THE LATE COMPLICATIONS OF URINARY TUBERCULOSIS*

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These complications, which are usually found in patients who have already had one kidney removed for tuberculosis, develop as a result of persistent active infection with its attendant pathology, viz. the destructive processes of caseation and ulceration leading to fistula formation, and the attempt at repair by granulation tissue and fibrosis leading to contraction and deformity. These changes usually develop very gradually and take years to establish themselves. Fistula formation is very rare except as a result of operative interference. Fibrotic contraction and deformity are common and occur in every chronic case in due course, although there is evidence that this process is more marked in some patients than in others. Ultimately the disease may become quiescent and appear to have burnt itself out. Unfortunately, by this time the urinary tract has often been converted to a deformed wreck of its former self, with more or less destruction of renal tissue.

Typical examples of these changes are illustrated by cases 1 and 2. In case 1, R.H. (Figs. 1, 2 and 3) the right kidney was removed for tuberculosis in 1945, and 5 years later infection started in the left kidney. He had intermittent specific therapy. At his last check-up a few months ago he was symptomatically free and the urine negative on microscopy. The deformity, which involves the pelvis and calyces, mainly took 8 years to develop.

An abortive attempt may be made by nature to cure the disease by auto-nephrectomy, in which the ureter becomes occluded and the caseous pyonephrosis above is partly calcified. Usually live tubercule bacilli are present and may break into activity at any time, with the development of further complications such as a perinephric abscess. This is illustrated by case 2, J.R. (Fig. 4). At nephrectomy the ureter was found to be converted into a thin fibrous cord without a lumen.

The ureter may be extensively involved in one of the two following ways:

(*a*) As a result of active disease in the ureter itself which produces irregular fibrotic contraction and dilation. The ureter is typically shortened and fixed to surrounding structures, with 'tenting' of the bladder on the same side.

(*b*) As a result of, and secondary to, extensive involvement of the bladder. The bladder undergoes fibrotic contraction and deformity, associated with widely patent and fixed 'golf-hole' orifices, up which reflux takes place. This leads to marked dilation of the upper urinary tract with elongation of the ureter, which becomes a thin-walled tortuous tube with the folds lightly bound together by adhesions. In severe cases the bladder may ultimately become converted into a small, deformed and partitioned, so-called 'thimble' bladder, with the walls largely replaced by fibrous tissue.

The following two cases illustrate these changes, viz. case 3, M.B. (Fig. 5) who had a right nephrectomy for renal tuberculosis 17 years ago, and case 4, K.V. (Figs. 6 and 7), who had a right nephrectomy for renal tuberculosis 8 years before the last X-ray. Fig. 7 is an example of a 'thimble'

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bladder with the tuberculous process probably burnt out. She had a very bad stricture of her urethra. All attempts at instrumentation failed and a filiform made a false passage

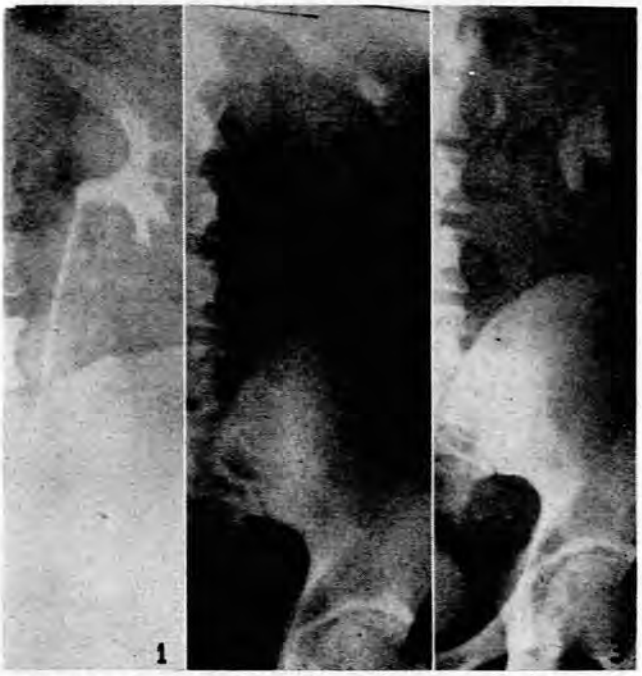
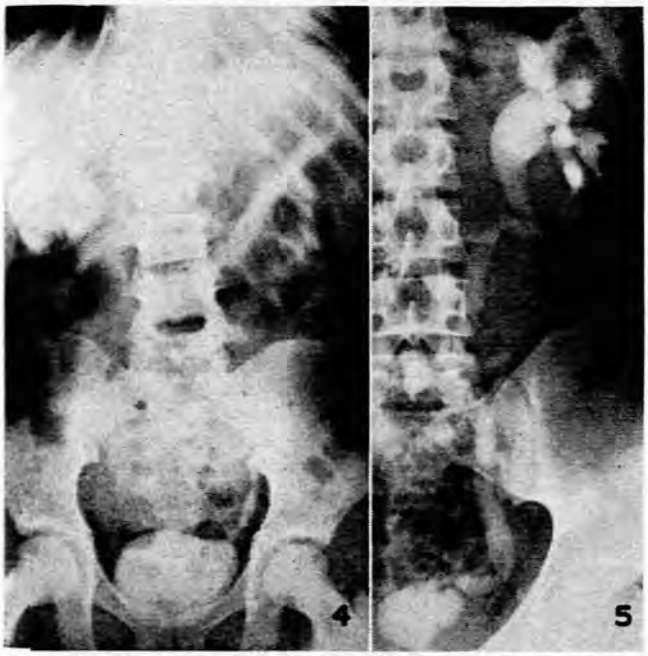


Fig. 1. Case 1, R.H. 18 September 1945. Left retrograde pyelogram, taken at time of right nephrectomy. *Fig.* 2. Case 1, R.H., 10 August 1953. I.V.P., showing involvement of upper calyx. *Fig.* 3. Case 1, R.H., 1 May 1958. I.V.P., showing obliteration of upper calyx or its complete obstruction, and dilation of lower calyces due to stricture at ureteropelvic junction.



Fig, 4. (touched up). Case 2, J.R. Partially calcified right caseous pyonephrosis. *Fig.* 5. Case 3. M.B. I.V.P., showing dilated upper renal tract and small contracted bladder.

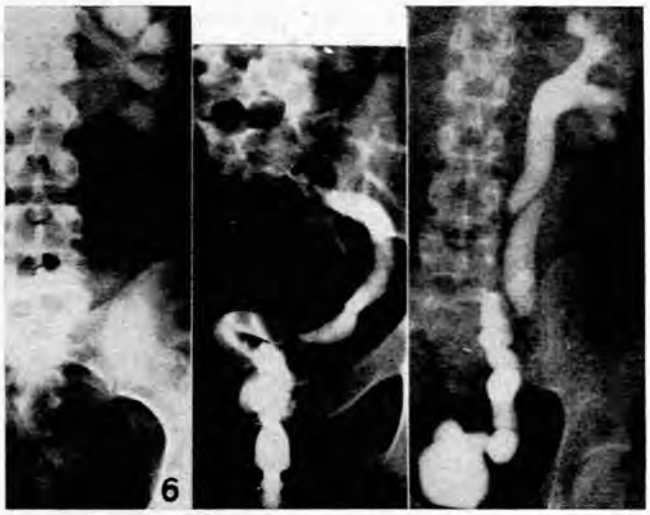


Fig. 6. Case 4, K.V. 7 April 1949. I.V.P. *Fig.* 7. Case 4, K.V. 6 June 1952. Retrograde urethrogram, showing extravasation of dye into vagina, thimble bladder and reflux up ureter. *Fig.* 8. Case 5, F.J.S.K. Cystogram showing reflux up renal tract.

into her vagina. The urethrogram shows the extravasation of dye through the fistula. The dilated tortuous ureters shown in Figs. 5 and 7 may be produced by a stricture at their lower ends but, as Pyrah and Raper[1] state, they are more commonly due to dilation of the lower ureter, with reflux. These authors' explanation of the pathogenesis of this process is that the persistent high intravesical pressure forces open the ureteric orifices and thus leads to reflux.

The question must be asked here why most cases after unilateral nephrectomy for tuberculosis should heal completely while some fail to do so. This failure will manifest itself mainly in the bladder, with gradually increasing contracture leading ultimately to dilation of the remaining renal tract, as illustrated. One problem to decide is in what part of the renal tract this persistent focus resides which prevents complete recovery. The original primary focus of infection in the urinary tract, as we know, is almost invariably in the kidney, very rarely in the bladder, and to my knowledge never in the ureter. It stands to reason, therefore, that the persistent focus which underlies the above changes will usually be in the kidney, occasionally in the bladder, and very rarely in the stump of ureter left after nephrectomy. This, of course, has a bearing on whether the routine operative treatment of renal tuberculosis should be by nephrectomy or nephro-ureterectomy. It is my opinion that after nephrectomy for tuberculosis the stump of the ureter left behind, although extensively involved, will in due course usually heal and be converted into a thin-walled normal-looking ureter, although fibrosed.

It is interesting to compare this late result of tuberculosis of the bladder with that found in the late stages of bilharziasis in Egypt as described by Sayegh and Dimmette.[2] These authors state that the calcification of ova and associated ulceration with fibrosis lead to contraction of the bladder in a short period of time. Associated with this is an enlargement of the trigone with dilation of the posterior urethra and also of the ureters and renal pelves. Here also the rise in intravesical pressure is thought to force open the posterior urethra

and also the ureteric orifices, with resultant dilation of the upper renal tracts. The ureteric orifices themselves may be dilated. This description is in sharp contrast to the usual bilharzial changes seen in the Cape, which lead to narrowing of the ureteric orifices and stricture formation in the lower third of the ureter.

The prostate may be involved by haematogenous or secondary spread from the upper renal tract. In the advanced condition there may be extensive destruction and fibrosis associated with a dilated posterior urethra and prostatic ducts and contraction of the bladder neck. Veenema and Lattimer[3] state that the tuberculous process may be so destructive as to produce a cavity below the trigone, described as a *voorblase*. There is often associated calcification and rarely fistula formation to the rectum or skin. Pyrah and Raper[1] describe marked dilation of the prostatic urethra and ducts secondary to the small contracted bladder and produced by the dilating action. This must be very rare. The involvement of the *anterior urethra* is rare and usually a late complication of renal tuberculosis. It commonly leads to severe and extensive stricture formation.

Secondary infection must be regarded as a late complication and even modern antibiotics may fail to eliminate it. It is commoner in cases with dilated upper urinary tracts which become distended during the act of micturition and thus maintain a residual urine. It is noteworthy how the infecting organisms seldom remain constant and tend to change from time to time. They may lead to recurrent attacks of pyelonephritis or to secondary pyonephrosis with severe toxaemia, necessitating nephrectomy where the other kidney is adequate. In case 5, F.J.S.K. (Fig. 8), secondary infection which developed after repeated cystograms could not be eradicated. The urine became alkaline and on repeated occasions soft phosphatic calculi were removed from the bladder *via* the cystoscope.

Medical Treatment

Specific therapy, although of great value, may lead to the rapid development of changes resembling the late complications which normally take years to develop. This has never been seen to happen in early cases and fortunately only in a minority of the chronic cases. Jacobs[4] refers to this increased cicatrization associated with the use of streptomycin. The illustrations of the following 3 cases showing these changes have been reproduced from an article of mine in the *British Journal of Urology*.[5] In case 6, Mrs. E.A.B. (Figs. 9, 10 and 11), approximately 6 months after treatment with streptomycin and PAS, marked dilation of the upper renal tract developed with associated reflux up the ureter. A stricture was excluded by the passage of a large catheter up the ureter. In case 7, Miss V. M. (Figs. 12, 13 and 14) within 3½ months after therapy with streptomycin and PAS, marked dilation of the upper urinary tract occurred, associated with reflux and further contraction of the bladder. Here also stricture of the ureter was excluded by the passage of bulbs. Both these cases had already undergone a nephrectomy for renal tuberculosis. In case 8, Mrs. M.T., (Figs. 15, 16 and 17) after 6 months' therapy with streptomycin and PAS marked contraction of the bladder took place, without reflux or dilation of the ureter. Instead there was an exacerbation of a previous stricture of the ureter, leading to marked renal deterioration and uraemia. This case suggests that whether, on the one hand, reflux and dilation or, on the other hand, stricture formation develops, depends on the resultant of two opposing forces, viz. (1) the dilating action of the spastic bladder, and (2) the degree and strength of the fibrotic contracture of the ureter. Thus if we compare the pyelograms in case 1 (Fig. 1) with those in cases 3 and 5 (Figs. 5 and 8), we may speculate that the reason why no stricture formation took place in the latter two cases is the dilating action by the reflux of urine produced by the spastic bladder. The reason for the rapid development of the above complications is put down to the increased destruction of tubercle bacilli and liberation of toxin leading in the first place to exacerbation of the inflammatory process and

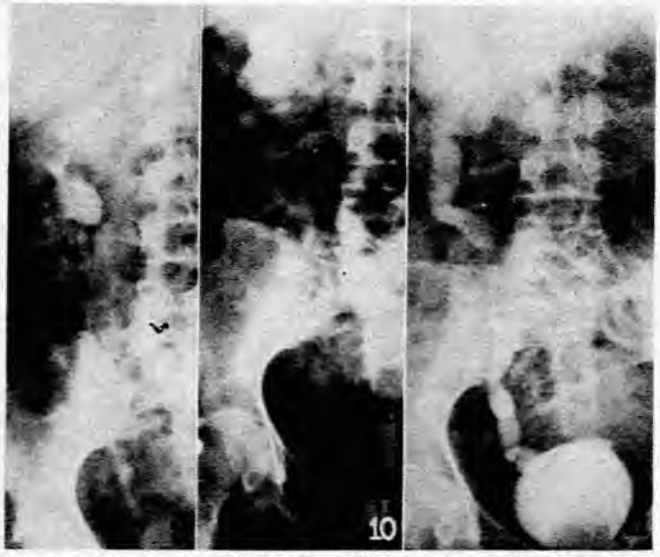


Fig. 9. Case 6, E.A.B. 19 July 1950. I.V.P. *Fig.* 10. Case 6, E.A.B. 8 March 1951, I.V.P. *Fig.* 11. Case 6, E.A.B. 19 July 1951. Showing cystogram with reflux up the ureter. (*Figs.* 9, 10, and 11 were published in the *British Journal of Urology*.[5])

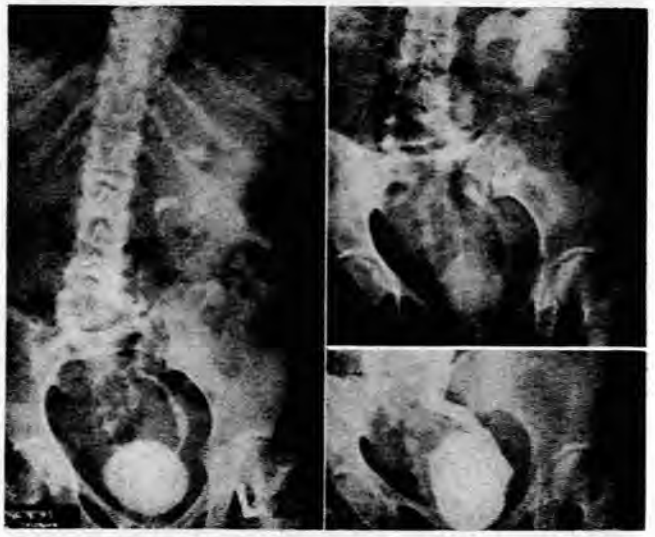


Fig. 12. Case 7, V.M. 28 February 1951. I.V.P. *Fig.* 13. Case 7, V.M. 18 June 1951. I.V.P. *Fig.* 14. Case 7, V.M. 11 July 1951. Showing cystogram with reflux up ureter. (*Figs.* 12, 13, and 14 were published in the *British Journal of Urology*.[5])

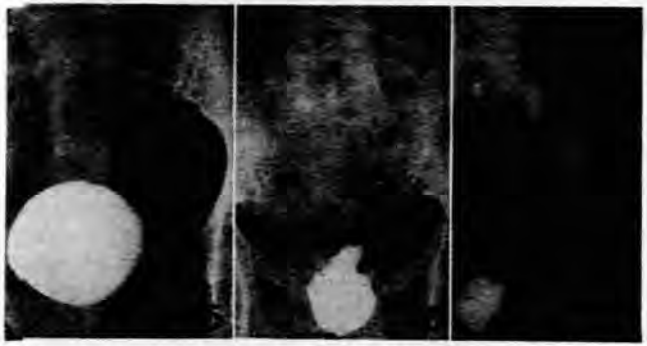


Fig. 15. Case 8, M.T. 3 January 1952. Cystogram. *Fig.* 16. Case 8, M.T. 18 July 1952. Cystogram. *Fig.* 17. Case 8, M.T. 23 July 1952. Retrograde ureterogram showing stricture of ureter. (*Figs.* 15, 16 and 17 were published in the *British Journal of Urology*.[5])

subsequently to increased fibrosis, but why this should occur in only a small proportion of cases is unknown. Secondary infection may be a factor, but is not always present. As regards the value of the various specific drugs, Sporer and Oppenheimer[6] report that INH has the greatest penetrant action in a caseous lesion and PAS the least, and also that INH causes increased vascularity and diminished fibrosis.

I have tried cortisone therapy in 4 cases in attempting to lessen the fibrotic changes, but have not been impressed by its value. Once these changes have been established the subsequent course is usually one of slow deterioration, but they may remain stationary or show some regression to a more normal appearance.

SURGICAL TREATMENT

1. In so-called 'auto-nephrectomy' with a partially calcified tuberculous pyonephrosis, nephrectomy is advisable.

2. In the small contracted bladder with reflux, there are a number of alternative procedures available, viz:

(*a*) Uretero-intestinal anastomosis to the large bowel. This cannot be recommended owing to the grave risk of ascending pyelo-nephritis.

(*b*) Cutaneous ureterostomy. The risk here is stricture formation near the skin, necessitating an indwelling catheter which leads to secondary infection. Three of the above cases were treated with cutaneous ureterostomy. Two died from uraemia approximately 1 and 2 years later. The third is still alive and well 7 years later. He is working full-time and has recently been promoted.

(*c*) Cutaneous uretero-ileostomy, and

(*d*) Ileo-cystoplasty. The latter two procedures are of considerable value, but I have no personal experience of them.

3. Where there is evidence that the lesion is localized to one pole of a remaining kidney, partial nephrectomy should be considered before the train of complications described above takes place (see case 1).

SUMMARY

1. The main late complications of renal tuberculosis are due to fibrotic contracture and deformity.

2. This leads to stricture formation in the upper renal tract with dilation above it and consequent renal destruction.

3. The contracture of the bladder, however, is usually associated with reflux up the ureter and associated dilation of the whole upper renal tract.

4. These changes may occasionally be exacerbated by specific therapy.

5. It is suggested that the persistent focus which maintains the disease after unilateral nephrectomy is usually in the kidney, occasionally in the bladder and very rarely in the stump of the ureter.

6. Various surgical measures utilized in these late complications are briefly referred to.

REFERENCES

1. Pyrah, R. H. and Raper, F. P. (1958): Société internationale d'Urologie Tome I, pp. 67-92.
2. Savegh, E. S. and Dimmette, R. H. (1956): J. Urol., 75, 671.
3. Veenema, R. J. and Lattimer, J. K. (1957): *Ibid.*, 78, 65.
4. Jacobs, A. (1952): Postgrad. Med. J. 28, 78.
5. Scher, S. (1953): Brit. J. Urol., 25, 103.
6. Sporer, A. and Oppenheimer, G. (1957): J. Urol. 78, 278.