RENAL TUBERCULOSIS.

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INTRODUCTION.

The writer's objective in preparing this article has been to provide a simple statement of the principal features of urinary tuberculosis and to indicate the factors which should guide in diagnosis and treatment. Controversial matter has been avoided. Tuberculosis of the urinary tract is sufficiently common in any urological practice but will probably not occur with much frequency in a given general practice. It appears to be much less common in Britain than on the Continent. It is, however, a disease to be kept ever in mind because of the ravages which it produces in the bladder etc., if allowed to progress unchecked.

Renal tuberculosis is a good subject for a special article in as much as it is a disease *sui generis* and its pathological and clinical behaviour cannot be foreseen as a result of a knowledge of surgical principles. On the other hand, it is so lengthy a subject that adequate room cannot be allowed it in a general text book on surgery.

PATHOLOGY.

There are five pathological points which require discussion:—

- (1) Which is the organ of the urinary tract first invaded?
- (2) How does the infection reach it?
- (3) The macroscopical and
- (4) The microscopical appearances presented by the kidney.
- (5) The method of spread of the disease within the tract.

(1). **Which is the Organ first invaded?** For a long time our knowledge of the pathology of this disease was chaotic, the basic misconception being that the bladder was primarily involved and that the kidney infection was a secondary one. A quarter of a century ago this view became suspect, and evidence began to accumulate that the kidney was almost invariably the organ to be first involved and that the infection travelled to other parts of the urinary tract subsequently. This view is firmly held at the present day and rests on the following principal bases:—

(a) The finding of a lesion within the kidney in certain cases before there is any other discoverable lesion in the remainder of the tract.

(b) The fact that the degree of advancement of the renal disease points, in many cases, to its having preceded that in any other part of the system.

(c) The knowledge that after nephrectomy there is a strong tendency for the bladder to heal and that in many cases healing is complete, but if it is incomplete that there is at least an amelioration of the condition.

(2). **How does Infection reach the Kidney?** It is obvious that with an organ which is so highly vascular as the kidney, an organ which acts as a filter for the blood and through which the whole of the blood of the body passes many times an hour, there is considerable chance of the infection being a blood-borne one. By analogy also it might be argued that, as many of the simple infections are hæmatogenous, so would the tuberculous lesion be blood-borne. The difficulty has been to explain why it should be a unilateral condition, for in nine out of ten cases the disease is confined to one side. Because of this difficulty the hæmatogenous theory has had to compete with a lymphogenous theory. In this latter it was suggested that the kidney was infected by retrograde passage of bacilli along lymphatic channels from the lung. This view is now completely discredited. It was founded on a few unsatisfactory clinical and post-mortem observations but on no experimental work. It has nevertheless survived in text books for a surprisingly long period. How then do we account for the one-sidedness of the disease? We will postpone the answer to this question until we have looked into another problem.

Frequently in the surgical literature on renal tuberculosis it has been reiterated that no solitary instance can be produced in which a tuberculous focus has healed in the kidney. This statement, so far as I know, owes its origin to Albarran, but it has been a guiding principle in surgical urology up to the present time, there being almost absolute agreement that in unilateral disease the offending kidney should be removed. So confidently has this view been held by urologists that a challenge issued twenty years ago in Chicago offering "to eat any kidney which would show healed renal tuberculosis" has never been taken up (Cabot). Recently, however, evidence has been accumulating that tuberculous lesions of the kidney can and do heal, and an altered attitude to the pathology of this disease is making itself evident. Investigations have for some time been progressing from three standpoints, the post-mortem, the clinical, and the animal-experimental. A prominent position in this work must be allotted to Medlar who has been a pioneer in the field and who has made important contributions both to the investigation of post-mortem material and also to the experimental aspect of the question. It now appears that bacilli circulating in the blood descend on the kidney and cause microscopic lesions which are quite unrecognizable unless serial sections are cut. The vast majority of these minute lesions heal and fibrose, leaving no macroscopic evidence of their previous existence. Occasionally one of them runs an unfavourable course and develops. It is generally in the cortico-medullary region of the kidney that this happens. Once it has advanced so far as to become a naked eye lesion there is no turning back. Provided the patient survives long enough, it will run one of a variety of courses (which will be described later) but all of them have ultimately the same end—the destruction of the organ as a gland. It would appear that instead of the kidney offering no resistance whatsoever to the development of tuberculosis, as previously believed, it actually in the first instance presents a very high resistance, but that once a focus has got out of hand it proceeds unchecked and never heals. This makes clear the reason why renal tuberculosis presents itself as a unilateral disease. The original bacillæmia probably caused multiple lesions in both kidneys but all were overcome in one of the kidneys and presumably all except an odd one in the affected organ.

(3). **Macroscopic Conditions.** These are very diverse according to the course run by the disease, which depends, amongst other things, on the virulence of the infection, the susceptibility of the patient, the tendency to calcification, and

FIG. 1.
Congenital cystic kidneys.

Ureteric pyelogram showing extreme lengthening and narrowing of pelvis. One or two misshaped calices are seen, the rest being represented by round or oval spaces at the end of long narrow branches of the pelvis. From a girl aet 12 years. The kidneys filled practically the whole abdomen, the left being the larger and extending into the pelvis.

FIG. 2.
Photograph of specimen of congenital stricture of upper end of ureter.

FIG. 3.
Pyelogram of double hydronephrosis "Intermittent type." The catheters have been prevented from entering the pelvis by strictures at the upper ends of the ureters. Male aet 9½ years.

FIG. 4.
Open type of hydronephrosis. Cystogram showing great dilatation of left ureter and kidney. The right kidney and ureter which are in a similar condition are only partly shown. Male aet 10 years.

FIG. 5.
Ureterocele.

Drawing of post-mortem specimen. Double ureter on right side, and ureterocele (A) projecting into bladder. The ureterocele is connected with the dilated ureter. There is also some dilatation of left ureter.

FIG. 6.

Photograph of post-mortem specimen. Congenital valve of urethra, shown in front of probe (A). Dilatation of prostatic urethra. Back pressure bladder, ureters and kidneys. Note hypertrophy of bladder. Male aet 4 years.

FIG. 7.

Enlarged photograph from previous figure to show membranous valve at lower end of prostatic urethra. Bougie behind valve. Urethra opened on dorsal surface.

FIG. 8.

Congenital stricture of female urethra. Cystogram showing dilatation of bladder and lower ends of ureters and proximal half of urethra. Female aet 4 years.

FIG. 9.

Uroselectan B. urograph in a female aet 23 years who had been treated for many years as case of simple chronic *B. coli* pyelitis. Not only was the right kidney heavily infected and the seat of calculi but both kidneys were hydronephrotic, proved at operation to be caused by aberrant renal vessels.

FIG. 10.

Radiogram taken in lateral plane to show the superimposition of the renal upon the bodies of the lumbar vertebrae.

FIG. 11.

Shadow of a gallstone well in front of the vertebral column.

FIG. 12.

A shadow in the line of the left ureter in ilio-vertebral space which might be mistaken for a ureteric stone. Note the irregular density of the shadow due to a calcareous mesenteric gland.

FIG. 13.
A calculus situated in the terminal left ureter.

FIG. 14.
The same case after the passage of a radiographic catheter showing slight alteration in the position of the stone, but which was proved to be a ureteric calculus by a stereoscopic pair.

FIG. 15.
The shadow of a vesical calculus.

FIG. 16.
This left vas deferens (A) and seminal vesicle (C) injected with 15% solution of Sodium Iodide. The injection has been made into the proximal end of the cut vas in the upper part of the scrotum. Note that the prostatic urethra (B) has been distended with the Sodium Iodide. The shadow (D) above that of the S.V. is the excess of the solution which has flowed out of the prostatic urethra.

the degree of obstruction which develops in the ureter. It would be impossible in the available space to describe all the varieties and I propose therefore to confine myself to the common ulcero-cavernous type and to regard this as a standard from which the others are merely variants. It is exceedingly important to get a clear conception of the macroscopic pathological changes which occur in the kidney and urinary tract, because the diagnosis by cystoscopic and radiographic means hangs almost exclusively on these facts, and the findings cannot be correctly interpreted unless the surgeon is familiar with the naked eye pathological changes. I shall therefore not apologise for entering into these somewhat fully.

In the kidney we have seen that the original focus is situated at the base of a pyramid. For some unexplained reason it also exhibits a preference for the poles of the kidney. This focus passes through the usual changes—caseation, suppuration, etc.—and in due course ruptures into the pelvis. There is from this point onwards a continuous supply of tubercle bacilli to the pelvis, and pus and bacilli may henceforth be found in the urine in greater or less quantities. Sooner or later the pelvis itself is infected and submucosal tubercles form which ultimately go on to ulceration. The whole pelvis becomes affected and, whilst this is happening, the disease attacks the papillæ and a return flow of infection to the interior of the kidney sets in. If a kidney at this relatively early stage is microscopied, it will be found that everywhere there are lymphatic lines of tuberculosis extending through the parenchyma to the capsule, under which they radiate and from which they send off-shoots into the peri-renal fat. Some of those in the renal parenchyma go on to the formation of fresh “caverns,” so that the disease is by no means limited to the site of the original focus. In this way multiple cavities which have eroded the renal medulla drain their contents into the pelvis—the ulcero-cavernous variety. Such is the commonest type of renal tuberculosis, but the disease is very polymorphic and the following are amongst the most important variants:—

(a) *The pelvic type.* Manifestations in the pelvis are very pronounced and this leads to considerable ulceration of the papillæ. Sometimes the pelvic type causes severe hæmaturia.

(b) *The chronic type.* A high resistance on the part of the kidney leads to fibrosis of that organ. The pus may be thick and creamy and in some cases calcification is added—always a sign of chronicity. In the chronic type stenosis of the ureter readily occurs and the above mentioned thick creamy pus accumulates in the kidney. The contents of the kidney are very prone to undergo calcification and the so-called “putty” kidney results. This type is easily recognizable in the radiogram.

(c) When sepsis supervenes on the tuberculous process a very rapid destruction of the parenchyma takes place and the purulent contents are thin and may be foetid.

(4) **Microscopic Conditions** are not important from the clinical aspect and for this reason I shall not discuss them.

(5) **Spread within the Tract.** The *ureter* undergoes a process similar to that seen in the pelvis. The changes produced by this on the ureter are first of all *dilatation*, which is due to the muscular invasion leading to dysfunction. The wall of the ureter becomes oedematous and fibrotic. It is therefore palpably thickened and this thickening frequently goes on in combination with a dilatation of the lumen. Later sclerosis and *contraction* of the ureter set in. They manifest themselves in two directions. The first is in the form of a stricture which may be localized to one small area, or may obliterate the lumen over a longish distance.

An even more constant contraction occurs in the long axis of the tube so that its total length is diminished, a fact which is recognizable, as we shall see later, both by cystoscopy and on a urogram.

The bladder. Probably the bladder is invaded somewhat later than the pelvis and ureter. The invasion occurs as the result of (i) the constant stream of infected urine which pours over the trigone and (ii) it occurs directly from the infected intra-mural ureter. That section of the ureter which lies within the bladder wall is amongst the most severely damaged portions of the tube. It does not take long before the thin curtain of tissue separating the ureter from the bladder is eaten through and the disease presents within the bladder. This fact gives one reason for the position of the earliest manifestations of tuberculosis. They are above and external to the ureteric mouth and have reached this situation by direct permeation of the valve-like cusp which exists here. It is not long before the thin flap ulcerates away, the ureteral opening now appearing as an irregular, crateriform, gaping orifice with moth-eaten margins. Small necrotic tags of debris adhere to it. From here the disease spreads chiefly to the fundus and middle of the bladder. The trigone is more resistant. It also spreads to the roof of the bladder by contact when the bladder is empty. I do not propose to describe the evolution of tubercles in this mucous membrane. They pass through caseation, rupture and ulceration. An important fact, however, is that they extend to the deeper coats of the bladder, and this is the cause of that rapidly progressive diminution in the capacity of the bladder which is so constant a feature of this disease. The bladder becomes hypertonic and will accept only seven, five, three or even fewer ounces of urine.

SYMPTOMS.

The majority of the symptoms of renal tuberculosis are produced by the disease in the bladder, the kidney itself remaining silent, not only at the beginning but frequently throughout the whole course of the disease. It is only in about 15% of cases that at any time throughout the illness the kidney breaks its silence. This 15% is equally divided between two types of pain; (a) in which transitory pain occurs, a small morsel of pus, calcareous debris, or blood clot temporarily blocking the ureter and giving rise to colic; and (b) in which perirenal inflammation occurs and produces localized and usually quite unimportant loin pain. When present, renal pain should be carefully noted because it may be an indicator of the site of the original mischief, but as it is absent in 85% of the cases we generally get no guidance from this source. Moreover, it may prove an unsafe guide for it is not very uncommon for the opposite kidney, carrying a double responsibility and moreover striving to hypertrophy to shoulder the additional burden, to become a tender and perhaps even a painful organ.

In the typical case all complaints are referred by the patient to the bladder and there are few symptoms or none at all until that organ is involved. During the prodromal period it is true that there is known to be some degree of polyuria, and pyuria + bacilluria will be found if the urine happens to be examined at this stage, but they are usually overlooked as there is no call to examine the urine. The symptoms when they do arise are those of cystitis. Can we by symptomatology distinguish tuberculous cystitis from other forms of bladder inflammation? Amongst the points helping in this differentiation are:—

(a) The insidious nature of the onset. The patient cannot tell to a month or two when he started to have bladder irritation and in this way the disease singles itself out, at least from those acute invasions which have a sudden onset.

(b) Once established the evolution is steady and is but little influenced by treatment. It progresses to an extreme degree of vesical irritation and tenesmus, so that the pain and frequency of micturition eventually become intolerable—this complaint being much more emphatic than it is in simple cystitis.

For the rest, the illness generally, though not always, runs an afebrile course and the deterioration which it causes in the patient's health is remarkably small. Males and females are equally affected and the two kidneys are involved in like proportions. The age incidence, however, is more significant. Marion gives the following details. In 1393 cases he found that

before	20 years of age there were					185	cases
between 20 and 40	„	„	„	„	„	933	„
„ 40 „ 50	„	„	„	„	„	175	„
50 and over						100	„
						<hr/> 1393	

These figures show that the third and fourth decades are the periods of election. Below the age of fifteen and above the age of sixty I find that it is a rare disease. The patients are therefore as a rule young and middle-aged adults.

Occasionally atypical varieties of onset are witnessed. Hæmorrhage, as an initial symptom, is in my experience rare but nevertheless is well recognized as such. When it occurs it is usually discovered at nephrectomy that the pathological type is that in which the papillæ are severely affected. Tubercle bacilli are found in profusion in this type so that the unusual onset does not mislead. Severe hæmorrhage may also come on at a later time in the disease and I have seen it from a kidney of the "putty" type, the focus of origin in the kidney being quite evident on examination of the nephrectomized specimen and involving the cortico-medullary region and probably the remnants of the arcuate vessels.

Other recognized but atypical forms of onset are incontinence, renal pain, and polyuria, but, these need not delay us.

INVESTIGATION OF CASE.

When the usual symptoms of tuberculous cystitis present themselves, they are very suggestive though not diagnostic. They lead to a determined search for the tubercle bacillus and once that organism has been found the whole aspect of the case changes. It is well known that the tubercle bacillus is not easily discovered in urine and in passing I would recommend those who are interested in the search for this organism (not only in urine but also in sputum and other body fluids) to try the method of Ellerman and Erlindson which is used in my department, whereby the surrounding organic matter is completely digested away, leaving as a precipitate only the isolated bacilli which are thus much more easy to identify.

Having found tubercle bacilli the experienced practitioner lays a plan of campaign. He knows that one kidney is diseased and hopes to find the other healthy. He expects to remove the tuberculous organ but must first find out which it is and must then satisfy himself that the other kidney is completely healthy. How shall he proceed? There are two possible lines of action:—

- (1). Through cystoscopy, separate renal function examinations, examinations of the separated urines and instrumental pyelography.
- (2). Through excretion urography.

Retrograde pyelography is the older and has stood the test of time. It is more thorough, giving much detailed information which cannot be obtained by excretion urography. On the other hand, it is a more formidable and troublesome procedure especially as the bladder is a small, irritable, and intolerant viscus when infected by Koch's bacillus.

I. Cystoscopy and associated Examinations.

The reduced capacity of the bladder announces itself during bladder preparation and its refusal to take the usual measure of lotion is very emphatically expressed. An attempt to overdistend a tuberculous bladder will probably prove fatal to the whole examination and may necessitate its abandonment because of increased irritability or hæmorrhage. It is a good plan to discover the maximum capacity of the bladder by gradually working up to it and, having once sensed it, never again to ask the bladder to hold its full measure.

The characteristic lesion of tuberculosis is the tubercle, and when the circumstances are favourable tubercles may be seen through the cystoscope. They consist of small, convex, yellowish spots surrounded by a red areola and tend to grow in clusters. They are most often seen near the ureter and frequently group themselves at the bifurcation of a blood vessel. When ulceration has taken place tubercles may also sometimes be seen on the fringe of the ulcer.

Actually it is only in a minority of cases that you will find any tubercles at all within the bladder, so that their absence must not be regarded as negating the diagnosis. The customary findings are:—

(i) *The reduced bladder.* The viscus is but little larger than an egg. All its walls are therefore close to the lamp and to the prism and are thus brightly illuminated and (in the absence of a muddy medium, which is quite customary) the details of the mucosa are easily seen. The bladder is globular and it is obvious to the observer that it is full, i.e., its small size is not due to underdistension.

(ii) *Cystitis* is generally well developed. In early cases it is localized to the neighbourhood of the infected ureter. It spreads to other areas later and may become ubiquitous. Secondary infections are common and tend to make the cystitis generalized.

(iii) *Ulceration.* The tuberculous ulcer is found in the bladder but it is not always easy to recognize its features when seen under water. The red base may merge into the red areola which surrounds it and the transition is not obvious. Ulceration, like its forerunner the tubercle, is most common in the precincts of the ureter. It should be looked for carefully because it is a very suggestive discovery.

(iv) The ureteric meatus suffers changes which have in part been described, the formation of tubercles, ulceration, and the loss of the ureteric cusp do not require further mention. But an important change results when the ureter commences to contract. The shortening of this tube brings about the retraction of the ureteric orifice which is almost pathognomonic of the disease and which has come to be known as the "golf hole" ureter. In contracting, the ureter pulls its point of insertion into the bladder to a higher level. The trigone thus, becomes asymmetrical and the affected ureter comes to lie quite considerably higher than previously.

(v) The cystoscopist has already in many cases, though not by any means in all, gained some idea of the side affected. An injection of indigo-carmin (.4%) is now made into a vein at the bend of the elbow. If one kidney is healthy the dye will be seen emerging from the corresponding ureter in $3\frac{1}{2}$ to 5 minutes. It will be delayed and may never emerge from the side of the disease, thus strongly

confirming the suspicions already arrived at, and, as it were, giving the surgeon permission to proceed to nephrectomy.

(vi) The ureters may now be catheterized. From the *healthy* kidney urine of good quality—good specific gravity, clear, free from pus or organisms—will be collected. A trace of albumin may be present, but if inconsiderable it may be ignored as the sister organ not infrequently shews a “sympathetic” nephritis, which clears up after the removal of its diseased fellow. The albumin may also result from traumatic blood due to catheterization. From the *diseased* kidney will be obtained urine containing pathological material such as pus, blood, tubercle bacilli, albumin, etc. mixed up with urine of inferior quality.

(vii) An *ascending urogram* is now made, particular care being exercised to avoid the least overdilatation of this greatly diseased organ. The shadow obtained shows up the pathological changes which have been detailed above. Of these an important early change is the inflammatory dilatation of the pelvis and ureter and it can be detected with remarkable precision. The next thing to look for is erosion of the parenchyma where the ulcerated areas exist. Of course if the pelvis itself or the cavities are full of semisolid debris this will modify the picture, but granted a knowledge of the alteration which the disease is capable of effecting in the kidney, the urogram is not difficult to interpret. The changes in the ureter are also various from dilatation to stricture formation but are generally coarse in their development and easily assessed.

II. Excretion Urography.

The alternative method of diagnosis is *excretion urography* and its evidence can be extremely valuable in cases where the bladder conditions are very bad. On the *healthy* side it shows a normally shaped pelvis and ureter and indicates the level of renal function.

On the *diseased* side it will, so long as sufficient excretory power remains to throw a satisfactory shadow, show changes in the contour of the pelvis similar to those described above. The shadows are however less intense and therefore less easy to read.

The first method gives indications which are more precise and reliable especially in reference to the presence of infection in the presumed healthy kidney. Excretion urography may be relied on solely when the cystitis is exceptionally severe. It is also a valuable ally prior to cystoscopy for it quite frequently gives the cystoscopist certain knowledge which may speed the actual cystoscopy.

TREATMENT.

Given a good kidney on the other side, the treatment of renal tuberculosis may be summed up in the word *nephrectomy* and this proceeds on the usual lines with only a few points which call for discussion.

The exterior of the kidney when delivered into the wound in quite a large number of cases betrays no sign whatsoever of being diseased. The inexperienced operator may feel some compunction in removing such an organ. What shall he do? On no account must he open into it. This is fundamental. One of the worst mistakes that can be made in the surgery of renal tuberculosis is to open up the kidney and spill bacilli into the wound. If this happens, the wound in the majority of cases breaks down within a fortnight or three weeks, leaving a gaping cavern which may take months to heal. Accidental rupture of a tuberculous focus occurs on occasion in operations on difficult or adherent kidneys. In my experience it is very uncommon, but when it does take place I institute a

most painstaking toilet of the wound, sponging it out with ether and subsequently with acriflavine. These precautions may be successful in avoiding infection and a breakdown of the wound, but they presuppose a knowledge of impending danger. For identical reasons the greatest care is exercised in the ligature and cautery (pure carbolic) of the ureteric stump.

To come back to my starting point the surgeon must never open the kidney for diagnostic purposes. Let him mobilize the organ fully and expose the ureter. Some change will be discoverable in this structure in almost all cases and will confirm the fact that this is the diseased kidney. When least well marked the ureteric change takes the form of oedema and minor thickening of the tube. When better marked the thickening, fibrosis and shortening are very evident. If no external change can be discovered the surgeon, who should be satisfied before he embarks on an operation that his evidence is unimpeachable, will, with complete confidence, proceed to nephrectomy.

The treatment of the ureter is a subject on which surgical opinion is still divided. Some surgeons believe in taking out the whole length of the ureter. Others confine their operation to the removal of the upper end which can be reached from the usual renal incision, and this school maintains that the ureter heals satisfactorily after the removal of the kidney much in the same way as the bladder heals, and that it gives no further trouble. For the most part, the writer shares this view and prefers not to attack the lower ureter, at any rate in the first instance. In the great majority of cases it behaves satisfactorily and the very materially larger operation is avoided. Yet it must be confessed that there are occasions on which the ureter fails to heal, keeps up a supply of bacteria to the bladder, and so requires a secondary ureterectomy. It may be remarked that just as with the vas deferens, the upper and lower thirds of the ureter bear the brunt of the pathological change and that the middle section escapes till a comparatively late stage in the disease. It is easy to get as far as this healthy middle third from a loin incision, whereas to get the lowest section of the tube in its entirety requires a rather extensive operation. However, a study of the ureterogram for evidence of stricture formation or severe ureteric damage should be made, and if such is suspected the full operation should be undertaken.

Some authorities advise that the drainage tube be retained for a longer period than the customary one, alleging that pre-existing lymphatic infection of perirenal fat may cause a breakdown of the wound. For the same reason the removal of this fat has been advised. From a considerable experience the writer is satisfied that no wound breaks down unless the extra-renal tissues have been directly infected from the interior of the urinary tract, and that the special drainage precautions recommended are unnecessary.

RESULTS.

The results of the treatment of urinary tuberculosis vary with the stage at which it comes under notice. The mortality from the operation is small (about 1.5 per cent.) and is practically confined to such cases as have densely adherent kidneys or similar operative disadvantages. In the best cases there is an almost immediate cessation of the frequent micturition but in the average there is steady progress over a period of a few weeks or months after which the condition becomes stabilized. The improvement observed depends mainly on the depth and extent of the vesical ulceration. When this has been severe permanent contraction of the bladder may persist even though the urine is clear and free from organisms. In a few cases the bladder lesion refuses to heal and bacilli are still present in the urine. The question whether this continued infection is really vesical in origin, or comes from the ureter, or is due to a spread of the disease to the remaining kidney must then be considered.