THE RÔLE OF THE CORTICAL LYMPHATICS IN THE EXTENSION OF TUBERCULOSIS WITHIN THE KIDNEY

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Clinical experience has established the futility of partial nephrectomy as a surgical measure in cases in which the lesion of the kidney is tuberculous. The purpose of this communication is to contribute further pathologic evidence as to the paths of extension of tuberculosis within the kidney, in justification of operations now employed, rather than to discuss what is already established as a sound surgical procedure.

The observations here recorded were made on a group of twenty-one kidneys, obtained at operation, which showed macroscopically a single small lesion at some point in the renal substance with an attendant tuberculous pyelitis. In some instances, the cortex was almost, if not entirely, free from gross lesions, yet in most instances there were a few small cortical tubercles to be seen overlying the gross lesion of the interior of the kidney and connected with it by lines of tuberculous infiltration. There was in each instance a considerable portion of the kidney cortex which seemed grossly to be normal. To these apparently uninvolved portions of the cortex my study was directed with the purpose of demonstrating new lesions, if any were present, which might be concerned in the subsequent destruction of the kidney. These early cases are not common. The twenty-one kidneys studied have been collected from the surgical services at the Massachusetts General Hospital and from private practice.

The studies of Ekehorn¹ have thrown much light on the manner in which a tuberculous focus in the kidney spreads in the pelvis of that kidney. It is generally recognized that a primary tuberculous focus in the kidney is almost invariably situated in the region of the medullary portion of the kidney cortex at the base of the pyramid in the region of the loops of Henle. That these may exist for a time without symptoms seems to be a clinical fact. Eventually the process extends in all directions and breaks by the shortest path spontaneously into the pelvis which is nearer than the cortical surface. After the tuberculous process has ruptured into the pelvis, a tuberculous pyelitis with its attendant symptoms soon develops. Ekehorn

1. Ekehorn: Die Ausbreitungsweise der Nierentuberkulose in der tuberkulösen Niere, *Folia Urolog.* 2:412, 1908.

has demonstrated by serial sections of all the pyramids of such a kidney, that all the pyramids rapidly become infected. Tubercle bacilli are found in greatest numbers in the recesses of calices where urinary drainage is probably less complete, and the pyramids soon become involved in these regions. From these lesions, he believes ascending infection takes place within the kidney along the lumens of partially obstructed tubules and by way of the intertubular lymphatics and perhaps by the blood vessels.

The presence of tuberculous pyelitis is, in itself, sufficient evidence to justify the complete removal of a tuberculous kidney no matter

Fig. 1.—Microscopic section, $\times 50$, from the apparently normal portions of a kidney in the opposite pole of which there is a small active tuberculous focus. Note that the tubercle apparently takes origin from the interstitial tissue and not from glomeruli. Aside from the row of cortical tubercles, two of which are shown here, the tissue of this pole of the kidneys shows no tuberculous changes. There is tuberculous of the pelvis throughout all its parts. (Photomicrograph by L. S. Brown.)

how small the amount of renal tissue destruction which may have taken place. There is, however, a small group of cases in which there is a double kidney with separate pelves, only one of which is infected; another group in which there are fused kidneys, and more rarely a group in which there are normally developed kidneys in which a small chronic lesion of one pole has resulted in the formation of a sclero-

caseous partition which separates that portion of the pelvis from the remainder. In these cases the temptation to perform a partial nephrectomy is great. I have one double kidney in my series of twenty-one, only one pelvis of which shows a tuberculous pyelitis, yet the outlying cortical tissue of the portion connected with an uninvolved pelvis shows microscopic tubercles.

I have repeated the work of Ekehorn and believe that it represents the facts. Yet in many instances, I find in apparently normal portions of the cortex, remote from the region of the primary focus, numerous

Fig. 2.—A small area of diffuse tuberculous infiltration, $\times 70$, just beneath the capsule in a portion of a kidney macroscopically normal. There is a small focus of active tuberculosis in the opposite pole from where this section was taken. Note that the glomeruli are not involved. Tubercle bacilli were found in the region of the giant cells near the central portion of the area. (Photomicrograph by L. S. Brown.)

microscopic tubercles just beneath the true capsule which seem not to be connected with the pelvis by the usual fine lines of round cell and endothelial cell infiltration which is usually found where there is extension to the cortex from an underlying caseating cavity. Also in the one double kidney there was no demonstrable process in the pelvis or medullary portions of the other half, yet the cortex showed numerous small tubercles beneath the capsule. I believe that these lesions are due to lateral extension by way of the cortical lymphatics.

Kumita,² Sakata³ and Sugimura⁴ have demonstrated in the fatty capsule of the kidney two intercommunicating plexus of lymph channels which in turn connect with a lymph plexus just beneath the true capsule. Lymph channels from the parenchyma drain into the lymph plexus just beneath the true capsule and from that may communicate with those of the fatty capsule. Undoubtedly the intertubular lymph spaces play an important part in spreading infection between pelvis and cortex. With these spaces invaded, infection of the greater lymph spaces of the cortex is easily accomplished. By this means tubercle bacilli may be distributed along the cortex to portions of the kidney remote from the primary lesion; but since the lymph channels of the separate portions of a double kidney and of fused kidneys intercommunicate, cortical invasion of the second kidney may also be expected.

I am unable to offer an opinion as to how great a part these microscopic tubercles play in accomplishing the destruction of the kidney. The small cortical tubercle, indeed, is often found sclerosed. Yet I am not prepared to deny that secondary tubercles may not develop deeper in the kidney tissue from these cortical tubercles, and these secondary lesions be of considerable significance in accomplishing the destruction of the kidney. In one or two instances, I have found beginning tubercles containing bacilli a short distance down in the kidney substance directly beneath the cortical tubercle, situated in a medullary ray and connected with the tubercle by lines of tuberculous infiltration. The fact that the small cortical lesions actually do contain tubercle bacilli; that they may be found in portions of the kidney that are macroscopically normal, and that they may even extend across the line of division between the halves of a double kidney, seems to me to indicate an origin separate from the process in the pelvis.⁵

2. Kumita: Ueber die Lymphgefäße der Nieren- und Nebennierenkapsel, Arch. f. Anat. u. Entwicklungsgesch, 49-58, 1909.

3. Sakata, K.: Ueber den Lymphapparat des Harnleiters, Arch. f. Anat. u. Entwicklungsgesch, 1, 1903.

4. Sugimura, S.: Ueber die Beteiligung der Ureteren an der akuten Blasenentzündung usw, Virchow's Arch. f. Path. Anat. **206**:20, 1911.

5. The contents of this paper are embodied in the section on "The Pathology of Renal Tuberculosis" in Cabot's System of Urology.