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.

* Read at a meeting of the New England Branch of the American Urological Association, Feb. 25, 1909.

That tubercle bacilli, in cases of pulmonary tuberculosis or tuberculosis of other organs, may pass through and be excreted by the kidneys, without setting up recognizable lesions in the latter, must be accepted apparently as true. The investigations of Walsh and Flick in this country, and of Küster, Feodorov, Oppel, Fullerton, Hilier and others in Europe, leave little doubt of it. Just why in certain cases the bacilli will cause lesions, and in others pass through harmlessly, must be ascribed to variations in that rather indefinite and hard-worked term, "power of resistance."

When the bacilli do become arrested in a kidney and begin to colonize, they set up quite characteristic lesions. The commonest form is the disseminated tubercles of acute miliary tuberculosis, an acute general disease usually rapidly fatal, with which we are not concerned.

It is the more chronic form, the renal phthisis or surgical tuberculosis of the kidney, which is of interest to surgeons. The pathological lesions of this disease are fully described in the textbooks, and well known to this audience, and will not be taken up here. In passing, mention should be made, however, of the frequency with which the ureter is secondarily affected, likewise the bladder. Ulceration of the mucous membrane, with thickening and caseation of its walls, convert the ureter, sooner or later, into a shortened, rigid tube, in places greatly dilated, in others with lumen almost occluded. These various changes produce corresponding more or less characteristic variations in the appearance of the mouth of the ureter, which may be retracted, dilated, gaping, "golf-hole," ulcerated, — all of familiar import to the cystoscopist. The neighboring portions of the bladder may show miliary tubercles or ulcerations. In many early cases, and some advanced ones, no changes are found in the ureter or the bladder. Occasionally, the ureter becomes completely occluded, giving rise to the condition known as closed tuberculous pyonephrosis, one of considerable clinical perplexity.

When one kidney only is affected with tuberculosis, changes of a non-tuberculous character in its fellow are not infrequent. In long-standing cases, amyloid degeneration may be found; more often and, in fact, quite commonly, lesions of nephritis occur. These cases of nephritis often clear up after removal of the tuberculous kidney, and are to be ascribed to the toxic effects of the latter.

How often does renal phthisis occur, and how often is it unilateral? For statistics on this and other allied questions, I will quote from that excellent book, Kapsammer's "Nierenchirurgie."

Kapsammer found, out of 20,770 autopsies at the Vienna Allgemeine Krankenhaus, in the decade from 1893-1902, 191 cases of renal tuberculosis, slightly less than 1%; acute and sub-acute miliary tuberculosis was not included. Küster found that 10% of all those dying of tuberculosis had tuberculous lesions of the kidney. According to Krönlein's statistics of surgical diseases of the kidney, tuberculosis takes first place, with 29.8%.

The statistics of operated cases of renal tuberculosis show a considerable preponderance of females over males. According to Israel, 66% are females. On the other hand, Kapsammer's autopsied cases showed 135 men and 56 women, a marked preponderance of men. Kapsammer explains this by saying that up to the present time more women than men with tuberculous kidneys have been operated upon, because in the former the disease is diagnosed earlier, when there is a better chance of operation. More men die of the disease for the opposite reason. He thinks the incidence of the disease is somewhat greater in men.

Kidney tuberculosis is, as a rule, unilateral, at least at first. Krönlein's figures are 92%; Israel's, 88%. Of 62 cases observed clinically by Kapsammer, 48 were unilateral and 14 bilateral. Of the 191 cases found at autopsy, 67 were unilateral. Halle and Motz found, in 131 autopsy cases, 89 unilateral. Of the bilateral cases found at autopsy, many showed an old severe process on one side, and a manifestly more recent and slighter process on the other.

Concerning the question as to which kidney, right or left, is the more often affected, small figures show some variation, but the larger collections of statistics show practically no difference.

An analysis of 2,300 post-mortem examinations at the Massachusetts General Hospital since 1896 show only 18 cases of caseous tuberculosis of the kidney. The tuberculosis was unilateral in 11 cases and bilateral in 7; of the 7 bilateral ones, it was caseous in both kidneys in 3; in the other 4 cases there was a miliary process in the second kidney.

In the 11 unilateral cases the non-tubercular kidney was microscopically normal, except for amyloid disease in 1 case. In only 1 of these cases had nephrectomy been done. In 8 cases there was active tuberculosis outside the genito-urinary tract. In 3 cases the disease was apparently confined to the genito-urinary system. In 1 of these cases the only other focus beside that in the kidney was in the vesicle; in another, a nephrectomized case, in the ureter and bladder; in the third, the sole focus found in the body was in the kidney. This was a remarkable case in many ways and deserves mention in some detail.

Autopsy No. 2089. Feb. 25, 1908.

"Anatomical diagnosis: Extensive phlegmon of left thigh and left anterior abdominal wall.

"Lobar pneumonia of the inferior lobe of the left lung. Focal pneumonia of the inferior lobe of the right lung.

"Serofibrinous pleuritis (left).

"Serofibrinous pericarditis.

"Old cholecystectomy.

"Dilatation of common and hepatic bile ducts.

"Icterus.

"Obsolete tuberculosis of right kidney (renal phthisis with compensatory hypertrophy of left kidney).

"Soft hyperplastic spleen.

"Acute degeneration of the liver and slight chronic interstitial hepatitis.

"Septicemia; streptococcus.

"Left kidney is larger than normal and weighs 286 gm.; measures 15.5 cm. in greatest dimension. Capsule

strips easily. Cortex wider than normal, 7 mm.; in narrowest portion markings retained. Renal pelvis and ureter normal. The right kidney is much smaller than normal, weighing 42 gm., and measuring 7.5 cm. in length. The capsule strips with difficulty, leaving a very irregular granular reddish surface. At either pole there is an ill-defined yellowish coloration.

"On section in the situation of the pyramids there are cavities of more or less spherical form, varying in diameter up to about 2 cm. Some of these cavities are filled with a white, opaque semifluid material of about the consistence and appearance of cream cheese. They are lined with a yellowish rather opaque membrane about 1 mm. in thickness, but not sharply defined from the surrounding tissue. The remaining portion of the cortical substance of the kidney about these cavities shows little or none of the renal markings and is of a reddish color. In places the cortical substance together with the lining membrane of the cavities forms a layer less than 2 mm. in thickness. The right ureter is smaller in diameter than normal and its lumen is partly obliterated and admits a small probe with difficulty. The opening of the right ureter into the bladder is much smaller than the opening of the left ureter. The bladder is not remarkable except for some injection of the veins of the mucosa. Vagina, uterus and oviducts not remarkable, ovaries small and fibrous, right ovary contains a small cyst.

"Colonies of streptococci from heart culture. Smear preparation of the cheesy material from the right kidney fails to show tubercle bacilli.

"Microscopical examination of kidney: Extensive atrophic changes are present. The caseous material is walled off by dense connective tissue. No epithelial cells are present. No tubercles observed. The process is apparently obsolete."

A brief review of the clinical history of the case is as follows:

The patient, who was a married woman, fifty-one years of age at the time of her death, entered the Massachusetts General Hospital in January, 1894. One brother had died of phthisis. She had two healthy children. Her previous health had been good except for rheumatic fever six years ago. She had suffered from varicose veins for many years. For two years she had had an abscess of the thigh, from which bone had been discharged. This was incised and the bone curetted and she was discharged from the hospital with the wound healed. The examination of abdomen and chest was at this time negative. Urinary examination showed trace of albumen with considerable sediment, consisting of pus, squamous cells, medium sized round cells, blood and calyceic oxalate crystals. No record was made of urinary symptoms.

Nov. 2, 1907, she re-entered the hospital with acute cholecystitis. There was history of gallstone attacks during the last two years. Previous to this attack she had been in good health, weighing 185 lb. At this time it is noted that the right kidney is not felt, the left is palpable but not tender. The patient was jaundiced, with tenderness in the region of the gall bladder. She had a cholecystectomy done, and a stone in the common duct crushed and pushed on. At this time the urine showed a trace of albumen with leucocytes, squamous cells and a few hyaline and epithelial casts. There were no urinary symptoms. The patient was discharged to the Convalescent Home with a small sinus, but re-entered on the Medical Service, on Jan. 2, 1908, with sore throat, hoarseness, cough and fever. She was still jaundiced and this deepened. The patient gradually failed, pneumonia developed and she died on Feb. 24.

This is evidently one of those rare cases of nature's cure of tuberculosis of the kidney by complete destruction of the organ. This is probably what takes place in those cases reported as clinically cured or arrested. I have yet to see the report of an arrest or true cure of renal tuberculosis which will withstand searching inquiry. As far as I am aware, there is no pathological evidence of a healed focus of tuberculosis in the kidney, such as is so often found in the lungs and other organs. By this is meant a local focus of obsolete tuberculosis which can be recognized as such, by caseation or calcification, without either destruction of the entire organ or an accompanying active process in some other part of the same organ. In other words, pathological evidence of spontaneous healing of renal tuberculosis is lacking. Accurate and convincing clinical evidence is likewise lacking; and until this much-to-be-desired evidence is forthcoming, I believe we must look upon renal tuberculosis as a progressively destructive process.

REMARKS ON THE PROGNOSIS AND TREATMENT OF RENAL TUBERCULOSIS.*

BY RICHARD F. O'NEIL, M.D., BOSTON.

THE prognosis and treatment of renal tuberculosis have been more or less modified the last few years because of the now very generally conceded fact that genito-urinary tuberculosis is most often primary in the kidney and is often unilateral. The prognosis, therefore, depends considerably upon the promptness with which the diagnosis is made, as the process rarely begins simultaneously in both kidneys, except in general miliary tuberculosis, and the accuracy with which the indications for operation are worked out. This applies particularly to the condition of the opposite kidney.

The study of some of the recently reported series of nephrectomies by various operators is of interest in this connection. Rafin¹ reports 63 primary nephrectomies for tuberculosis with 7 deaths from operation, 11%. In 5 out of these 7 fatal cases, lesions were found in the opposite kidney. In 1 case there was anuria, and death in twelve days, the remaining kidney being found to have been entirely destroyed by caseation. The ureter catheter gave reliable results in 3 of these cases, misleading in 1, and was not used in 3, because of intravesical lesions making it impossible. His view is never to operate without knowing the state of the other kidney, which should be ascertained early, before bladder change makes it difficult, and to operate while the patient's condition is good. The importance of studying the other kidney is further shown by consulting a total of 316 nephrectomies, with 35 deaths, or 11%, by Isreal,² Nicolich, Zuckerkandl, Wildbolz, Caspar and Rafin. Many of these deaths were due to insufficiency of the other kidney, whose activity had not been tested by catheterization or any of the functional

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