CASES OF RENAL INFECTION IN PULMONARY TUBERCULOSIS

EVIDENCE OF HEALED TUBERCULOUS LESIONS *

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Renal tuberculosis as seen in clinical practice comprises, as stated by Caulk,[1] 30 per cent of all surgical lesions of the kidney. These cases are usually diagnosed at a date when there is no apparent clinical manifestation of tuberculous lesions in any other part of the body and hence are looked upon as primary lesions. The extensive destruction of kidney tissue as found in such cases has led to the belief that tuberculosis of the kidney is a progressive destructive lesion which does not heal. Because of this interpretation it is now a common belief that whenever tubercle bacilli are found in one ureteral urine and not in the other, the kidney from which the bacilli are excreted should be removed regardless of the size of the lesion. The truth or falsity of this belief, that renal tuberculosis does not heal, is the main consideration of this paper.

Renal tuberculosis with cavitation, that is renal phthisis, would appear to correspond to the stage in pulmonary tuberculosis where cavitation is present. They are both advanced stages of the disease. Since it is a well established fact that all cases of tuberculous infection of the lung do not go on to cavitation, it would seem quite probable that the same fact would apply to tuberculous infection of the kidney. If the pathologic study of pulmonary tuberculosis were limited to cases with cavitation it would not give a true conception of the disease in its entirety as it occurs in the lung. It would seem plausible that this might also be the case in tuberculous infection of the kidney.

With the above consideration in mind, it was decided to study the kidneys from patients dying of pulmonary tuberculosis. The cases chosen gave no clinical symptoms of renal involvement. No case of renal phthisis was included. Thirty cases were selected all of which showed active tuberculous lesions of the lung with caseation. The

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immediate cause of death was advanced pulmonary tuberculosis in twenty-seven cases, tuberculous meningitis secondary to pulmonary tuberculosis in two cases and streptococcal abscesses of the brain with advanced pulmonary tuberculosis in one case. The ages range from 18 months to 70 years.

Both kidneys were examined in fourteen cases. In sixteen cases only one kidney was available. In all, forty-four kidneys have been examined.

The technic of gross examination used was as follows. The kidneys were split open lengthwise and fixed *in toto* for forty-eight to ninety-six hours in 10 per cent formalin. They were then separated into two halves and each half was cut into strips from two to three millimeters in thickness. Portions of the kidney tissue which appeared abnormal were cut out for microscopic study. In those cases where definite tuberculous lesions were numerous, the smaller lesions only were saved. All areas where there was a depression in the kidney surface were also removed for histologic examination. With this technic it was much easier to be certain of small lesions in the kidney tissue than in fresh unfixed organs. The technic is admittedly faulty in that without doubt many small lesions were overlooked. But the labor involved in proper histologic preparations of entire kidneys necessitated the removal of small blocks of tissue for microscopic study.

The blocks of tissue were embedded in paraffin in the usual manner. The tissue was then serially sectioned and mounted on glass slides. Every third slide was stained for tubercle bacilli by the use of Verhoeff's carbol-fuchsin. The sections were first overstained with hematoxylin and then placed in cold carbol-fuchsin over night. One per cent hydrochloric acid in 80 per cent alcohol was used as the discharging fluid, the sections being treated for five to ten minutes. The remainder of the sections were stained with hematoxylin and eosin.

In this study approximately 100,000 sections have been carefully examined. Serial sections were found invaluable as entire lesions could be followed through. In the case of scars it was thus possible to determine whether the reparative process simply surrounded a lesion incompletely healed or whether the lesion was completely healed. As high as fifty sections in some of the scars were carefully examined for the presence of tubercle bacilli. By this method it was

also possible to demonstrate tubercle bacilli in lesions where single sections would have yielded negative results. In some lesions the bacilli were so scarce that but one bacillus could be found in ten sections. In other lesions a single section would show hundreds of the organisms. With this technic one was able to determine whether the lesion was entirely cortical, entirely medullary or cortico-medullary. It was possible also to find many microscopic lesions that would have escaped detection by any other technic.

In all, 367 definite tuberculous lesions were studied. These lesions varied from small tuberculous abscesses to lesions in which the only evidence of their tuberculous nature was revealed by the presence of an occasional giant cell or of a microscopic mononuclear tubercle. Tubercle bacilli were found in 265 of these lesions. Many of the small lesions were not stained for tubercle bacilli. Others were stained but no tubercle bacilli were found and in these cases the only evidence that the process was tuberculous in nature was the presence of one or more giant cells. There was great variation in the number of tubercle bacilli in the different cases. In one case fifty-four separate lesions were studied and tubercle bacilli were numerous in every lesion. In another case twenty-nine separate lesions were studied and tubercle bacilli were found, after prolonged search, in but one lesion.

The distribution of the lesions within the kidney tissue was as follows. Cortical lesions numbered 277 or a fraction over 75 per cent; medullary lesions were present forty times or about 11 per cent; and cortico-medullary lesions numbered 50 or about 13 per cent.

A study of the point of origin of the lesions proved of interest. For this study the smaller or microscopic lesions were relied upon, as the larger lesions involved too much tissue to make it possible to determine with assurance the exact point of origin. The smaller lesions were of two main types. The most common of these types was of vascular origin within the capillary tuft of a glomerulus, within a capillary between the convoluted tubules or within a capillary between the collecting tubules in the pyramids. Of these points of origin the most common was within a glomerulus and the least common in the pyramid. In the early glomerular lesion several instances were found in which one half of the glomerulus was involved while the remainder was normal.

The second type of small lesion had its origin within the lumen of

tubules. These lesions were found with about equal frequency in the lowest point of the loop of Henle and in the collecting tubules in the pyramid. They always subtended an ulcerating tuberculous lesion of some portion of the kidney substance. It appears that this type of lesion is always secondary to a vascular lesion which has developed to a point where destruction of tissue has supervened and tubercle bacilli have been discharged into the lumen of a tubule. In some instances the primary vascular lesion was a small tuberculous abscess involving but one glomerulus.

The points of prime importance in this study were the search for scars in the kidney tissue and the study of them. At the outset the difficulty of the interpretation of scars in the kidney was realized. Scars as the end result of infarction are so typical that their interpretation affords little difficulty. Scars occurring in kidneys where there was evidence of atherosclerosis were disregarded in the study. The healed lesion of infections other than tuberculosis would appear to be impossible to separate from healed tuberculous lesions in which caseation had been absent or at most was scanty. In other words, in a considerable portion of healed tuberculous lesions one could hardly expect to find anything which would label it as tuberculous in nature. This certainly is true in other organs of the body. Sclerosed glomeruli even in young individuals where there was no evidence of atherosclerosis were disregarded in this study, although it was felt that in all probability at least some of them represented the healed stage of a tuberculous process within the glomerulus.

Of the thirty cases studied, two gave no evidence of tuberculous lesions or scars. In twenty-two cases, definite tuberculous lesions were found and of these cases fourteen showed scars also. The remaining six cases showed scars only. Of the twenty cases which showed scars, three showed definite atherosclerotic lesions. Two of these sclerotic cases had definite tuberculous lesions and the third did not. The seventeen cases which did not have atherosclerotic lesions were also free from infarction except in connection with some of the larger tuberculous lesions. Not all of the scars were tabulated, but the following was the distribution of 100 of these lesions: cortical, 80; medullary, 14; and cortico-medullary, 6. From this it will be seen that the scars were distributed in the different renal regions in approximately the same proportion as were the active lesions.

The size and histologic picture of these scars varied considerably. Most of the scars were microscopic in size. Thirty-one scars of macroscopic size were found and the largest of these measured 3 mm. in diameter. All of the lesions showed destruction of kidney tissue, which varied from very slight to a rather large area of destruction, with a replacement of the area by connective tissue. In some of the larger lesions lymphocytic infiltration was marked but this was not constant, for many of the lesions showed no lymphocytic infiltration except perhaps an occasional lymphocyte in the tissue around the periphery. The density of the connective tissue varied greatly, being very compact in a few of the scars and of a rather loose texture in the majority of the lesions.

In all of the larger lesions some normal kidney tissue was present within the scarred area. If the lesion were cortical, normal glomeruli and convoluted tubules were present, while in the medullary region normal collecting tubules were present. These normal structures were always much fewer in number in these areas than in the surrounding normal tissue. At the periphery of the lesion it was common to find dilated tubules filled with hyaline material. This condition was interpreted as being due either to pressure upon a tubule as it passed through the scarred area or to the destruction of the portion involved in the scarred area with the peripheral portion remaining functional. In some of the larger cortical lesions a rare sclerosed glomerulus was found. Four scars contained small irregular particles which resembled bits of old caseous material.

One case in this series will serve to illustrate a condition which is probably quite common among tuberculous individuals. The patient was a man of about 35 years who entered the sanatorium at Mt. McGregor, New York, eight years before his death, as a case of far advanced pulmonary tuberculosis. For two years he underwent rigid sanatorium treatment. At the end of this time he was sufficiently improved to run the print shop at the sanatorium. This work he continued uninterruptedly until two weeks before his death. Monthly examinations were made during these six years and there was no evidence of a recrudescence of the pulmonary lesion, though his sputum remained positive. He never had any clinical symptoms pointing toward renal involvment. About two weeks before his death he developed a hemiplegia following a rather severe bronchitis.

Necropsy of the above case showed death to be due to two streptococcal brain abscesses. One lung showed an old fibroid phthisis and the other multiple old tuberculous lesions. There were many old tuberculous lesions in the spleen and liver.

Both kidneys showed tuberculous lesions. Six thousand sections from these kidneys were examined. There were 33 tuberculous lesions of which 25 were cortical, 5 were cortico-medullary and 3 were medullary. Tubercle bacilli were extremely hard to find and were demonstrated in but nine lesions. Only six lesions showed areas of caseation. In many of the lesions the only evidence suggesting tuberculosis was the presence of one or more giant cells. Besides these lesions there were numerous scars such as have been described above. These scars were present in the cortex and in the pyramids. Two lesions which in gross appeared to be caseous areas proved on section to be old caseous material, containing numerous cholesterin crystals, with a fibrous wall infiltrated with lymphocytes and an occasional mononuclear leucocyte completely surrounding it. Over 200 sections in these two lesions were carefully searched for tubercle bacilli. Two bacilli were found in one lesion and none was found in the other. These lesions at a previous date were undoubtedly active, ulcerating, caseating lesions. When examined, while not completely healed, they were so thoroughly walled off as to be innocuous to the individual. It would seem highly improbable that tuberculous kidneys such as these would excrete tubercle bacilli in the urine at this stage.

As stated above, both kidneys were examined in fourteen cases. Two of these cases were entirely negative as far as my examination went. One case showed only scars in both kidneys. The remaining eleven cases had tuberculous lesions in both kidneys. In several cases the lesions were larger in one kidney than in the other. The reason for this difference is not certain, although the chance of bacillary dosage would appear the most logical explanation.

Small benign tumors were encountered in six of the thirty cases. These tumors were multiple. In three cases papillary adenomas of the cortex were present. Two cases showed fibromas of the medullary region. One case had both types of tumor present. Microscopically, neither type of tumor could be confused with scars or active tuberculous lesions, as they were encapsulated and had definite tumor architecture.

As a control to the above series of tuberculous kidneys, the kidneys from twenty-two necropsies on non-tuberculous individuals were examined by the same technic. Only sufficient sections were examined to ascertain the nature of the pathologic lesions found. It was not deemed necessary to follow the lesion through in serial section. The age range of the cases was from 6 months to 76 years. The list of diseases is as follows: bronchopneumonia, 3; pernicious anemia, 2; ascending bilateral pyelonephritis, 3; endocarditis, 3; cardio-renal, 3; malignancy, 4; chronic glomerulonephritis, 1; gummas of liver with ruptured esophageal varix, 1; and *Staphylococcus aureus* septicemia, 2.

The lesions observed in the kidneys of these cases were as follows: atherosclerotic scars, 7; infarcts (cases of endocarditis), 2; chronic glomerulonephritis, 1; acute infectious nephritis with abscess formation, 5; small cortical scars, 2 (both were cases of endocarditis); fibroma (medullary), 1; papillary adenoma (cortical), 2; and negative, 8.

The kidneys showing infectious lesions and scars were more thoroughly studied than the remaining cases. The cases of *Staphylococcus aureus* infection were in young individuals and the distribution of the lesions in the kidney tissue was very similar to the distribution in tuberculous infection. Cortical lesions were by far the most common, but cortico-medullary and medullary lesions were also present. The lesions were bilateral in both instances.

The examples of bilateral pyelonephritis occurred in adult males and were the result of obstruction in the lower genito-urinary tract. One of these cases showed rather extensive atherosclerotic scars. A second had slight diffuse scarring but no healed lesions similar to those seen in the tuberculous cases and the third was devoid of scars.

The two kidneys which showed cortical scars were in young adults with endocarditis. There was no evidence of atherosclerosis. All of the scars observed were cortical and appeared to be healed infectious lesions. These lesions were not numerous. They were indistinguishable from many of the scars found in the tuberculous kidneys.

Discussion

In order to interpret the pathologic picture presented in any tuberculous process it is essential to have in mind the pathogenesis of the tuberculous lesion. For a full consideration of the cytologic reaction

and its meaning in tuberculosis the reader is referred to two articles now in process of publication. These articles will appear in the American Journal of Pathology and they express my interpretation of the tuberculous process. A very brief résumé of this interpretation will be given here to aid in clarifying the opinions given below.

The first and typical reaction to the tubercle bacillus is the "epithelioid" or mononuclear tubercle. If the individual has high resistance, caseation does not ensue and the hyperplastic type of tuberculosis is produced. In such cases, if the tubercle bacilli are destroyed, as undoubtedly they often are, the end result is a small scar.

If the individual is unable to cope with the infection, polymorphonuclear leucocytes are attracted to the injured tissue and an abscess is formed. In case this area is so situated that the necrosing tissue can be discharged to the outside, ulceration or cavitation occurs. It is at this stage of the process that tubercle bacilli occur in the sputum or in the urine. In case this discharge cannot occur, caseation supervenes through the death and disintegration of all cells within the abscessed area. Subsequent to caseation the polymorphonuclear leucocytes do not appear to be further attracted to the lesion.

Following caseation, that is in the reparative stage of the disease, the mononuclear leucocytes and lymphocytes are attracted in large numbers. It is in this stage of the process that giant cells appear, so that whenever giant cells occur in a tuberculous lesion it is definite evidence of a reparative reaction on the part of the host. As a reparative process goes on toward completion the need for mononuclear leucocytes and lymphocytes becomes less and less until the end product is a scar with nothing pathognomonic of the etiologic factor which was the responsible agent in the infection.

If the above interpretation of the tuberculous process from its inception to the healed stage is, in the main, correct, then the interpretation of the tuberculous lesions as found in the kidney can be set forth with a fair degree of accuracy. From this study it appears that the tuberculous lesion in the kidney does not differ from that in other organs and tissues of the body, providing the differences in histologic and anatomic structure are borne in mind. As far as can be determined, the same types of cells participate in the defensive and reparative processes of the tuberculous lesion in all tissues and organs.

It was not uncommon in this study to find a great variety of tuberculous lesions in a single kidney. Mononuclear tubercles, tuberculous abscesses, areas of caseation, scarred areas infiltrated with lymphocytes and with one to many giant cells present, and scars devoid of lymphocytic or mononuclear leucocytic infiltration have all been observed in one organ. From this is would appear that the individual had had, at intervals, showers of tubercle bacilli in the blood stream and these showers have been followed by the development of tuberculous lesions in the kidney. The pathologic processes found in such organs represent, then, lesions of different age and severity, and the scars represent the healed stage in an area where the tubercle bacilli have been successfully overcome. The fact that out of twenty-two cases with definite tuberculous lesions, twelve with no evidence of atherosclerosis showed scars, whereas out of twenty non-tuberculous cases, in only two with no evidence of atherosclerosis were scars found, leads one to the logical conclusion that at least a portion of these scars, and in all probability a goodly portion, represent healed tuberculous lesions.

The cases cited above did not show completely healed renal tuberculosis. At least three of the cases would have gone on to extensive destruction of kidney tissue, if one may forecast this condition from the numbers of tubercle bacilli present and the severity of the inflammatory process. On the other hand, five cases out of thirty studied showed scars only. These cases showed no evidence of atherosclerosis. If one grants that at least a portion of these scars represent healed tuberculous lesions, then there is definite evidence that, under favorable circumstances, renal tuberculosis can heal completely.

A fact of some surprise in this study was that none of the cases presented clinical manifestations pointing toward renal involvement. A study of the urine for tubercle bacilli was not made. Judging from the presence of inflammatory exudate and tubercle bacilli in the lumen of tubules, six of the cases or 20 per cent should have had bacilluria. Brown [2] reported 10 per cent positive urines in 104 cases and Hobbs [3] reported 6 per cent in 100 cases where there were no clinical manifestations of renal involvement. So it is apparent that renal tuberculosis with bacilluria can exist without causing clinical manifestations. The authors here quoted, and others, believe that bacilluria is at times encountered in the absence of kidney lesions.

My belief is that "excretory bacilluria" does not exist without ulcerative tuberculous lesions in the kidney. That these lesions are often microscopic and are many times overlooked is in all probability the reason for the belief in "excretory bacilluria." Such lesions may involve but a part of one glomerulus.

If one may judge by the absence of inflammatory exudate and bacilli in the lumen of tubules at least one-half of the cases in this series would not have shown tubercle bacilli in the urine. From this it would seem that renal tuberculosis can exist without bacilluria being present.

The common occurrence of tuberculous lesions in the kidney in cases of pulmonary tuberculosis was unexpected. This report shows a very high percentage. If a much larger number had been examined the percentage might have been lower. A probable explanation of the findings is that the examination of the kidneys have been much more thorough and that serial sections have revealed many lesions unsuspected on gross examination. If the findings in this study represent the true facts, it is apparent that every case of progressive pulmonary tuberculosis is a potential candidate for renal infection. It would also seem that cases of renal tuberculosis are secondary to some other tuberculous focus, usually pulmonary, in the body and that the infection is hematogenous. That the infection is hematogenous is indicated by the preponderance of cortical lesions.

Caulk [1] states that there is no authentic case on record of healed tuberculosis of the kidney. I have not been able to find such a case recorded. Hobbs [2] states in his article that he found an occasional scar and gives an illustration of the lesion. The illustration appears more like the fibromas I have encountered than like the scars I have described above. It would appear that the reason for no recorded case of healed renal tuberculosis is that the majority of cases studied have been of renal phthisis and that where tuberculosis of the kidney has been observed in the routine of a necropsy its occurrence has been automatically recorded without a thorough systematic study of the kidney with a view to determine the possibility of healed lesions in the same kidney. Renal phthisis does not afford suitable material for the study of healing of the tuberculous process, as it is an advanced progressive lesion. Healing of a cavitated kidney probably

does not occur. There is reason to believe, however, that under suitable circumstances such lesions may be clinically arrested.

It is now known that tuberculous lesions of the lung, of the intestine and of other tissues do heal. With the evidence given above regarding scars in the kidney in cases of tuberculosis, it would seem illogical to maintain the attitude that renal tuberculosis never heals.

The purpose of this paper is to present as completely and as concisely as possible the pathologic side of renal tuberculosis. The clinical side is another study but it would seem plausible that the more nearly the pathology is understood, the more sane will be the course of clinical treatment advised. The following pathologic facts are emphasized. The presence of tubercle bacilli in ureteral urine establishes the diagnosis of renal tuberculosis but not of renal phthisis. The absence of tubercle bacilli in the urine does not rule out tuberculosis of the kidney. Renal tuberculosis is of hematogenous origin and, as far as this study goes, when it occurs it is always bilateral. Tuberculous lesions of the kidney heal. Renal tuberculosis can exist without clinical manifestations.

With the above discussion in mind it would seem advisable to establish the following facts before nephrectomy for renal tuberculosis is undertaken: (1) evidence of considerable destruction and cavitation of the kidney; (2) the presence of tubercle bacilli in the urine on several examinations; (3) the absence of tubercle bacilli in the urine from the opposite kidney on several examinations; and (4), the failure of treatment on the same basis as for pulmonary tuberculosis to arrest the condition.

I have not felt it advisable to quote extensively from the literature on renal tuberculosis in this article. For the more important articles on the subject the reader is referred to the bibliography in a previous article by Dr. Sasano and myself.[4]

Conclusions

1. Renal tuberculosis is common in advanced pulmonary tuberculosis, twenty-two out of thirty cases.

2. Renal tuberculosis is hematogenous in origin, 75 per cent of the lesions being cortical.

3. Bilateral infection was the rule in every case in this series in which both kidneys were examined and tuberculous lesions were present.

4. Tuberculous lesions of the kidney heal. Scars were present in seventeen out of thirty cases. Twelve of these cases also had tuberculous lesions.

5. Serial sections are invaluable in such a study.

REFERENCES

1. Caulk, J. R. *J. Urol.*, 1921, vi, 97.
2. Brown, L. *J. A. M. A.*, 1915, lxiv, 886.
3. Hobbs, F. B. *Tubercle*, 1923, v, 57 and 105.
4. Medlar, E. M., and Sasano, K. T. *Am. Rev. Tuber.*, 1924, x, 370.

DESCRIPTION OF PLATES

PLATE 78

FIG. 1. A tubercle in a glomerulus. The lower part of the glomerulus is not involved. The tubercle shows evidence of injury to the tissue but no caseation. The capsule and pericapsular tissue is involved to the left. Sclerosed glomeruli showing only partial involvement of the capillary tuft such as this have been found. × 350.

FIG. 2. A small tuberculous abscess between the tubules in the cortex. The inflammatory exudate consists largely of polymorphonuclear leucocytes. The tubules to the left contain inflammatory exudate and tubercle bacilli. There is no evidence of caseation. The lesion is microscopic in size. × 350.

PLATE 79

FIG. 3. A tubercle in a glomerulus undergoing caseation at its periphery. This is in reality a small tuberculous abscess. Note the normal portion of the glomerulus above. × 350.

FIG. 4. A caseous lesion undoubtedly arising in the glomerulus in the center of the field. The dark portion is closely packed with "nuclear dust." This is a later stage of a lesion like Fig. 3. × 350.

PLATE 80

FIG. 5. A microscopic mononuclear tubercle in the tissue to the right of the glomerulus. × 350.

FIG. 6. A scar in a position similar to the tubercle in Fig. 5. Such scars were quite frequently found and represent what I believe to be healed tubercles which have not gone on to caseation. Note the fibrous thickening of the capsule of Bowman. × 350.

FIG. 7. A giant cell tubercle. This was the only evidence of tuberculosis in a series of 300 sections from a block of kidney tissue. Other larger tuberculous lesions were found in this same kidney. This giant cell extended through 20 sections 10 microns thick. It represents a tubercle which has undergone necrosis or caseation, probably the latter, and which is now undergoing repair. × 350.

PLATE 81

FIG. 8. A large cortical scar of macroscopic size with normal glomeruli and tubules in it. There is marked fibrosis and some lymphocytic infiltration. No evidence that this lesion was tuberculous was found but there were low grade tuberculous lesions in the same kidney. Note the dilated tubules above. This is a healed infectious lesion and the probability is in favor of it being a healed tuberculous lesion. × 100.

FIG. 9. A higher power of Fig. 8, showing a normal tubule, scar tissue and some lymphocytic infiltration. × 350.

PLATE 82

FIG. 10. A medullary scar of macroscopic size. In other sections this scar contained two dilated collecting tubules. × 100.

FIG. 11. A tuberculous lesion. The only evidence is three giant cells and a small mononuclear tubercle above. After prolonged search one tubercle bacillus was found. For all practical purposes this lesion is healed though pathologically it is not healed. × 200.

FIG. 12. A tuberculous lesion. The only evidence in over 100 sections was these two giant cells. No tubercle bacilli were found. × 350.

PLATE 83

FIG. 13. A large cortical scar from a tuberculous case. No active tuberculous lesions were found in this case. × 100.

FIG. 14. An old caseous area walled off by fibrous tissue. This is one of the lesions mentioned in the text. No tubercle bacilli were found after very careful search. × 100.

PLATE 84

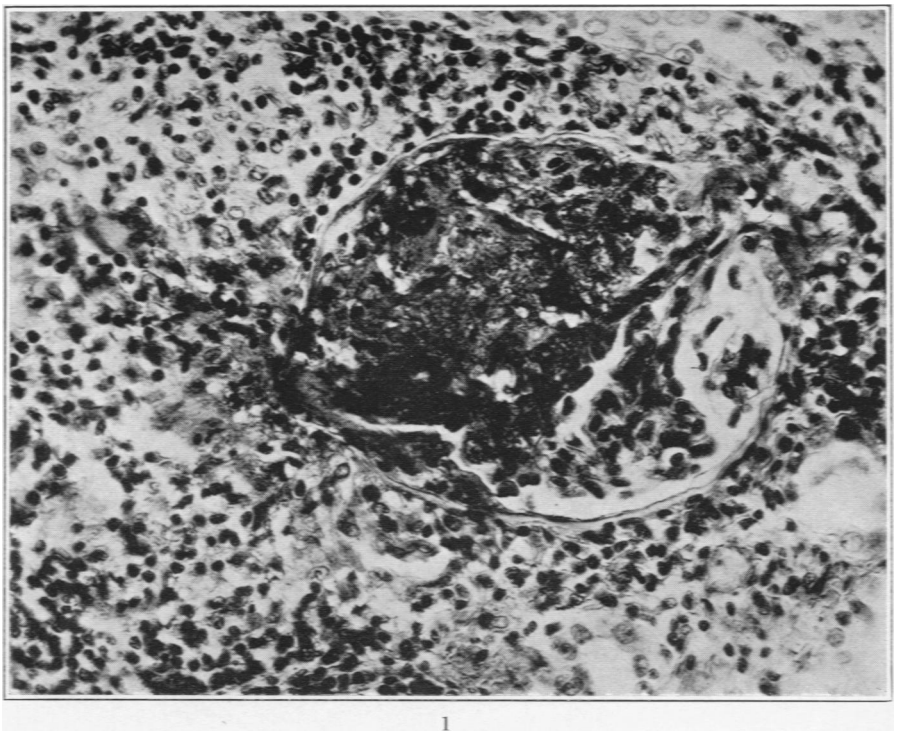
FIG. 15. A large cortical scar. The only evidence of its having been tuberculous is a giant cell at the left. × 200.

FIG. 16. A large medullary scar of macroscopic size. Note the normal tubules within the scarred area. Some of the tubules are considerably dilated. There are also small irregular hyaline masses in the tissue which I take to be bits of old caseous material. Four such scars were found in three cases of this series. × 200.

PLATE 85

FIG. 17. A medullary fibroma. Compare the architecture of the tumor with that of the scars in Fig. 9, 10, 12, 16 and 18. × 100.

FIG. 18. Dense fibrous scar. This represents a healed caseated tuberculous lesion. The sclerosed glomeruli shown above are typical of such lesions commonly seen in tuberculous kidneys which show no evidence of atherosclerosis. × 200.



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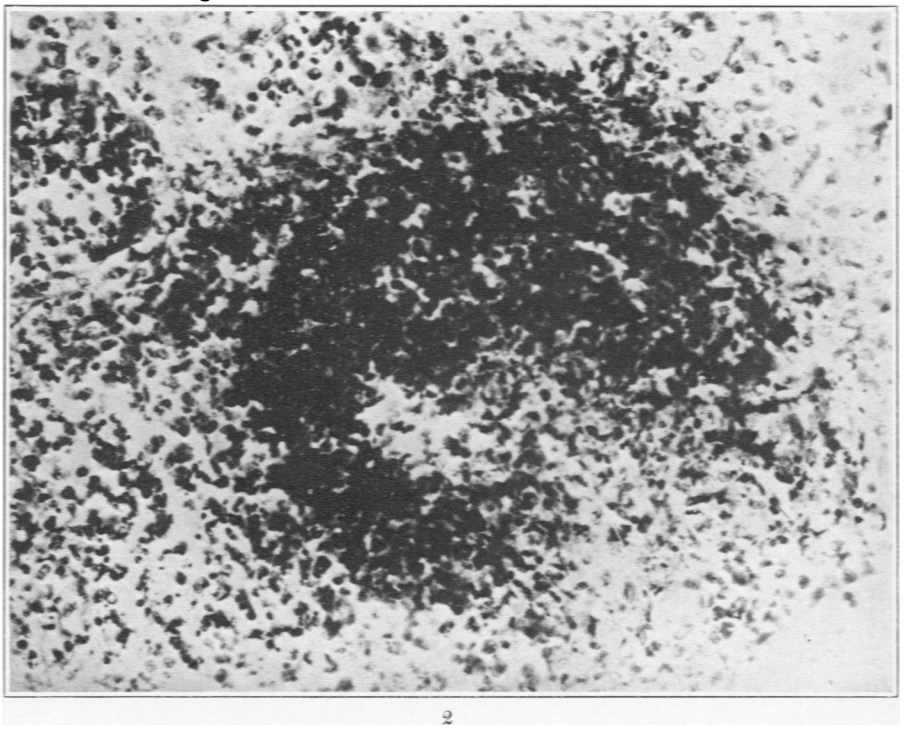

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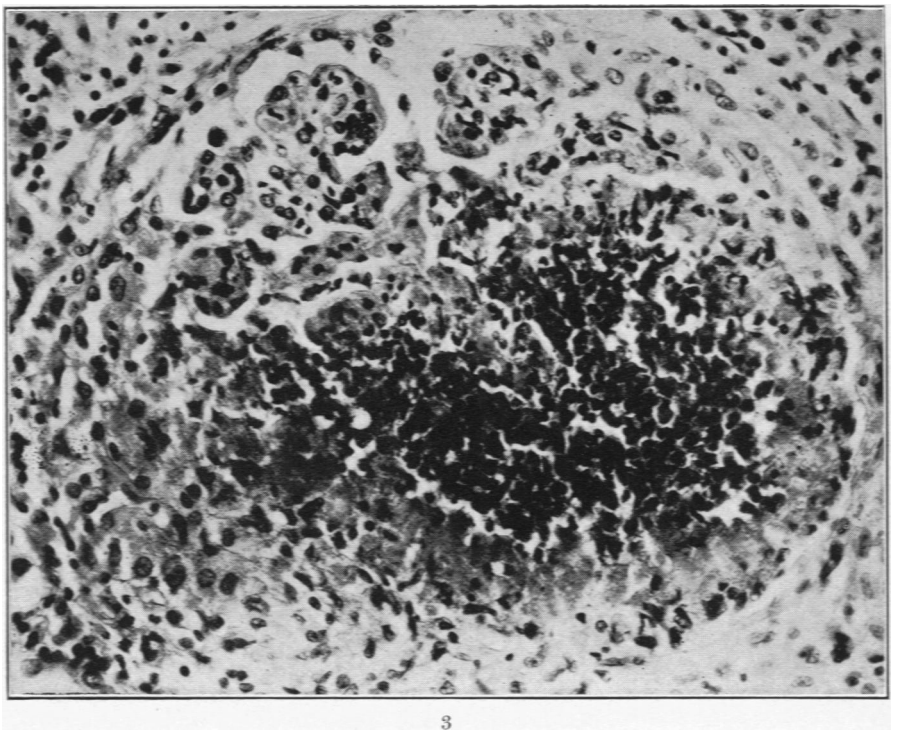

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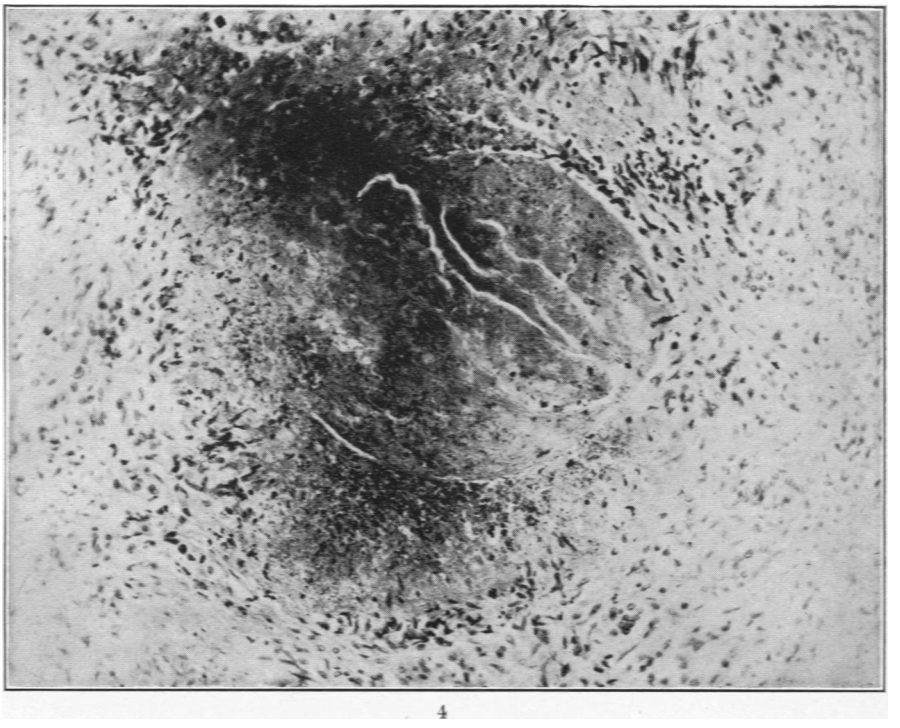

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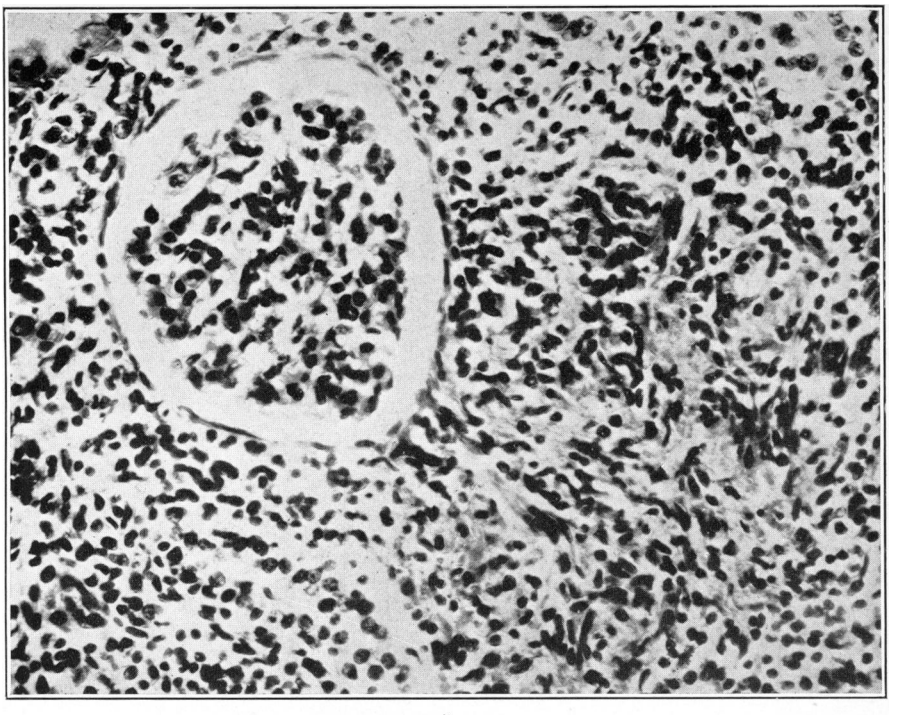

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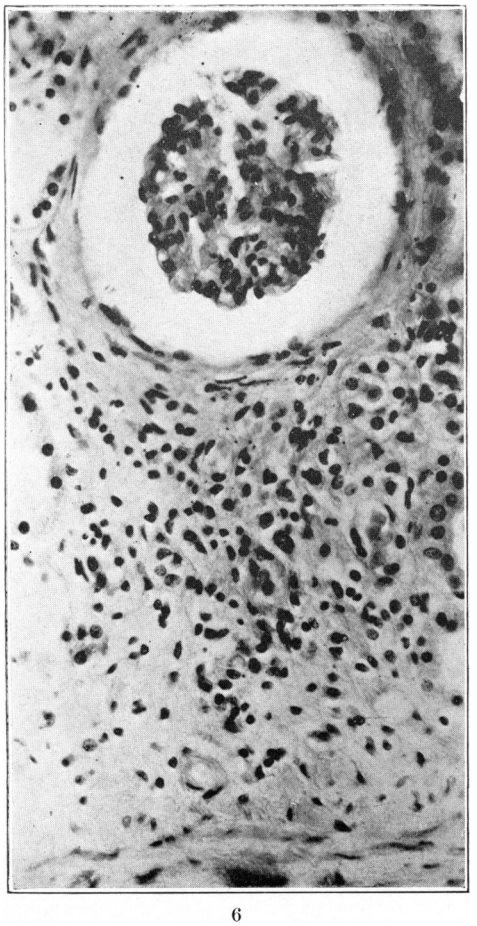

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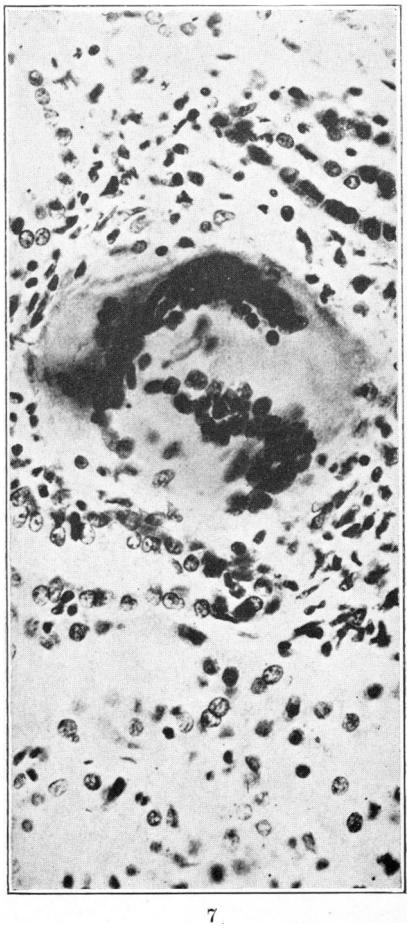

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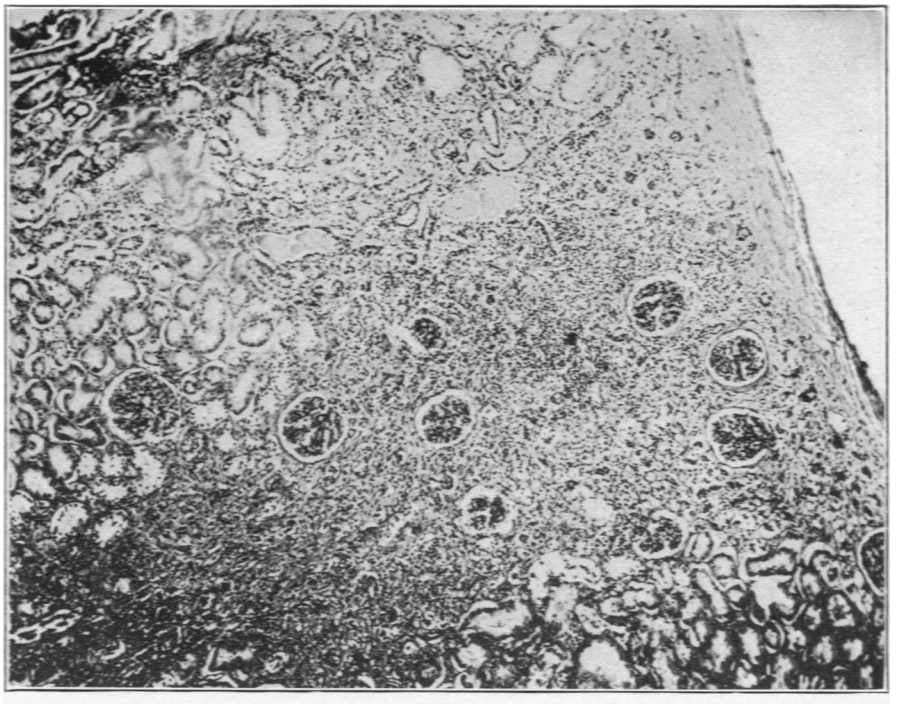
Renal Infection in Pulmonary Tuberculosis



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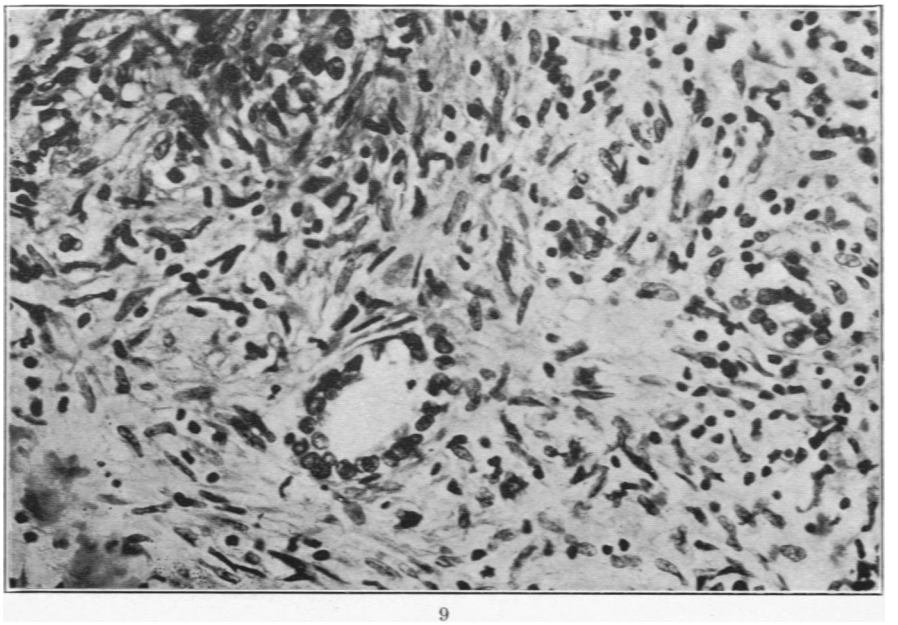

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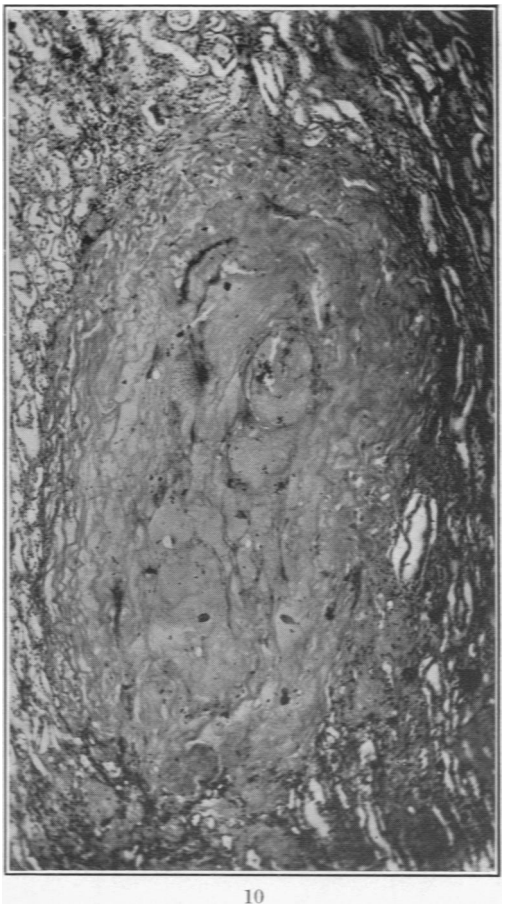

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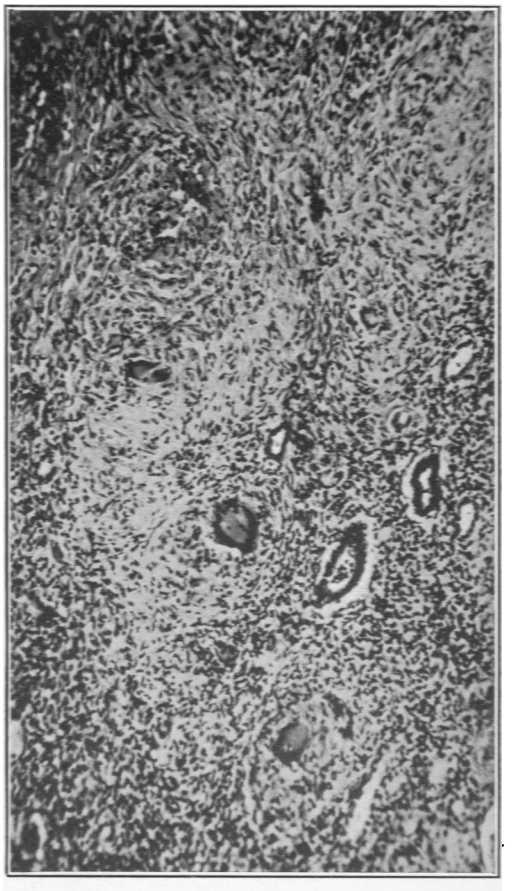

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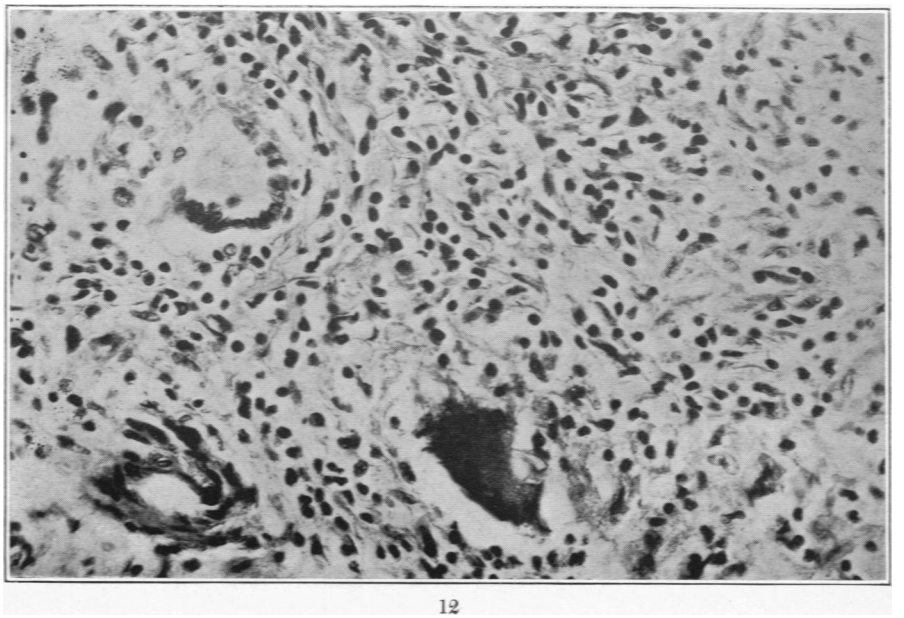

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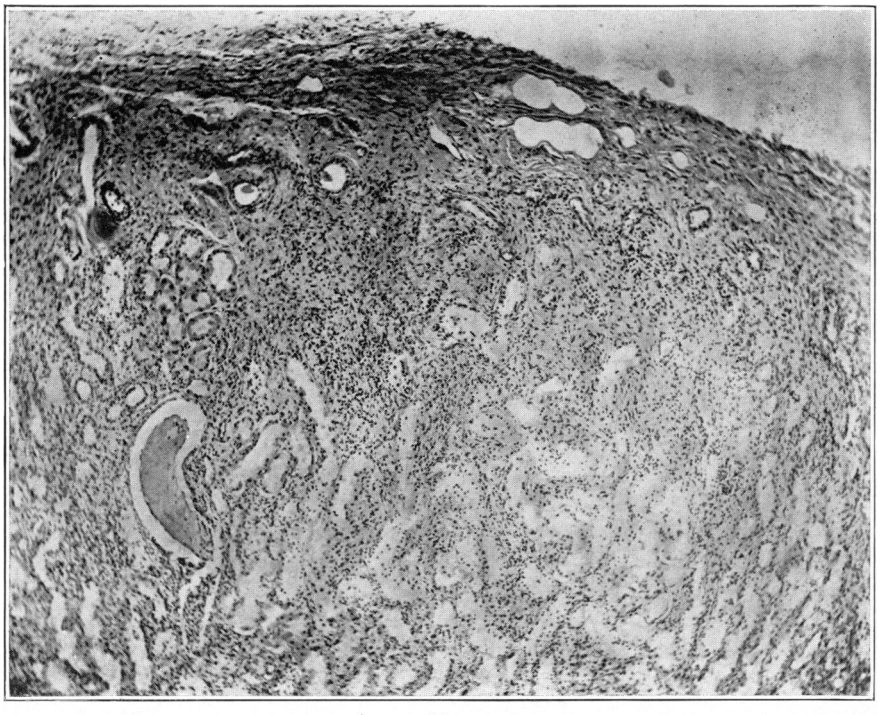

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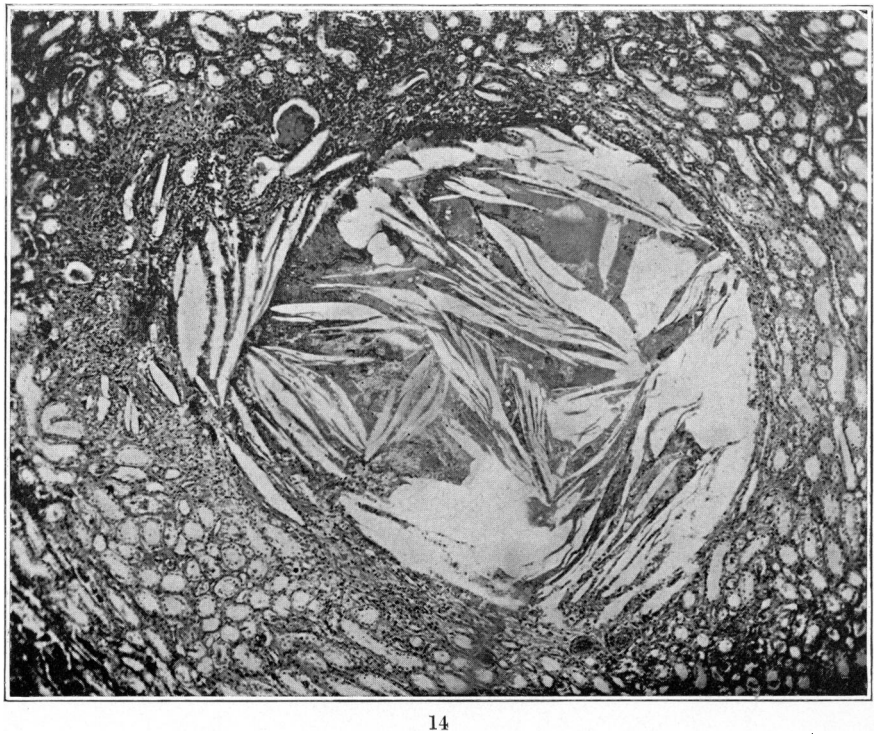

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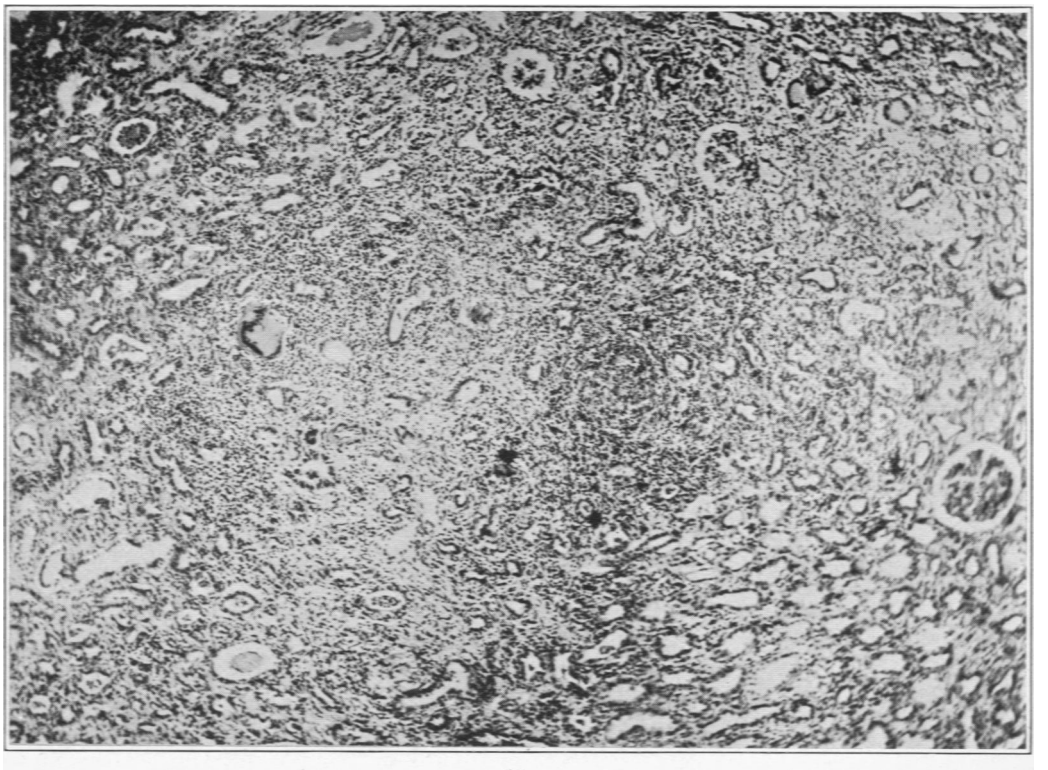

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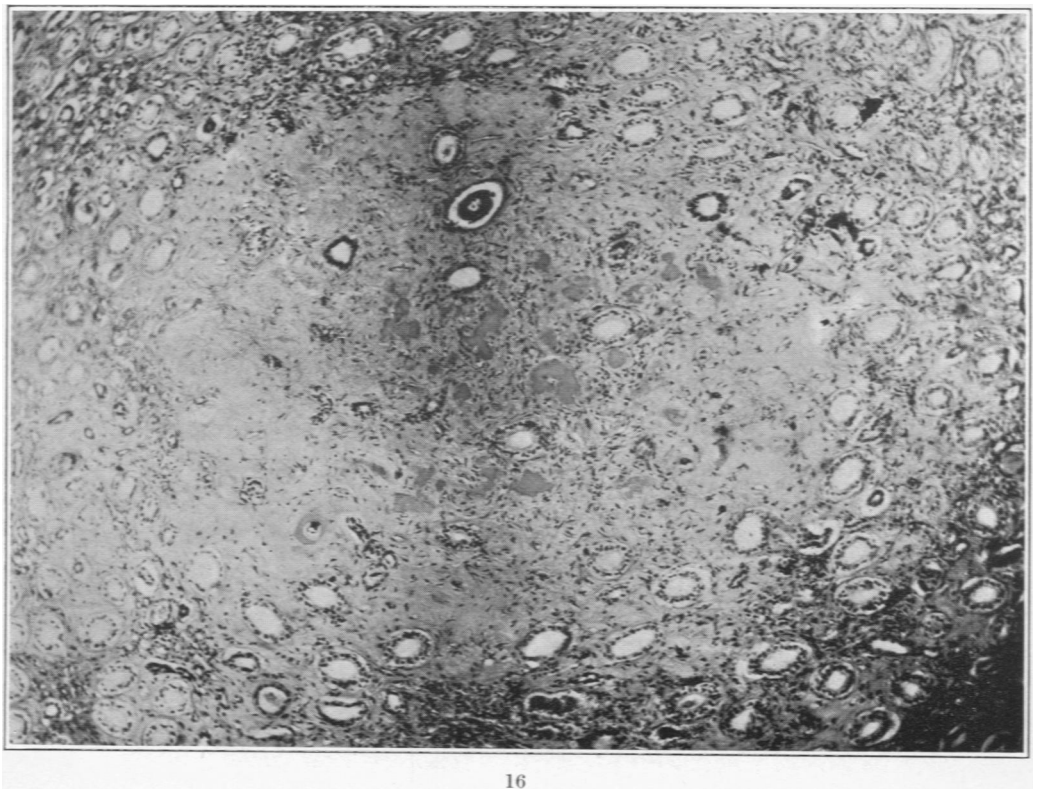

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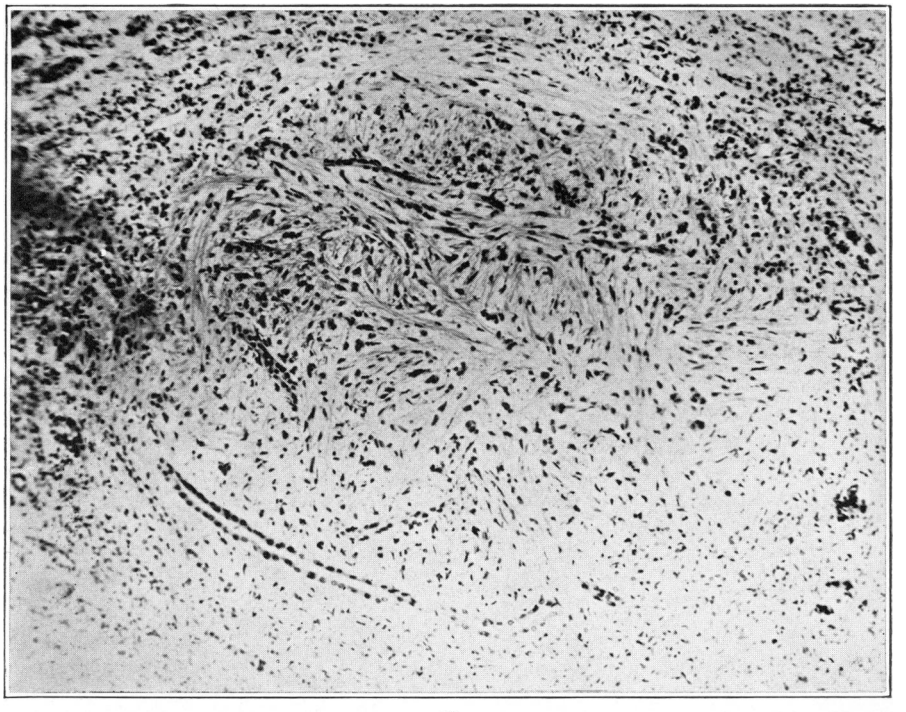

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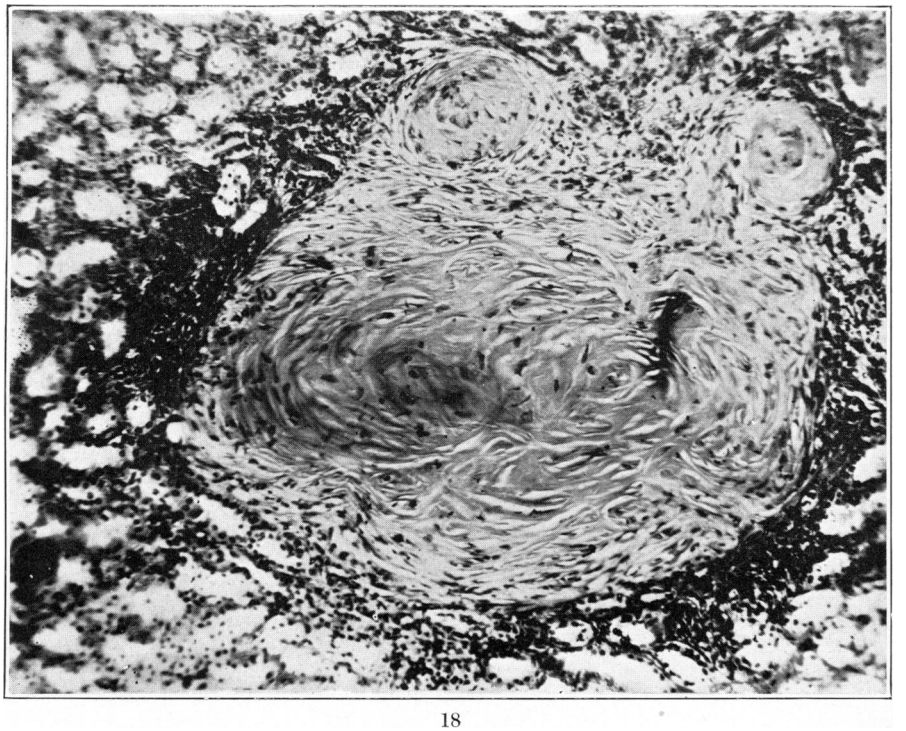

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