization of the middle group of calyces–the "phantom calyx" (black arrows) and a cavity communicating with a lower calyx (arrowheads)

Parenchymal scars are common, being seen in over 50% of patients [12]. They can be seen either overlying blunted calyces or in between the calyces.

As the disease progresses it may become difficult to differentiate between hydronephrosis and TB granulomas, as both are masses that do not opacify with contrast at urography/pyelography. US, CT, and MRI are helpful in such situations.

The late phase of progression of granulomatous destruction of the kidney, with subsequent obstructive uropathy, can lead to an autonephrectomy. This is considered typical of end-stage renal TB [6]. There are two types: (1) the caseo-cavernous autonephrectomized kidney, i.e., an enlarged kidney converted into a caseous filled sac, with or without calcification; and (2) the shrunken, fibrotic, and often calcified kidney. In both instances, there is usually obstruction of the ureter at some point, but this is not essential in type (1) [5]. However, both types will be non-functional on the IVU (Fig 15).

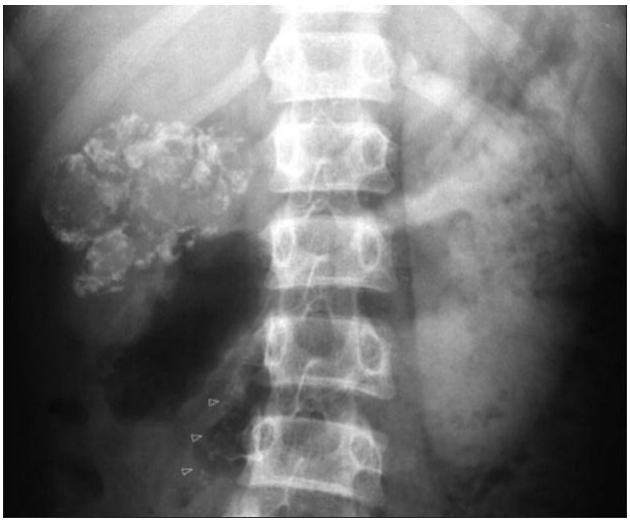

Fig 15: Nephrographic phase of intravenous urogram: Right subtotal autonephrectomy ("lobar calcification") with partial sparing of the lower pole, which revealed functional calyces on later films. Note scattered calcification in the right psoas region (arrowheads)

Non-functioning kidneys in TB can be seen in: (A) autonephrectomy, (B) obstruction (ureteric obstruction, including post-treatment fibrosis), and (C) renovascular hypertension due to renal artery disease in a fibrotic kidney filled with cavities [5]. US or CT plays an important role not only in evaluating non-functioning kidneys but also in patients with complications of renal TB.

The other important complications of renal TB are: (1) perinephritis, (2) perinephric abscess, (3) fistulae, (4) psoas abscesses and, rarely, (5) renal failure.

IVU features of perinephric abscess include restricted renal movement on comparative deep inspiration and expiration films, or on intentional double exposure films. Those of psoas abscess include lateral renal and ureteric displacement. These are better evaluated on USG, CT, and MRI.

The typical ureteric abnormalities include the saw tooth appearance, beaded or corkscrew ureter and the pipe-stem ureter. (Fig 16, 17 & 18).

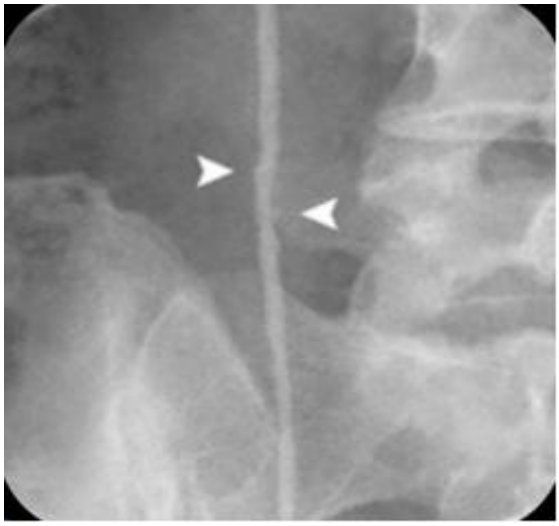

Fig 16: Showing the typical saw tooth appearance of the Ureter: Ulcerations causing mucosal irregularities of the ureter

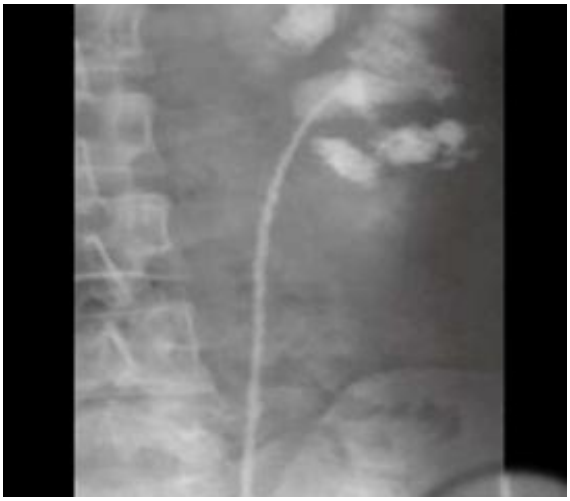

Fig 17: Showing beaded ureter: chronic fibrotic ureteric strictures

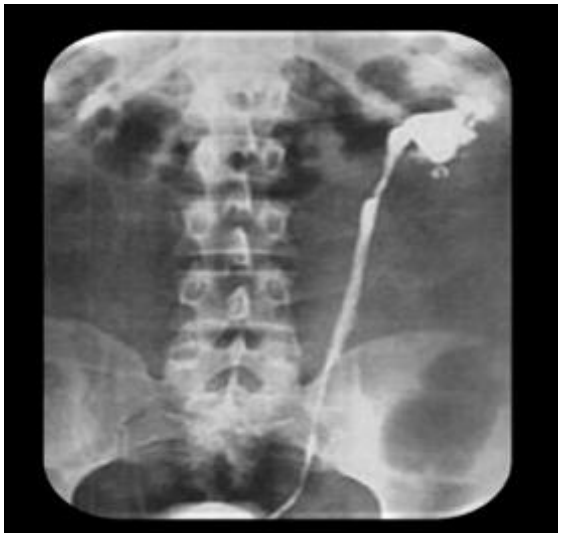

Fig 18: Showing the typical pipe stem ureter: rigid irregular ureter that lacks normal peristaltic movements. Also notable is the distortion amputation and irregularity of the upper pole calyces.

The bladder typically shows the thimble appearance (Fig 19A, 19B)

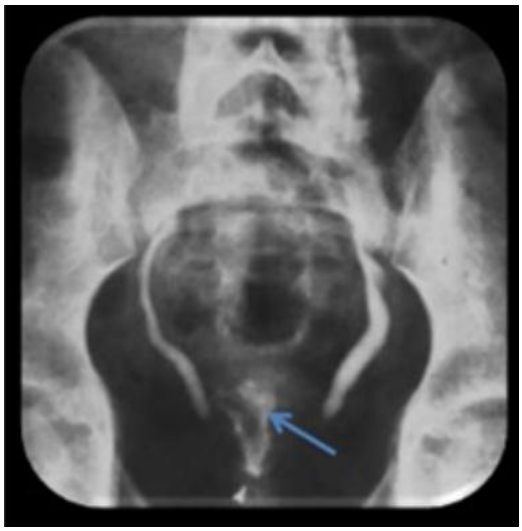

Fig 19A: Showing the typical thimble bladder: diminutive and irregular urinary bladder-simulating a thimble (arrow)

Fig 19B: Showing the thimble

3. Conclusion

Recent revolutionary PCR based technologies such as: a) cartridge based nucleic acid amplification techniques (Gene Xpert) diagnose TB much earlier (including drug sensitivity); within 2 hours, and (b) Line Probe Assay (LPA) systems diagnose TB by 2 days. We have successfully used Gene Xpert test to diagnose extra-pulmonary TB. This has tremendous potential to revolutionize TB, especially MDR TB, management.

Imaging will continue to play a key role, both in the initial diagnosis as well as in the follow-up of patients with GUTB. Diffusion Weighted MRI and other techniques such as MR elastography and USG elastography/tissue strain analytics (including exciting new variants such as acoustic radiation force impulse technology) should prove very useful in the evaluation of the complex changes the kidney undergoes with TB, especially key damaging factors such as fibrosis. These techniques will be useful in the initial diagnosis as well as in the follow-up of these patients, including the assessment of response to treatment and the monitoring of sequelae. Spectral imaging on CT (dual-/tri-/quad-band), when it becomes widely available, should further enhance the diagnostic armamentarium of radiologists investigating this scourge.

Molecular imaging is also expected to contribute significantly, especially as drugs to treat latent/dormant TB are looming on the horizon.

4. Limitations

This is a review article. The availability of necessary journals and online access made the study feasible. The readily availability of CT scan in the present era makes the diagnosis of tubercular changes easy. However, IVU is still done in places where CT scan is not available. A stronger clinical suspiscion is the needed presently.

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