CHAPTER XLI

TUBERCULOSIS OF THE KIDNEY

Etiology

Tubercular inflammation of the kidney occurs with tuberculosis of the genital tract or of the lungs. It also occurs as a primary lesion. The theory that the primary tubercular deposit may occur in the kidney was first developed by Vigneron.[1] It has been confirmed by but few autopsies. Whatever the original seat of the disease, death does not often occur until the tubercular inflammation has spread to several organs. But the strongest support of the theory of primary renal tuberculosis lies in the results of nephrectomy. The remarkably large percentage of cases that remain free from evidences of the disease for a number of years after this operation is sufficient evidence that in these cases, at least, the primary inflammation is in the kidney (p. 610).

Renal tuberculosis is a disease of youth. Its more acute forms occur during adolescence, while its chronic caseating forms are usually encountered between the ages of twenty and forty. Thus Israel[2] states that 80% of his operations were performed upon persons between these ages. Exceptionally the tubercular kidney is encountered in later life.

French and German authors are agreed that women suffer from renal tuberculosis twice as often as do men, though the smaller statistics of English and American authors (Morris,[3] Tilden Brown,[4] Watson[5]) show a preponderance in the opposite direction.

Pathogenesis.—The tubercle bacillus may reach the kidney by direct extension of a tubercular process from an adjoining organ, or by invasion from the lymphatics, or from the blood current.

[1] Thèse de Paris, 1892.
[2] Chir Klin. d. Nierenkrankh , Berlin, 1901, p. 180.
[3] *Op. cit.*, p. 484.
[4] N. Y. Med. J., 1897, lxv, 377, 447, 479.
[5] Boston Med. and Surg. J., 1895, xxxii, 121, 135.

The extension of a tubercular inflammation to the kidney through its capsule is most exceptional. Tilden Brown justly remarks that tuberculosis of the suprarenal capsule does not involve the kidney, and I have known a kidney to remain normal, although it lay for several months surrounded by a tubercular perinephritic abscess.

The constant association post mortem of vesical, ureteral, and renal tuberculosis long misled pathologists to believe that the kidney is always attacked by a tuberculosis mounting the ureter from a primary lesion in the bladder. Hence extension of the disease along this route is still generally regarded as an established fact, although we are learning to recognise that this method of invasion is by no means so frequent as has been believed. Yet the documentary evidence in favour of this view is singularly slight. It is known that the disease travels down the ureter from the kidney, and that extirpation of the kidney is often succeeded by a cure of the ureteral and vesical inflammation; but, so far as I know, there is no recorded case of a vesical tuberculosis extending up the ureter without reaching the kidney. The discovery of tubercular lesions in the kidney, ureter, and bladder may be explained in several ways. The infection may work its way up along the ureteral mucous membrane; it may proceed by a lymphatic infection along the ureteral wall; or it may reach the kidney from the circulation, and thence pass down the ureter to the bladder. This last explanation seems the most plausible, and, according to it, every renal tuberculosis, whether primary or secondary, is hematogenous in origin. Such a theory has at least the merit of explaining the occurrence of tuberculosis in the kidney and genitalia—a not uncommon phenomenon—without any lesion whatever of the bladder.

Infection from the blood stream is the usual source of primary renal tuberculosis. Why the kidney should be able sometimes to transmit tubercle bacilli and at other times fall victim to them, why so many cases show involvement of one kidney years before that of its fellow—in other words, what the predisposing causes of renal tuberculosis may be, the future must decide. Our ideas upon this subject are not yet sufficiently clear to merit debate here.

Morbid Anatomy

Primary Tuberculosis.—There are four varieties of primary renal tuberculosis—viz.:

1. Acute miliary tuberculosis.
2. Subacute diffuse tuberculosis.
3. Chronic papillary ulceration.
4. Chronic caseous tuberculosis.

1. Acute miliary tuberculosis of the kidney is a phase of acute general miliary tuberculosis interesting only to the pathologist.

2. Subacute diffuse renal tuberculosis holds a middle place in point of virulence between acute miliary tuberculosis and chronic caseation. It usually attacks both kidneys simultaneously and is habitually associated with tubercular lesions in other parts of the body. The kidneys are riddled with tubercles, which are found chiefly in the cortex, in the glomeruli, and along the vessels. As they break down they form caseous and purulent foci radiating from the pelvis, itself inflamed, simulating the purulent collections seen post mortem in the acuter forms of surgical kidney—a condition for which this variety of tuberculosis is often mistaken.

The course of the disease under this form is rapid and fatal. It occurs in children, and only interests the surgeon from the fact that it is likely to be mistaken for chronic renal tuberculosis. If so diagnosed and operated upon the surgical intervention only hastens the fatal issue.

3. Tuberculous ulceration of a papilla is a very rare form of primary tuberculosis. A tubercular deposit occurs in the tip of one or more of the papillæ, ulcerates, and gives rise to considerable hemorrhage with but few other symptoms. Such an ulcer appears to have little tendency towards dissemination. Hurry Fenwick and Israel have operated successfully upon such cases.

4. Caseous renal tuberculosis is the *surgical* tuberculosis of the kidney. The tubercular process usually begins in one or other extremity of a single kidney. The usual stages of tuberculization, ulceration, and caseation occur, so that, as seen by the surgeon, the disease presents itself under the form of multiple tubercular nodules and caseous areas. The kidney may be affected in whole or in part. Section reveals a number of tubercular nodules in various stages of development, some solid, others caseous. As the inflammation progresses new tubercular deposits appear, and the old nodules break down and coalesce. Secondary pyogenic infection occurs, and finally the kidney becomes more or less of a shell surrounding one or more large caseous foci (Fig. 149). The pelvis and ureter become thickened and studded with tubercles. In extreme cases the ureter may be obliterated and the dilated pelvis filled with the solid caseous mass.

Secondary Tuberculosis.— Secondary or ascending renal tuberculosis may be either miliary or caseous. Exceptionally a hydronephrotic kidney becomes tubercular, so that, while it retains the macroscopic appearance of an aseptic hydronephrosis, microscopic examination reveals tubercular lesions in it and tubercle bacilli in its fluid contents.

Secondary Pyogenic Changes.—The tubercular kidney, like the tubercular lung, is liable to secondary infection by pyogenic micro-organisms. Such a double infection adds to the virulence of the case. Mixed infection in subacute, diffuse tuberculosis hastens

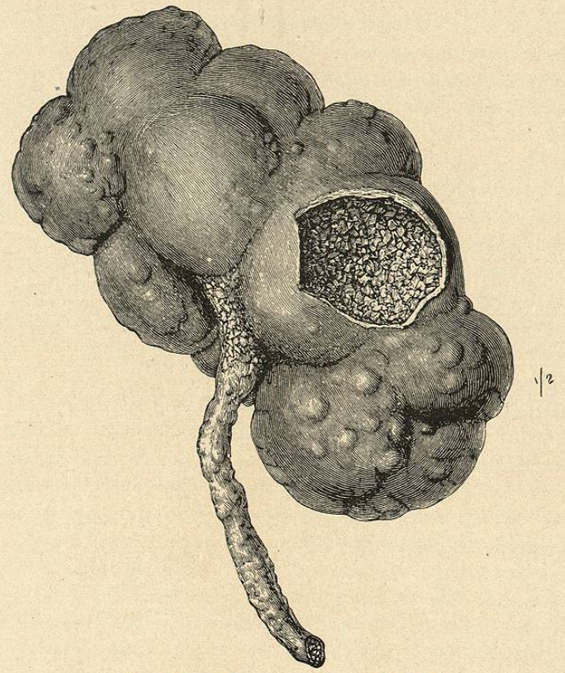

Fig. 149.—Tubercular Kidney (Morris).

the fatal issue; mixed infection in caseous tuberculosis causes tubercular pyelonephritis. The sacculated kidney suppurates throughout, and the tubercular and pyogenic inflammations spread rapidly to the bladder and soon affect the opposite kidney. In fact, nothing stimulates the progress of a chronic caseating tuberculosis so much as secondary infection by pyogenic microbes.

Accessory Lesions.—Fatty and fibrous *perinephritis* results from chronic renal tuberculosis as well as from chronic suppuration (p. 540). Perinephritic phlegmon and abscess are rare, and are usually due to mixed infection. *The ureter* is involved by the extension of the tubercular inflammation downward (or upward); its walls are much thickened and ulcerated, its lumen is pouched and usually con-

stricted, sometimes entirely obstructed. As the tubercular inflammation extends downward it reaches the *bladder* and extends to *that portion of the vesical wall surrounding the orifice of the ureter.* Even before the tubercular process has reached thus far some congestion or simple inflammation of this region often occurs as the result of mixed infection in the kidney. The presence of this congestion or tubercular inflammation in the bladder around the orifice of the ureter is of great importance. If tuberculosis of the kidney is suspected and cystoscopy reveals congestion, tuberculization, or ulceration about the orifice of one ureter, it may be concluded that the kidney to which that ureter leads is certainly tubercular, although it is by no means fair to infer that the opposite kidney is free from involvement. Israel estimates the frequency of this descending bladder tuberculosis at 59%.

The Opposite Kidney.—From a prognostic point of view the condition of the opposite kidney is of the greatest importance. Statistics derived from the dead-house are of no value here, for what the surgeon must know is not whether the second kidney was involved at the time of the patient's death, but whether the second kidney is involved at the time the patient presents himself for treatment. Vigneron [1] collected the records of 205 cases subjected to operation, of which only 106 were known to have bilateral renal tuberculosis. Israel records 21 cases of primary renal tuberculosis operated upon, of which only 2 (9.5%) were bilateral. The statistics of all other surgeons agree on this capital point: that *in chronic renal tuberculosis the two organs are not inflamed simultaneously; the second kidney becomes diseased many months after the first;* and, in about one half the cases, the disease is unilateral at the time the patient presents himself for treatment.

It is not to be forgotten, however, that amyloid degeneration and chronic inflammation may attack the opposite kidney even though it be not tubercular. This fact has a particular bearing upon the post-operative prognosis of nephrectomy for renal tuberculosis. Thus 3 of Israel's 9 deaths are attributed to this condition.

Symptoms

Tubercle of the kidney, like stone in the kidney, may run its whole course and lead to the patient's death without ever giving any recognisable symptom referable to the urinary organs. Yet this is rare. Usually there are symptoms, but these symptoms are most misleading. The ordinary evidences of renal surgical disease, such

[1] *Op. cit.*

as pain and tumour in the loin, are inconspicuous in all but the most advanced stages of the disease. The characteristic early symptoms are painful and frequent urination and hematuria, a symptom-complex which directs attention to the bladder, where the discovery of a tuberculous ulceration may well lead the surgeon to overlook the primary focus of the disease in the kidney.

Painful and Frequent Urination.—The first symptom of tuberculosis of the kidney usually is a frequency of urination. This is the case whether there is any tuberculosis of the bladder or not. This frequency is spontaneous, and may be quite inexplicable if the urine contains no pus or blood. There is usually some polyuria even in the early stages. After a time urination becomes painful. The pain occurs during the urinary act, or at its termination if there is ulceration about the bladder neck. Needless to relate, pain and frequency are uninfluenced by any local treatment of the bladder. They continue, varying in intensity, and even disappear at times. As a rule they are not extreme unless the bladder is tubercular.

Hematuria.—Sometimes the first evidence of renal tuberculosis is hematuria. The bleeding is not particularly characteristic. It has not the severity of a hemorrhage from a neoplasm, nor is it so continuous or so constantly associated with pain as that from stone. The primary bleeding is likely to last for a short time, to be followed by a protracted period during which there is little or no blood in the urine. Although most authorities incline to the belief that hematuria is a fairly constant sign of tuberculosis of the kidney, Israel only noted it 4 times among 18 primary cases, and I believe that its frequency is generally overestimated. In the later stages of the disease, if there is mixed infection the purulent urine is very likely to show some traces of blood. Violent hemorrhage rarely occurs, though the bleeding from an ulcerated papilla may be so severe as to endanger life.

Renal Pain and Tumour.—There is usually a dull soreness in the loin, but little active pain. Renal colic from the passage of clots of blood or shreds of caseous matter is exceptional. The kidney enlarges sufficiently to form a notable tumour only when such a complication as pyelonephritis or perinephritis occurs. The tuberculous ureter may sometimes be felt by rectal or vaginal palpation or through a thin abdominal wall. A tubercular thickening in the ureter has been mistaken for stone.

Urinary Signs.—The urine usually is of low specific gravity and from the beginning somewhat increased in quantity, and this polyuria accounts in some degree for the frequency of urination. Hematuria may occur, and pyuria appears with the occurrence of ulcera-

tion and mixed infection, whether in the kidney, in the bladder, or in both. The purulent urine of the later stages of renal tuberculosis is acid, always acid; it is albuminous, perhaps tinged with blood, and contains casts of various qualities.

General Characteristics.—The patient presents the usual tubercular characteristics: a history of tuberculosis in other members of the family and evidence of the disease elsewhere in his body, either in the lungs, the epididymis, the bladder, or the cervical lymphatics. He is usually under forty, and is pale, thin, and lymphatic in appearance. Israel states that there was the usual tubercular evening rise of temperature in only 22% of his cases of uncomplicated renal tuberculosis, while 80% of those patients whose bladders were involved had fever. The loss of weight so characteristic of tubercular disease is usually noted, and it has been my experience that the pulse is habitually rapid.

Diagnosis

It is the commonly accepted belief that diagnosis of tubercular kidney is extremely difficult. In many senses this is true. It is difficult even for an expert to diagnose a tubercular kidney in its earliest stages; it may be impossible for him to distinguish between chronic caseous renal tuberculosis and subacute diffuse tuberculosis. It is often quite impossible for the practitioner who is unaccustomed to use the cystoscope and the ureteral catheter to diagnose the disease in any but its most advanced stages, and it requires the greatest diagnostic acumen to decide in many cases whether or not the disease is of such a nature as to merit treatment by hygienic and climatic agencies or by the knife.

In order to simplify the matter as much as possible we may first enumerate the forms of the disease that can be readily distinguished. In its advanced stages the disease can almost always be recognised either by examination of the urine or by palpation of the loin.

The gross urinary aspects often suggest the existence of an inflammation of the kidney or of the bladder. The urine may be purulent; it may contain blood, albumin, and casts. It is habitually acid. If the bladder is involved the signs of bladder tuberculosis may be detected (p. 401), and *if the bladder is found to be tubercular it is the rule always to suspect a similar condition in the kidneys.* If, however, the bladder is not inflamed, the only urinary sign to distinguish a tubercular pyelo-nephritis from a suppurating one may be the presence in the urine of the tubercle bacillus. Unhappily, in a large proportion of cases, it is quite impossible to discover this bacterium in the urine (p. 403). Thus it was identified

microscopically in only 15 of the 67 cases reported by Küster, König, and Czerny. It is in such cases that injection of the suspected urine into guinea-pigs affords a positive and conclusive physiological test. Morris relates a convincing case in which tubercle bacilli could not be distinguished by a most careful examination until, after the success of guinea-pig inoculation, a second microscopical investigation disclosed the incriminated microbe. The tubercle bacillus should be thus sought by microscopic and physiological tests in every case of pyelo-nephritis of obscure origin.

The presence of a tumour or a markedly tender point in the loin may be the first thing to draw attention to the kidney. Here again obscure cases merit a searching investigation for Koch's microbe.

In other cases an unexplained dysuria is the most marked symptom. The urine may be clear and disclose no evidences of the renal condition beyond an insignificant polyuria, with perhaps a slight albuminuria. The tubercle bacillus may not be discovered in the urine and the cystoscope may not reveal the pathognomonic inflammation about the ureteral orifice. Such cases represent the earlier stages of the disease in which it may be impossible to make an absolute diagnosis. The patient's history and physical type, the presence of lesions elsewhere in the body, an unexplained loss of flesh, an afternoon temperature—all these are suggestive elements to the experienced diagnostician. But any or all of them may be lacking, so that the diagnosis cannot be made until the disease has reached a more advanced stage. And even if the presence of tuberculosis in the kidney is suspected, one cannot feel sure of it unless (1) tubercle bacilli are disclosed by the microscope or the guinea-pig, or (2) unless the cystoscope shows a tubercular process about the mouth of the ureter.[1]

Having diagnosed the presence of renal tuberculosis, the next point is to discover the condition of the two kidneys. Is the disease bilateral, or, if it be unilateral, is the opposite kidney present and functionating properly? This is the finest point of diagnosis, it has no interest for those who are committed to a purely medical treatment of the disease. But if there is question of nephrectomy it is absolutely essential to know which kidney is tubercular and what is the condition of its fellow. These points can be determined only by examining the urine obtained separately from each kidney; which is to say that the urine segregator or the ureteral catheter

[1] I formerly employed tuberculin injections for the diagnosis of these cases, but gave them up because I found them to contain an element of danger as well as an element of uncertainty.

must be employed.[1] Of the two, I rather prefer the ureteral catheter for most cases. This is especially true if there is any tubercular lesion of the bladder, for in such cases contact of the segregator with the ulcerated bladder wall is likely to provoke a hemorrhage sufficiently profuse to nullify the result of the examination. It is scarcely necessary to say that the insertion of a ureteral catheter into a ureter whose mouth is tuberculous is quite uncalled for. The duct undoubtedly leads to a tubercular kidney. It is the opposite and apparently healthy kidney that requires catheterization.

The examination of the urine obtained by the ureteral catheter should be both bacteriological and chemical. The pathologist should seek for tubercle bacilli, for the evidences of surgical inflammation, and for evidence of the renal function. The surgeon is not justified in assuming that this kidney is in a satisfactory condition unless the urine obtained from it shows (1) no tubercle bacilli, (2) no pus, (3) no more than a trace of albumin and a few casts, and no marked decrease in the excreted solids. It is worthy of note that a very considerable proportion of those persons who die at a greater or less length of time after a successful nephrectomy for tuberculosis lose their lives on account of amyloid degeneration or chronic nephritis of the opposite kidney. Another point that must be determined is the presence of tuberculosis elsewhere in the body. I need only enumerate the lungs, the testicles, the vesicles, and the prostate as probable seats of tubercular deposit.

The nature of the lesion in the kidney can be determined only by a just appreciation of all these data. The surgeon is not justified in assuming that he has to deal with chronic caseous tuberculosis (the only form of the disease for which a radical surgical cure can be expected) unless he knows that the tubercular lesion is confined to one kidney; and he cannot justly expect to achieve such a radical cure unless he knows that the disease exists only in a mild or latent condition elsewhere in the body. Thus tuberculosis of the ureter and of the adjoining portion of the bladder is no contra-indication to nephrectomy, nor is a slight lesion of the lung. But the presence of active pulmonary or genital tuberculosis will usually nullify the effect of the most successful nephrectomy.

Prognosis

The prognosis of tuberculosis of the kidney depends upon the nature of the lesion, the age of the patient, and the presence of le-

[1] If the case is known to be tubercular, the cystoscope or the segregator should never be employed academically for the mere purpose of distinguishing the extent of the bladder disease, but only as a preliminary to nephrectomy.

sions elsewhere in the body. The prognosis of the acute and subacute forms of renal tuberculosis is extremely bad. Usually the patient rapidly loses ground, the disease spreads from one organ to another, and runs its course within a year or two. On the other hand, the caseous form of tuberculosis, if not complicated by a secondary mixed infection and if associated with few lesions elsewhere in the body, is very slow to progress and may last for many years. Apparently the course of the papillary tubercular ulcer is equally chronic. The age of the patient has an indirect bearing upon the prognosis, for tuberculosis of the kidneys, like that of other organs, assumes its more malignant forms in the young and advances more slowly in later years. Finally, the distribution of the disease among the other organs of the body has a marked influence. For, even though the renal lesion be caseating and chronic, the patient may die of some more acute process in the lung or in some other portion of the genito-urinary tract. Tuberculosis of the kidney may progress to a spontaneous cure. Such an event is most unusual, and yet it may occur. Moreover, appropriate hygienic treatment will in many cases delay the fatal issue for years, and sometimes effect a cure.

Treatment

We are now on the crest of a wave of operative success in the treatment of renal tuberculosis. We look backward into the depths of the pre-operative period and appreciate—fairly, I think—that a great advance has been made; but looking forward there is another depth which can only be dimly outlined. A cursory review of current therapeutic reports would lead one to suppose that the treatment of renal tuberculosis is purely surgical. A few isolated voices are raised in protest; but it is impossible to deny the great success of modern surgery in eradicating tuberculosis of the kidney, and in our first flush of appreciation of this success it is quite impossible sanely to appreciate how much evil there may be intermingled with the good. Perusal of the works of Tuffier, Israel, Simon, Küster, and Morris is calculated to enforce the conviction that surgery is the ideal treatment—the only treatment deserving of the name. Yet I believe that the future will modify this view. I believe we shall learn that the operative successes are not so permanent as the figures now before us would seem to indicate, and that, lasting as they may be, in the majority of cases hygienic and tonic treatment will prove more effective still. In short, tuberculosis of the kidney, like tuberculosis of any other organ, is not a local disease. I doubt if the surgeon is ever able to diagnosticate tuberculosis of the kidney at a time when there are no other tubercular lesions in the body. Cer-