Original Articles.

THE DIAGNOSIS OF RENAL TUBERCULOSIS.*

BY ARTHUR L. CHUTE, M.D., BOSTON.

THIS paper is an expression of my personal ideas of the points that are important in the diagnosis of renal tuberculosis. In it I have set forth the things that I have found useful in recognizing this not uncommon disease.

As a preface to a discussion of the diagnosis of renal tuberculosis, it is necessary to say that the first subjective sign of renal tuberculosis is usually that of bladder irritation, just as the first objective sign is the passage of a turbid urine. Aching or pain in the loin is not an early symptom; in fact, a considerable proportion of cases have but little lumbar pain at any time. It is important, I believe, to emphasize the fact that the first symptoms of renal tuberculosis do not usually point to the kidney, but to the bladder. Though this is a well-recognized fact, it is not always remembered. Within a year a patient suffering from the usual bladder symptoms of renal tuberculosis, and to whom I had proposed the removal of his diseased kidney, consulted one of our best internists as to the wisdom of this procedure. The internist told the patient that he could not see how the removal of a tuberculous kidney could be expected to give relief to his bladder symptoms. The kidney was removed, however, and this man now passes a crystalline urine, usually not getting up at night to urinate.

In this connection I wish also to call attention to the extreme rarity of tuberculosis of the bladder except as it is secondary to renal tuberculosis. In every patient with tuberculous cystitis in whom I have catheterized the ureters, I have found that there was a process above; that the cystitis was purely secondary to a renal tuberculosis. We hear tuberculous cystitis spoken of as though it were a distinct and unrelated pathological process; as though it were often the only tuberculous involvement of the urinary tract. I cannot deny that tuberculous cystitis may not at times exist as the sole tuberculous infection of the urinary tract, but I have never seen it and believe that it is at least very rare. In my opinion, the proportion of cases in which one finds tuberculosis primary in the bladder diminishes in direct ratio to the care with which one examines these cases.

The first signs, then, of a renal tuberculosis will be those of a cystitis. Our task is to consider what things should lead us to suspect that a given cystitis, or the signs of a cystitis, may actually depend upon a tuberculous infection of the kidney.

We will first consider certain things that are brought out in the patient's history. A strong family history of tuberculosis is, without doubt, suggestive; but, on the other hand, the absence of any tuberculous family history is of no particular weight against the probability of tuberculosis, though I have seen a good deal of importance given to it.

The history of a cystitis that made its appearance without evident cause should always be looked upon as suspicious of a possible renal tuberculosis. A cystitis of this sort is more suspicious when it occurs in men than when it occurs in women, since in women we have a certain number of "causeless" infections that ascend the urethra, while in men they are very rare. If the history is that the "causeless" cystitis has tended slowly to get worse rather than to improve under appropriate treatment, it heightens the probability of tuberculosis. A cystitis of this sort that has become very painful will, in a large proportion of cases, prove to be tuberculous. While a history of the insidious onset of a cystitis is suggestive of a tuberculous origin, one may not assume that because there was the history of a definite cause in the beginning that a cystitis of long duration may not have become tuberculous. Both gonorrhea and renal tuberculosis are diseases of young adult life; there is, therefore, no reason why they should not occasionally be coincident. It also seems perfectly reasonable to suppose that a serious gonorrhea may predispose to renal tuberculosis, either through general lowered health or by the crippling, to a greater or less degree, of a kidney by an ascending infection. However that may be, I have seen several cases in which the transition from a gonorrheal cystitis to a renal tuberculosis had been overlooked.

The history of a chronic suppurative joint or of a chronic non-suppurative joint that has ended in ankylosis has some suggestive value. I have seen at least four cases of renal tuberculosis in which there was the history of a previous joint lesion. This joint trouble may have preceded the urinary symptoms by some considerable time; in the last instance of this sort that I saw, the history of the joint trouble went back a long time,—some six or seven years before the patient's first symptom of any urinary trouble. The cases of renal tuberculosis that are secondary to old joint lesions emphasize the fact that renal tuberculosis is probably always a secondary disease, secondary to some other focus of tuberculosis in the body, though it may be impossible to tell what the primary focus was. The recognition of this secondary character of renal tuberculosis is, I believe, important. It should lead us to supervise for some time, in so far as this is possible, the manner of life of any patient from whom we have removed a tuberculous kidney, since it is probable that a primary focus of tuberculosis remains behind and that this focus is capable of doing further harm should the patient's condition be allowed to become enfeebled.

While points brought out by the history may have a good deal of suggestive value, they are never of sufficient value in themselves to warrant one in making a definite diagnosis. They will often, however, be sufficient to draw our attention to the possibility and lead to a careful investigation of the patient.

Once our suspicions have been aroused that a patient may be suffering from renal tuberculosis, our first step is to see if we can confirm or disprove

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this. Some things that are of slight suggestive value are to be brought out by a general examination. The finding of scars or other evidence of an old tuberculous process, such, for instance, as a tuberculosis of a testis, or the finding of any active tuberculous process, is suggestive that the urinary process is also tuberculous.

Emaciation, if it be considerable, is in favor of this tuberculous nature of a urinary suppuration. The grade of emaciation, however, that is suggestive of tuberculosis usually occurs so late that it is of little practical use as a diagnostic sign. In the early stages of renal tuberculosis there is often no appreciable loss of weight, and this condition may persist even when the process is well developed. I have even seen two distinctly fat patients with renal tuberculosis.

Most patients present very little temperature. A good portion of a kidney may be honeycombed with tuberculous abscesses, some of which may be under tension, or at least not draining, and yet there may be no appreciable rise of temperature. Temperature is supposed to be seen most often with mixed infections, though I have recently seen a man who has at times had a consid-

erable temperature and yet whose urine showed no mixed infection, even on cultural investigation. It may certainly be said that the absence of a temperature is not against tuberculosis.

Another sign of less value that depends on this same thing, that is, the absence in the urine of the common bacteria, is a purulent urine that after sedimentation by gravity leaves a perfectly transparent layer of supernatant fluid. In the ordinary purulent urine the bacteria multiply so that this layer is always at least a little murky. As I have said, the urine for either of these tests must be aseptically drawn and preserved. Both of the above signs naturally fail when there is a mixed infection, as there sometimes is. It is, however, surprising to see the proportion of tuberculous cases in which, even in spite of repeated examinations and sometimes considerable local treatment, there is no mixed infection. For this reason this test of a purulent urine that shows no organisms is of use in a considerable proportion of cases.

In the cases that show a mixed infection, and, in fact, in most cases, we will be better satisfied of the tuberculous nature of the process if we find red-staining acid-fast bacilli in the urinary sediment or produce tuberculosis by the inoculation of the sediment into guinea pigs.

I feel that the finding of red-staining acid-fast bacilli in catheter specimens of urine is definite proof of the tuberculous nature of the process. I have had many guinea pig inoculations made of such urines and the result has always been positive. I remember, however, a case that I saw some years ago in which a voided specimen of urine showed red-staining acid-fast bacilli and in which the process was not tuberculous. These bacilli were probably smegma bacilli.

The inoculation of guinea pigs and the recovery of tubercle bacilli from their glands and other lesions is, I believe, without question the most accurate way of determining the tuberculous nature of a urinary infection. I believe, however, that the finding of sterile pus or of acid-fast bacilli in a catheter specimen of urine is pretty definite proof.

When one finds a urine that contains tubercle bacilli and inflammatory products, he is justified in assuming that he has to do with not only a tuberculosis, but with a renal tuberculosis. It is so rare to have a tuberculous cystitis that is not secondary to a renal tuberculosis that we need hardly consider this theoretical possibility, but may assume that a tuberculous urine means practically invariably a renal tuberculosis.

I spoke of the necessity of finding inflammatory products in connection with tubercle bacilli in order to make a diagnosis of tuberculosis. I wish particularly to insist that the finding of tubercle bacilli alone is not enough to establish a diagnosis of tuberculosis. It is, to be sure, highly improbable that we can have tuberculous bacteriurias, in the way that we have bacteriurias with some

CHART I.

erable temperature and yet whose urine showed no mixed infection, even on cultural investigation. It may certainly be said that the absence of a temperature is not against tuberculosis.

I have only exceptionally seen cases of renal tuberculosis present the marked "up and down" temperature that one often sees with other tuberculous infections, and this has been, I think, only in very late and advanced cases.

While the things brought out by a general examination have some value, the question as to whether a given process is tuberculous or not will usually be settled by a study of the urine. For this purpose it is necessary that the specimen of urine examined be aseptically drawn and uncontaminated. A point that is very suggestive of tuberculosis, one may almost say definitely diag-

of the rapidly proliferating pyogenic organisms, but I see no reason why tubercle bacilli should not at times be eliminated by the kidneys, at least in sufficient quantity to produce positive results in guinea pigs, without there being a renal tuberculosis. For this reason I believe that the presence of inflammatory products is necessary to determine that the tubercle bacilli are attacking the tissues and, therefore, producing a definite tuberculosis. I shall have occasion to refer to the practical application of this a little later on.

After we have settled to our satisfaction that a given urinary infection is tuberculous, and drawn the inference from the rarity of primary bladder tuberculosis that the process is probably renal, we still have to establish this last fact beyond a doubt and prove definitely which side is infected. Not only this, but to apply our most satisfactory therapeutic measure (nephrectomy), we must have definite assurance that the process is limited to one side. Both these latter points are determined by the examination of the urinary tract, and incidental to establishing these we gather considerable accumulative evidence as to the nature of the process. If we have already determined definitely that the process is tuberculous, this cumulative evidence is of little importance, but in cases of mixed infection, for instance, where we are awaiting the outcome of guinea-pig inoculations, this additional evidence will be of great interest at least, and often of considerable help.

The local examination of the urinary tract is carried out by means of cystoscopy. This procedure does not differ here from the procedure as used in other sorts of disease, with the exception that it is usually more painful, and will, therefore, more often require ether. Some of the pictures are quite typical. Thus a number of small military tubercles *about one ureteric orifice* are, I believe, definite evidence of a renal tuberculosis. In order to have these tubercles typical they must have little inflammatory areolas about them; otherwise the cysts seen in some types of cystitis may be mistaken for them. If there are small ulcers where the superficial layer of mucous membrane has been destroyed by the breaking down of these little tubercles, they make the appearance more typical. Only the most superficial inspection can mistake the little bubbles that occasionally appear on the prism of the cystoscope for submucous tubercles. These bubbles, of course, have no areola and bear the same relative position when the cystoscope is moved. If these tubercles or ulcerations are grouped about one ureter, they not only allow us to make a diagnosis, but furnish us with the means of pretty definitely localizing the process as well.

When the ulceration of the bladder becomes generalized, there is nothing to distinguish a tuberculous ulceration from one of some other origin, unless at the advancing edge one finds tubercles. An ulcerated bladder that is hypersensitive to distention is very suggestive of tuberculosis, but one occasionally sees equally sensitive bladders that are not tuberculous. Not all cases

of renal tuberculosis, however, show an ulceration of the bladder. In a few one may see only a little redness about the affected ureter — no more than would accompany a sub-acute grade of pyelitis. There is nothing characteristic of tuberculosis about this. In other of the non-ulcerated bladders the ureteric orifice is gaping and irregular in contour, due to an infiltration of the ureter or region of the bladder about its orifice. Where there is no ulceration about a gaping tuberculous ureter, it shows nothing to definitely distinguish it from the gaping ureters seen in other types of renal suppuration, though I think the tuberculous type is more apt to be irregular and jagged than round. When tubercle bacilli have been found in the urine, a gaping ureter, even if not typically tuberculous, serves to localize the seat of the process.

The thickening of the ureter that makes it possible to feel it by vagina is due to the same infiltration of its walls, and I can remember some instances in which a gaping ureter has also been felt thickened by vagina. In men, I have been quite *unable* to feel such ureters by rectum.

CHART II.

We have in most cases of renal tuberculosis things that offer pretty definite proof where the process is localized, without having recourse to catheterization of the ureters. Nevertheless, there will be very few of these cases in which we shall not want the knowledge that is to be gained by ureter catheterization, especially that to be gotten by the catheterization of the supposedly well side.

Within a few weeks the importance of this has been impressed anew upon me by finding that a kidney that I had supposed normal, from the appearance of its ureter, was actually infected. Had I catheterized only the ureter I suspected to be tuberculous, I might have been led to remove one of two pretty nearly equally involved kidneys. It may exceptionally be necessary to remove the more diseased kidney in cases of double renal tuberculosis, but we should do this intentionally, with our eyes open to its possible consequences

to remove a kidney unintentionally in double renal tuberculosis is a great calamity. Occasionally we will find that the side on which the ureter looks normal is the side on which the patient has complained of some pain, or on which we have found the kidney enlarged or slightly more tender than on the other side. It is essential, under these conditions, that we get the urine from that side. *Great* increase in size, *great* pain or *great* tenderness in a kidney means disease, but a moderate degree of any or all of these may be the result of a compensatory hypertrophy quite as well as of disease. An examination of the urine, as it comes from the ureter, is the only way to tell, in some instances, which of these conditions we have to deal with.

This leads me to take up certain points about the catheterization of ureters that apply especially to tuberculous cases.

CHART III.

There has been, I think, a pretty general feeling against the catheterization of the well ureter in these cases, but I am inclined to believe that this opposition is for the most part not well founded. While I do not see how any one can deny the possibility of carrying a few bacilli from the bladder into a ureter, I do not believe that a few bacilli on a healthy mucous membrane means the implanting of a tuberculous infection.

My personal experience has led me to believe that this infection cannot occur often; I have never seen it at all. I believe that the advantages to be gained by a definite knowledge of the condition of the supposedly well kidney far outweigh the dangers of infection. While the common pyogenic bladder infections show a considerable tendency to extend upward toward the kidneys, the tuberculous infections are almost always hematogenous and the progression is from above downward.

It is claimed, especially by those who advocate

the catheterization of ureters under air distention, that there is danger of drawing a false conclusion from a catheterization that has been carried out through a water-distended bladder that contains, perhaps, some bacilli. They contend that, especially if pigs are inoculated, one may get a positive test from a few bacilli that accidentally entered the catheter on its passage through the bladder medium. This is a possibility, but it can be guarded against with the greatest ease by taking the precaution to examine the sediment from the supposedly well ureter. It will contain pus if the kidney is tuberculous, a point that I referred to a short time ago. If there is no pus, there is no tuberculous process, and any tubercle bacilli that may be present are accidental.

While I do not hesitate to catheterize the well ureter, I am becoming more careful about catheterizing, as a routine measure, the ureter on the diseased side, especially if that ureter is much diseased. This is generally held to be quite safe, but I am not perfectly convinced that this is so, or at least that it is not quite upsetting at times. It is a common thing to have an elevation of temperature lasting from forty-eight to seventy-two hours after catheterization of the diseased side (see charts); there is likewise often an increase of renal tenderness. As this rise of temperature and tenderness is seldom seen after catheterization of the well side, I do not believe that infection plays any part in it, but that it is due to some stirring up of the tuberculous process. I do not see why harm might not come of this. Further, I do not feel sure that it is not easier than we have believed to push a ureter catheter through a friable, thickened tuberculous ureter. While I can give no definite proof that this has occurred, I have my suspicions that it has happened. I believe it is absolutely impossible that this should happen with a normal ureter; certainly I have seen nothing to make me believe that it has happened.

Catheterizing a much diseased, dilated tuberculous ureter is occasionally absolutely unsuccessful so far as obtaining a specimen of urine goes. With the ureter dilated and the sphincter destroyed, the catheter lies perfectly loose in the ureter; under these conditions it is easier for the urine to flow down beside the catheter than through its relatively small lumen. Any caseous masses entering the catheter increase this tendency for the urine to flow along the catheter rather than through it.

The most satisfactory procedure in the ordinary case is simply to catheterize the supposedly well ureter, collecting the urine synchronously from the diseased side by means of a catheter placed in the bladder. The diseased ureter may be catheterized should there be any reason for so doing.

A urine from the supposedly well kidney that shows no pus allows us to say at once that the kidney is not diseased. One that contains pus will presumably be tuberculous, but this cannot be assumed without finding bacilli or getting positive inoculation tests.

To recapitulate:

The subjective symptoms of renal tuberculosis

are usually those of a cystitis. A causeless cystitis, or a cystitis that began so imperceptibly that the patient cannot definitely fix the time when it really started; a cystitis in a person who had the history of former tuberculous troubles; a cystitis that is rebellious to treatment, or especially painful; all these should make one suspicious that he may have to do with a tuberculous process. The development of tuberculosis in guinea pigs that have been inoculated with the sediment demonstrates absolutely the tuberculous nature of a urinary infection, as does also the finding of red-staining, acid-fast bacilli in catheter specimens of the urine. We are practically sure of the tuberculous nature of a process when we can find no organisms in a purulent urine.

The rarity of tuberculous cystitis, except as it is secondary to renal tuberculosis, warrants us in almost making a diagnosis of renal tuberculosis whenever we find a tuberculous urine.

The most accurate way of telling which kidney is involved is by cystoscopic examination. The changes in the bladder about the ureteric orifice, or the changes in the ureter itself, will usually make clear which kidney is involved. Occasionally these changes may not be decisive and catheterization of the ureters may be necessary to determine which is involved. One should usually catheterize the supposedly well ureter to demonstrate definitely that the process is unilateral and that the well kidney is functionally competent. The danger of this, I believe, is very small when it is carefully done.

RENAL TUBERCULOSIS. PATHOGENESIS AND PATHOLOGY.*

BY LINCOLN DAVIS, M.D., BOSTON.

I HAVE nothing new to contribute to this much studied and important subject, and will attempt only to review briefly its present status as determined by the acknowledged authorities of to-day.

Renal tuberculosis is caused by the bacillus of Koch, which may gain admission to the kidney in three ways: (1) by the blood stream — hematogenous infection; (2) by the urinary passages — ascending urogenous infection; (3) by extension from diseased neighboring tissues — infection by contiguity.

It is now very generally agreed that infection occurs in the first way, that is, by the blood stream, in the great majority of cases — exhaustive clinical, pathological and experimental researches have quite definitely settled this point; also that the kidney is primarily affected, as far as the urinary tract proper is concerned, in the great majority of cases, and that from the kidney the process tends to extend in the direction of the urinary current down the ureter to the bladder, which is involved secondarily. Tuberculosis is probably never actually primary in the kidney in the sense that the initial lesion of the body occurs here, since tubercle bacilli cannot enter it directly from the outside. There are probably always

other and earlier foci of the disease, either in the lungs, bronchial lymph nodes or elsewhere, which may become clinically quiescent, but yet have served as the starting point of an hematogenous infection of the kidney. These primitive foci may become healed or obsolete, and are easily overlooked in a carefully conducted post-mortem examination, thereby accounting for the not rare instances in which the only focus of disease at autopsy appears to be in the kidney. The only other explanation of this anomalous fact is the less plausible supposition that tubercle bacilli may gain admission into the blood current without setting up any local lesions whatever. Whatever the solution of this somewhat academic question may be, the fact remains, as borne out by plentiful post-mortem, clinical and operative proofs, that the chief and essential lesion of tuberculosis in the body lies in one or the other of the kidneys in a very considerable number of cases. Just what the proportion of cases is will be considered later.

Very little is now heard of ascending tuberculosis, by which is meant a process beginning in the bladder and extending up the ureter to the kidney. This was the theory of renal infection ten years ago, founded on a clinical misconception, but taught with conviction by the best authorities. Careful observation has shown that this mode of infection, while theoretically possible, is practically of very rare occurrence. In the first place, genito-urinary tuberculosis almost never begins in the bladder; those cases in which the bladder does become infected without previous involvement of the kidney are usually cases of extension of the disease from the male genitalia; it seems to be impossible for the tubercle bacilli to make their way up the ureter against the urinary stream, and it is only when the ureteric orifice has become involved in the disease, resulting in incompetence of the valve, and stasis, that the ureter and kidney become secondarily infected by direct extension from below. That, under these conditions, such an infection may occur is shown by a remarkable case reported by Rovsing, in which a tuberculous process primary in one kidney descended the ureter of that side to the bladder, extended across the latter to the opposite ureter, which it ascended as far as the second kidney, but without actually infecting that organ. While the authenticity of this case is beyond question, it represents merely the rare exception which exists to every rule. Kapsammer reports a somewhat less conclusive instance of what he regards as ascending infection of the kidney, — one case out of sixty-two studied by him.

I have personally seen two cases in women which I at first regarded as primary tuberculosis of the bladder; prolonged study and, in one case, operation, finally disclosed undoubtedly pre-existing foci of the disease in the kidney.

Infection of the kidney by extension of the disease from contiguous organs, most often the spine, is an uncommon but well-recognized occurrence which presents no particular problems.

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