

THE PATHOLOGY OF RENAL TUBERCULOSIS

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Portal of Entry and Primary Focus

Renal tuberculosis is usually a blood borne infection, although an ascending infection from tuberculosis of the bladder or genitalia by way of the peri-ureteral lymphatics cannot be denied. Direct infection by sexual connection or by dirty instruments (e.g. catheters) is sufficiently rare to be a bacteriological curiosity. The primary focus must therefore be in one or the other of the two common portals of entry, the respiratory or the intestinal tract. This is not a mere academic point of interest, for there may still be activity in this focus or in one of the satellite lesions connected with it by lymphatic drainage, and to concentrate entirely on the renal lesion is to acquire a wrong perspective of the disease. Primary foci in the lung are usually readily detected, but those in the intestinal tract are often clinically silent and inaccessible to X-ray examination. With the working hypothesis of renal tuberculosis as a blood borne infection from a primary focus, the cases may be divided into three groups:

- (1) Those in which the primary focus or its satellite lesions are still active (generally instances of chronic pulmonary tuberculosis);
- (2) Those in which other blood borne infections are present (e.g. bone and joint tuberculosis) with or without an active primary focus;
- (3) Those in which the primary focus has become arrested and calcified and in which there are no active lesions of tuberculosis other than the renal one.

The first group of cases find their way to the medical wards and are essentially the physician's province, for they are part of a general picture of chronic disseminated tuberculosis, usually with a bad prognosis. The question is sometimes asked, Can the phthisical patient with intact kidneys excrete tubercle bacilli in the urine? This is an old question which has never been satisfactorily answered. Whilst it is impossible to deny that it can occur, it is probable that the presence of tubercle bacilli in the urine usually implies a renal lesion, though it may be small and may even occasionally heal.

Anatomical Structure

Certain anatomical features of the kidney are important in studying its pathology.

(1) The first is the *division of the organ into lobes*. The interlobar arteries do not anastomose freely and are in the nature of end-arteries. Running along each side of the pyramids, they divide dichotomously at the junction of the cortex and medulla into branches—the arciform or arcuate arteries—which do not anastomose with similar vessels of neighbouring lobes. This vascular independence tends to limit the infection.

(2) The *arteriole to the glomerulus* takes part in the formation of two sets of capillaries where the tubercle bacillus may get held up, the vas afferens breaking up into one set, then the vas efferens after leaving the glomerulus dividing up into another to supply the convoluted tubule of the cortex. The circular muscle fibres in this efferent arteriole which are important in regulating the pressure in the glomerulus, may indirectly determine the hold up of bacilli.

(3) *Terminal portions of the renal arteries* anastomose in a capillary network in the periphery of the cortex with branches of the lumbar, phrenic and suprarenal arteries whence the organ may receive tubercle bacilli. Miliary tubercles are frequently found immediately beneath the capsule.

(4) According to Maximow and Bloom the *glomerular lining* has both a visceral and a parietal layer of epithelial cells. Those of the former are adherent in a continuous layer to the capillary loops. Those of the latter are of a simple squamous type resting on a basement membrane; they become cuboidal as they merge into the epithelium of the proximal convoluted tubercle. These epithelial layers act as bacterial barriers.

(5) *The uriniferous tubules* have two convoluted portions, a proximal and a distal, in either of which tubercle bacilli may be held up. They may also be held up at the calyces.

(6) *The venous return* in the kidney begins with the stellate veins of the cortex and the blood passes by way of interlobular to the interlobar and then to the renal vein.

(7) There are two sets of *lymphatics*, a subcapsular and a peri-tubular. Collecting vessels follow the renal veins and end in the lateral aortic lymph glands. Sometimes these collecting trunks join up with those in the upper part of the ureter before reaching the glands, a point which may be important in the passage of tubercle bacilli from kidney to ureter and ureter to kidney. There are therefore a number of anatomical nodal points in the capillaries, lymphatics and uriniferous ducts, where tubercle bacilli may lodge, but the permanent resting place of the bacilli is probably the lymphatic system of the organ, to which point they are carried by phagocytes.

Morbid Anatomy

The naked eye pictures of renal tuberculosis are many and various, and may be classified into four groups.

(2) A direct invasion picture in which the disease has spread from neighbouring infected parts, such as tuberculous Fallopian tubes, vertebrae, or peritoneum—a tuberculous perinephritis in fact. This form was well described by Osler many years ago, and is probably less common than it used to be.

(2) The miliary and submiliary types. The tubercles resemble those of miliary tubercles elsewhere, at first discrete, grey and opalescent, later coalescing into conglomerate tubercles, attaining the size of small peas, and becoming opaque and yellow.

(3) A toxic nephritis of tuberculous origin with cloudy swelling and albuminuria, as described by Wakeley and Hunter.

(4) The so-called surgical variety, sometimes also referred to as renal phthisis, a term which is more expressive than pathologically accurate.

The miliary and toxic varieties are by their nature bilateral. Tuberculous perinephritis is usually unilateral, so also is the surgical variety, at least in its earlier stages.

This last named variety should be regarded as the true tuberculous kidney. It is analogous to other blood borne lesions of tuberculosis such as those of bones and joints or genitalia, and like them is accessible to successful surgical treatment. The disease assumes many forms, e.g.:—

(a) small yellowish ulcerating granulations at the apices of the pyramids with thickened lymphatics leading from them;

(b) granulations and ulcerations of the pelvis and calyces forming a tuberculous pyelonephritis;

(c) tuberculous hydronephrosis and pyonephrosis;

(d) rounded caseating nodules like small marbles, single or multiple, embedded in more or less healthy looking kidney tissue, and situated either at the poles of the organs or between cortex and medulla, or less frequently in the pyramids or occurring as small bosses beneath the capsule;

(e) breaking down of these rounded nodules, with tuberculous pus formation leading to pyonephrosis which ultimately becomes nothing less than a bag of pus contained within a thickened capsule;

(f) rupture through the capsule to form a perinephritic abscess.

Sometimes these lesions are classified as open or closed renal tuberculosis, according to the patency or non-patency of the ureter, and once this duct is reached serious consequences follow. Ureteric infection may take the form of (i) an infiltration of the coats of the ureter by way of the lymphatic plexuses, or (ii) a blockage of the fairway of the duct with tuberculous detritus. The former is liable to lead to a uniform thickening of the duct—the “pipe stem” type of tuberculous ureter—the latter to localised thickenings with saccular dilatations.

Histology

The microscopical appearances of the renal tuberculous lesion do not differ significantly from the type of lesion elsewhere, viz. a collection of reticulo-endothelial cells, phagocytosis, and giant cell formation—what is universally known as the giant cell system. Beyond this again are zones of round-celled infiltration and thin collagenous fibres. This picture constitutes the

unit lesion of tuberculosis and extension and coalescence of these minute lesions usually follow, resulting in caseation. Calcification is uncommon, but small areas of fibrous tissue the size and shape of the giant cell systems occasionally occur, suggesting that the miliary tubercles of the kidney may heal as they do in the lung; but this is a very exceptional phenomenon, and proof of it is by no means complete.

Clinical Pathology

The essential point is of course the detection of the tubercle bacillus. For this purpose the demonstration of an acid fast bacillus in the urine is not enough, since there are other acid fast organisms living as harmless saprophytes in the urethra and external genitals. It is customary to speak of these by the loose term *B. Smegmatis*, but there are probably numbers of these harmless acid fast saprophytes, and they are found in the female as well as in the male. Some are morphologically quite unlike the tubercle bacillus, others indistinguishable, and they vary also in the degree of their acid and alcohol fastness, especially in the latter. Tubercle bacilli will retain the carbol fuchsin after exposure to alcohol longer than most of these saprophytic bacilli will do. These saprophytic acid-fasts do not grow readily on culture media at 37° C. and it is not unlikely that some of them are anaerobic. Furthermore, they are non-pathogenic to guinea pigs. The tubercle bacillus is usually accompanied by pus cells in the deposit, and acid fast bacilli which are not should be viewed with suspicion. A catheter specimen will generally settle the point. Examination of the 24 hours' output is sometimes resorted to. This is helpful in cases where the ordinary single specimen has been found to contain pus cells without any organisms, but the method has most of the disadvantages of the single specimen. The whole of the 24 hours' output is allowed to stand in a tall cylinder overnight and the sediment centrifuged at 3,000 revolutions for half an hour, after which the centrifuged deposit is examined in the usual way. Cultivation of these deposits was popularised some years ago by the introduction of the Lowenstein technique which consists in planting the centrifuged deposit on a special egg medium. Doubts have been cast upon some of the positive results obtained and the risk of mistakes where a major surgical operation is pending are serious. Guinea pig inoculation still remains the final arbiter. If possible not less than 100 c.c. urine should be centrifuged for this purpose, and if it is heavily infected with secondary organisms the deposit should be treated with N/10 NaOH for half an hour followed by neutralisation with N/10 hydrochloric acid before injection, two animals being inoculated in order to guard against failure through the premature death of one animal. Examination of the segregated urine from ureteric catheterisation often presents a difficulty. The urine collected is generally small in amount and the pathologist is faced with a choice between film examination or culture or animal inoculation of the centrifugal deposit, since there may not be enough for all three tests. Usually the urgency of the clinical situation determines the choice, because the surgeon is rarely prepared to wait for cultures or biological tests. The nature of the type of tubercle bacillus isolated is not important from the clinical angle. Bovine strains are probably responsible for about one-sixth of the cases. At one time it was thought that bovine strains were less virulent for man than the human, but Stanley Griffiths's opinion at the end of a life-time's research on this subject was that this was not so. There is, however, some evidence that strains of low virulence do occasionally occur, as they do in lupus. There has been a suggestion too that avian strains may be very rarely responsible, but as far as the writer is aware none of these questions has been explored in this country. At all events the marked chronicity of some of the cases is striking, and might well repay further bacteriological research.

Secondary infection is generally due to *B. coli* or the streptococcus. Lardaceous disease can generally be diagnosed by the association of a high protein content with polyuria. The congo red blood test is in the writer's experience valueless and not free from risk.



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