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BY

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Reprinted from THE LANCET, February 27, 1909

ANURIA WITH NECROSIS OF THE RENAL CONVOLUTED TUBULES.

TEN years ago Dr. J. Rose Bradford and Mr. T. W. P. Lawrence published a paper entitled "Endarteritis of the Renal Arteries Causing Necrosis of the Entire Cortex of Both Kidneys."[1] Their case was that of an anæmic-looking woman, aged 36 years, who was stated to be quite well up to a day on which she gave birth to a dead child. She lost a good deal of blood, but otherwise her labour appeared to have been a normal one. After her confinement she was sick once and complained only of headache and slight drowsiness. Yet there was practically complete anuria from the time of her confinement until her death seven days later. A few teaspoonsfuls which were obtained by catheterisation contained a small quantity of albumin and some pus. There was no fever. Though the patient gradually lost strength there was throughout a remarkable absence of the usual uræmic symptoms, such as coma, delirium, convulsions, vomiting, dyspnœa, amaurosis, &c. The clinical picture was, in fact, not that generally supposed to be associated with so-called "non-obstructive" suppression of urine (as occurring in Bright's disease), but rather that of "obstructive" suppression of urine, as occurring, for instance, when in an individual with only one kidney the ureter of that kidney becomes blocked by a calculus.

At the necropsy practically no disease was found except in the kidneys, both of which were slightly enlarged and as well macroscopically as microscopically similar to each other in appearance. The convoluted tubules throughout the whole of the cortical substance (including the columns of Bertini) of both kidneys were necrotic, with the exception of

[1] Journal of Pathology and Bacteriology, Edinburgh, 1898, vol. v., p. 195.

a very narrow zone at the junction of the cortex and medulla. The epithelial cells lining these tubules would hardly stain at all with logwood and their nuclei could not be differentiated from the cytoplasm. The glomerular epithelium was to some extent affected in the same way. A zone of the cortical tissue just beneath the capsule and a zone adjacent to the medulla showed congestion and small cell infiltration, but there was no increase of interstitial fibrous tissue in any part of either kidney. The renal artery and its primary branches showed some thickening, but the change on which the authors chiefly insisted was an arterial thrombosis, which apparently involved (and was practically confined to) all the interlobular arteries of both kidneys and their branches, including all the glomerular arterioles given off distally to the point of thrombosis (distally to the junction line of medulla and cortex). As far as I can make out from their paper Dr. Bradford and Mr. Lawrence regarded the thrombosis of the interlobular arteries as a sudden lesion perhaps in some way associated with the pregnancy and as the cause of the cortical necrosis and fatal anuria.

In 1905 Dr. W. S. A. Griffith and Dr. W. P. Herringham described a similar case under the heading "A Case of Necrosis of the Entire Renal Cortex of Both Kidneys, together with Thrombosis of all the Cortical Arteries, occurring in the Puerperal State."[2] The patient, a woman, aged 35 years, a multipara, who had had symptoms of chronic nephritis for several years, was delivered of a dead child and 12 ounces of old clot immediately after admission to the Queen Charlotte's Hospital. From that time until her death, nine and a half days later, she passed only ten ounces of urine, and none at all was passed during the last six days of her life. The small quantity of urine obtained for examination was clear and contained a trace of albumin and renal casts. Ordinary uræmic symptoms were almost absent till the end. At the post-mortem examination the left ventricle of the heart was found to be somewhat hypertrophied. The kidneys to the naked eye closely resembled those in Bradford and Lawrence's case, but microscopic sections showed a difference between the cases in two respects. In Griffith and Herringham's case, unlike that of Bradford and Lawrence,

[2] Published somewhat later in the Journal of Pathology and Bacteriology, 1906, vol. xi., p. 237.

there were (1) decided changes due to chronic interstitial nephritis, whilst (2) evidence of endarteritis was practically wanting. But the cortical necrosis, the thrombosis of the interlobular arteries, and the presence of areas of small cell infiltration were common to both cases.

On reading the account of the last case Dr. H. C. Lloyd of Melbourne, Australia, published an almost identical one in THE LANCET.[3] The patient, a woman, aged 39 years, a multipara, was delivered of a stillborn child in the Women's Hospital at Melbourne. From that time she passed no more than about 11 ounces of urine (which contained albumin) until her death, eleven days afterwards. There were several eclamptic convulsions during the first few hours after delivery and later some vomiting and uræmic twitchings, but consciousness was preserved till the end. The temperature was normal or subnormal throughout. At the necropsy the kidneys, according to the description of the pathologist, Dr. Constance Ellis, who examined them, resembled closely those in Griffith and Herringham's case, but (on microscopic examination) unlike those in Bradford and Lawrence's case (which they likewise macroscopically resembled), they showed the presence of chronic interstitial fibrosis without obvious endarteritis. In regard to the thrombi in the arteries Dr. Lloyd remarks that they "seem to indicate the possibility of some agglutinin, though why it should operate only in arteries in such a limited area is mysterious."

I gather that all the foregoing authors regard the thrombosis of the interlobular arteries as the cause of the necrotic changes in the renal cortex. This conclusion seems to me an exceedingly improbable one. There is nothing in the descriptions of the thrombi to indicate that they were formed several days before the death of the patients. Moreover, how can the simultaneous occurrence of thrombosis involving (but limited to) all the interlobular arteries of both kidneys be accounted for except as being in some way secondary to the inflammatory and degenerative changes involving the active secreting tissue (convoluted tubules) of both kidneys? In all these cases the presence of an inflammatory reaction of some kind was proved by the small cell infiltration observed. In the first two cases this was specially located in the portions of the cortex adjacent to the medulla, where,

[3] THE LANCET, Jan. 20th, 1906, p. 156.

in Griffith and Herringham's case, the infiltrating cells somewhat resembled pus cells. An explanation which makes the occurrence of thrombosis in some way secondary to acute changes in the convoluted tubules appears to me the most likely one to be correct, but before considering this question further I will shortly describe a recent case at the German Hospital which may perhaps help to throw a little light on the subject.

The patient was a strongly built man, aged 69 years, a cabinet-maker, who said that he had never been seriously ill before. His legs were somewhat swollen in August, 1908, and he had difficulty in passing urine. This was said to have contained albumin. Early on Nov. 20th he failed to pass any urine at all, and, after a medical man had tried in vain to draw off some with a catheter, he was sent to the German Hospital (Nov. 22nd) on the supposition that an operation was needed.

On admission to the surgical side of the hospital, under Dr. E. Michels,[4] considerable prostatic enlargement was felt and was thought to be due to carcinoma. The right leg was somewhat œdematous. Bronchitic sounds were heard over the chest. The systolic brachial blood pressure was found to be 180 millimetres of mercury. There was considerable tremor, which, however, appeared to be habitual and not of recent onset. The mental condition was quite normal. In the hospital no urine was passed and none could be obtained by catheterisation. Treatment by the hot-air bath, purgatives, subcutaneous injection of normal salt solution, and caffeine by the mouth made no difference. Dr. Michels (Nov. 24th) cut down on the right kidney from the loin, incised it, and inserted a drainage-tube into the renal pelvis, but the operation had no effect and no urine came from the wound. There was never any fever. The pulse-rate varied from 72 to 102 per minute. After Nov. 26th the patient was sometimes very restless, especially in the night, and he wandered in his mind. He became decidedly weaker and died on the evening of the 28th, about eight and a half days after the onset of complete anuria. His systolic brachial blood pressure on the morning of the day of his death was found to have

[4] I have to thank my colleague, Dr. Michels, with whom I saw the case in the hospital, for his kind permission to write this account. The case will probably be later described in greater detail by Dr. Jurasz, one of the house surgeons at the hospital.

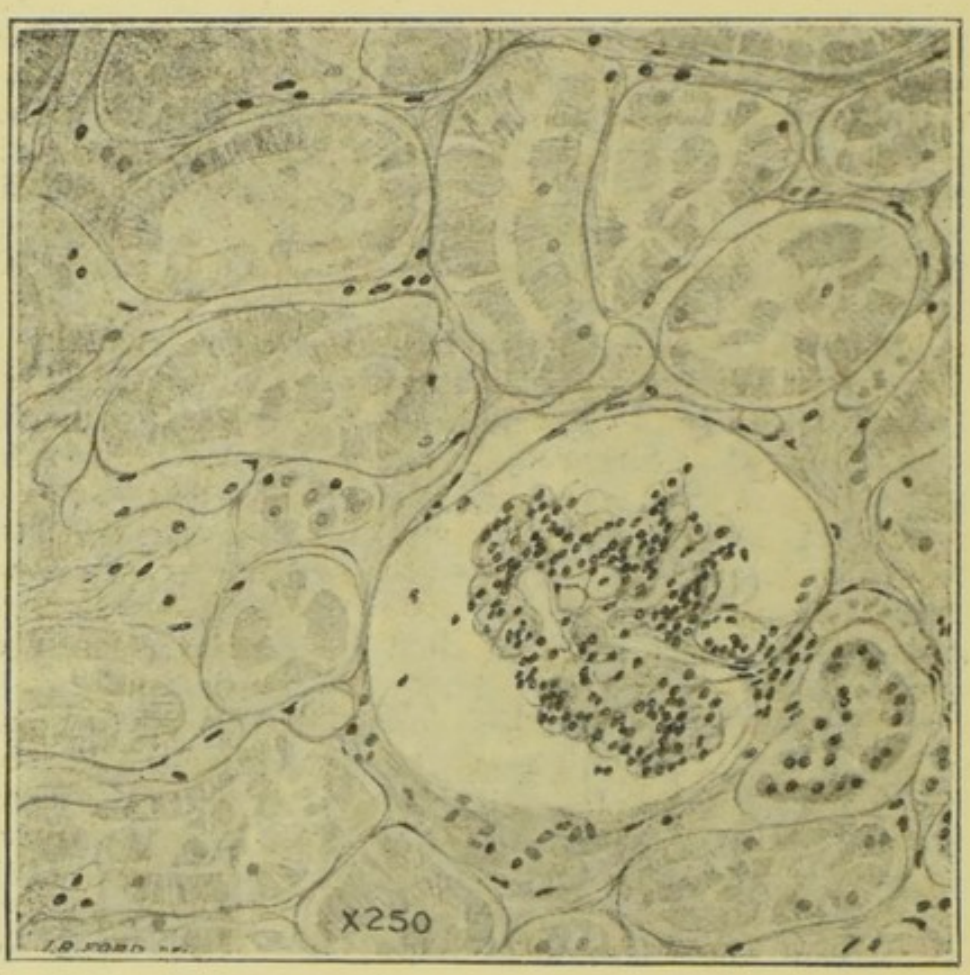
fallen (from 180) to 140 millimetres of mercury. There was considerable œdema of his legs at the end.

The post-mortem examination was practically limited to the prostate gland and the two kidneys. The prostate gland was much enlarged and microscopic examination showed the enlargement to be due to carcinoma. The prostatic tumour had, however, not obstructed the ureters, for there was no dilatation of ureter or renal pelvis on either side. The right kidney was somewhat contracted. The left kidney (the one not operated on) was large and congested; the markings dividing it into cortex and medulla were quite distinct; its capsule stripped readily.

Microscopic examination showed decided evidence of old chronic interstitial nephritis in both kidneys, but chiefly in the right (smaller) one. In the left kidney the evidence of old disease was limited to a slight amount of interstitial fibrocellular increase and to chronic degenerative change in a few of the Malpighian corpuscles. The acute recent parenchymatous changes could be best studied in the left kidney on account of its substance having been relatively little altered by older disease. The epithelium of the convoluted tubules had more or less all of it undergone a necrotic change. The secreting cells had a granular, "woolly" appearance and hardly took on the logwood stain at all; their nuclei had mostly disappeared (karyolysis) or could hardly be distinguished from the cytoplasm. The other renal tubules, glomeruli, and blood-vessels showed practically no changes excepting those due to a certain amount of old chronic interstitial nephritis. None of the blood-vessels seemed to be thrombosed. In addition to a certain amount of interstitial fibrosis there was here and there a little small cell infiltration, especially near the Malpighian corpuscles. Some of the interstitial tissue in the medulla appeared swollen and transparent as if it had undergone a hyaline or mucoid change. In the right (smaller) kidney the degeneration in the convoluted tubules was similar but could not be so well studied owing to the presence of considerable changes due to the old interstitial nephritis and small cell infiltration, the latter especially in the subcapsular portion of the cortex. Sections of both kidneys were specially stained for microbes, but those found appeared (from their position, &c.) to be of post-mortem growth only.

The above case resembles the three other cases in the

association of fatal so-called "non-obstructive" anuria with widespread necrosis of the secreting cells of the renal convoluted tubules, but it differs from the others chiefly in the absence of obvious microscopic signs of thrombosis of the



The figure is drawn from a portion of the cortex of the left kidney (magnification 250) and shows a Malpighian corpuscle surrounded by convoluted tubules, the epithelium of which is necrotic. The secreting cells are mostly detached or broken up and scarcely take on the logwood stain at all. Their nuclei have disappeared (karyolysis) or are hardly to be distinguished from the cytoplasm.

interlobular arteries of the kidneys. The Malpighian corpuscles showed little or no acute changes. It seems to me that the one essential change in all the above described cases is the widespread necrosis of the secreting cells of the renal convoluted tubules and that this change is

merely an extreme degree of the change known as "granular degeneration" or (in its earliest form) as "cloudy swelling," a change which is probably present to a greater or less extent in all cases of acute nephritis and in many toxæmic conditions. I suppose that when for any reason (e.g., violence of the local or general exciting cause, want of cell vitality from previous disease, or otherwise) the cloudy swelling and degeneration rapidly pass on to actual cell necrosis the convoluted tubules become mechanically blocked and more or less complete anuria results.[5] At a later stage perhaps the interlobular arteries and their glomerular branches become thrombosed (as in the three cases first referred to) and then the glomeruli likewise degenerate. As I have already explained, it is, I believe, more rational to regard thrombosis of all the interlobular arteries, when it occurs, as secondary to the parenchymatous necrosis than to regard the parenchymatous changes as altogether secondary to the arterial thrombosis. Perhaps the necrosis of the glandular cells produces a substance having a hæm-agglutinative action which favours thrombosis in the neighbouring blood-vessels.

Hæmaturia at the commencement of attacks of acute nephritis may have a beneficial effect by relieving pressure—that is to say, it may represent an automatic conservative measure on the part of the organism. Probably all cases of so-called non-obstructive suppression of urine (non-obstructive anuria) should be regarded as representing an exaggerated form of the functional and morphological changes in the renal cells that occur in all cases of acute nephritis or acute exacerbations of chronic nephritis or as the result of the actions of various toxic substances on the kidneys. What was the immediate exciting cause of the acute parenchymatous degeneration in the present case is not quite clear, but certainly the kidneys were already diseased; and in regard to another abdominal viscus—namely, the liver—it may be remembered that acute parenchymatous degeneration of hepatic cells is known occasionally to supervene in previously diseased (cirrhotic or enlarged and fatty) livers.

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[5] Perhaps temporary anuria may sometimes be a mechanical result of swelling (without actual necrosis) of the secreting cells of the convoluted tubules. I do not, however, wish to imply that all cases of so-called "non-obstructive anuria" are really obstructive and due to mechanical blocking of the convoluted tubules from swelling (with or without actual necrosis) of their epithelium.