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The intrarenal arterial pattern in chronic pyelonephritis*

A micro-angiographic and histologic study

By

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With 5 Figures in the Text

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Those authors who have made a particular study of the structure of the intrarenal vessels in chronic pyelonephritis seem to agree that thickening of the walls of the pre-glomerular vessels often constitutes a prominent feature of the renal morphology. There is, however, considerable uncertainty as to the cause of these vascular changes and their functional significance.

Linder (1937) pointed out that the changes in the vessel walls in the chronic pyelonephritic kidney varied in severity with the intensity of the renal inflammation. In a comprehensive study, Weiss and Parker (1939) went further and ascribed the vascular changes to the chronic inflammation. Kincaid-Smith (1955) suggested that the vessels had been damaged by acute inflammation, with consequent thrombosis or scarring of their walls. Holle (1959), on the other hand, found no signs of arteritis in the pyelonephritic kidney, and attributed the wall changes to deficient nutrition, resulting from fibrotic obliteration of the perivascular lymphatics.

Weiss and Parker (1939) and Kincaid-Smith (1955) suggested that the narrowing of the intrarenal arteries and arterioles in chronic pyelonephritis gives rise to cortical ischaemia and hence to the hypertension that also others have found to be a common complication of the disease (e. g. Butler 1937, Longcope 1937, Raaschou 1948, Brod 1957, Saphir and Cohen 1959, Kleeman et al. 1960). This explanation of the hypertension has not been generally accepted, since severe vascular changes have been noted also in normotensives (Brod 1957, Kleeman et al. 1960); it has even been suggested that the occurrence of hypertension in chronic pyelonephritis is merely coincidental (Bell 1950, Gloor 1961).

The contraction of the chronic pyelonephritic kidney was considered by Kincaid-Smith to be mainly of ischaemic origin, and she suggested that chronic inflammation developed in the ischaemic areas as a secondary feature. Heptinstall et al. (1960) found such a mechanism unlikely and demonstrated experimentally that vascular changes may be very mild even in severely contracted kidneys. They ascribed the contraction solely to the inflammation.

Heptinstall et al. based their opinion on gross angiograms of the affected kidneys, which displayed some irregularity in the vascular pattern of the inflamed areas. Previous histologic investigations have shown, however, that the main changes occur in the finer intrarenal arteries, but no injection studies of these vascular changes have been performed. In the present study the angiographic technique is extended to the microscopic level and combined with histologic examination of the injected specimens. The findings are compared with

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those obtained in combined micro-angiographic and histologic examinations of the normal and hypertensive vascular patterns of the human kidney (LJUNGQVIST and LAGERGREN 1962, LJUNGQVIST).

Material and methods

The study was performed on 15 kidneys from as many patients with histologically verified chronic non-specific pyelonephritis. Eight of the kidneys were obtained at operation (cases 1 and 3—9, Table), and the others at autopsy. In all cases urine samples indicated infection of the urinary tract. The age and sex distributions and the presence of hypertension and hydronephrosis are given in the Table. In those cases where no ophthalmoscopy had been

Table

Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Sex	♀	♂	♀	♂	♀	♀	♀	♀	♀	♀	♂	♂	♀	♂	♀
Age	34	34	37	44	46	50	52	56	60	65	65	67	73	73	76
Hypertension	—	+	—	—	—	—	—	+	—	—	+	—	+	—	+
Hydronephrosis	+	—	+	—	—	—	+	+	+	+	—	—	—	—	—

performed, hypertension was considered to exist if repeated blood pressure recordings showed a minimum diastolic value of 100 mm Hg. Hydronephrosis was diagnosed on the basis of radiographic, postmortem and micro-angiographic findings.

The arterial tree of the kidney was filled with a 7.5 per cent aqueous suspension of fine grain barium sulphate (Micropaque) under pressure control, after which a radiographic examination was made down to microscopic level by a procedure described elsewhere (LJUNGQVIST and LAGERGREN 1962). After fixation in 10 per cent neutral formalin the kidney was cut into slices, 2—4 mm in thickness. On the basis of radiographs of these an average of seven pieces were selected from each kidney. These pieces, each of which comprised one whole lobe with the surrounding renal columns, were embedded and cut into serial blocks 200—1500 μ thick for stereo-micro-angiography. The microradiographed blocks were re-embedded and sectioned at 5 μ for histologic examination. Blocks from nine kidneys were serially sectioned. The sections were stained by the usual methods.

Results

The findings in the present study are in agreement with those of previous authors insofar as the pyelonephritic alterations were found to vary even in a particular kidney and as such changes alternated with uninflamed regions. As in the study by STAEMMLER and DOPHEIDE (1930) the alterations are divided into types, but since these occurred concomitantly in the kidneys and transitional forms were seen, this grouping is, apart from certain types of thyroid-like tissue, schematical and is used mainly to simplify the description of the renal morphology in chronic pyelonephritis. The histologic alterations, with the exception of those displayed by the vessel walls, are described under *Histology*, and the histologic and micro-angiographic alterations of the vessels under *Vascular morphology*.

Histology

Type 1. The chief features of this type were a moderate atrophy of the cortical tubules, with indistinct staining of cells and nuclei, and a more prominent interstitial connective tissue than normally, which contained a moderate number of fibroblasts and was infiltrated by lymphocytes and plasma cells to a varying degree. The glomeruli were densely distributed. Most of them were intact,

but some showed slight periglomerular fibrosis. Streaks of lymphocytes and plasma cells extended to the papilla. In these streaks the tubules displayed atrophy, some of which contained chronic inflammatory cells.

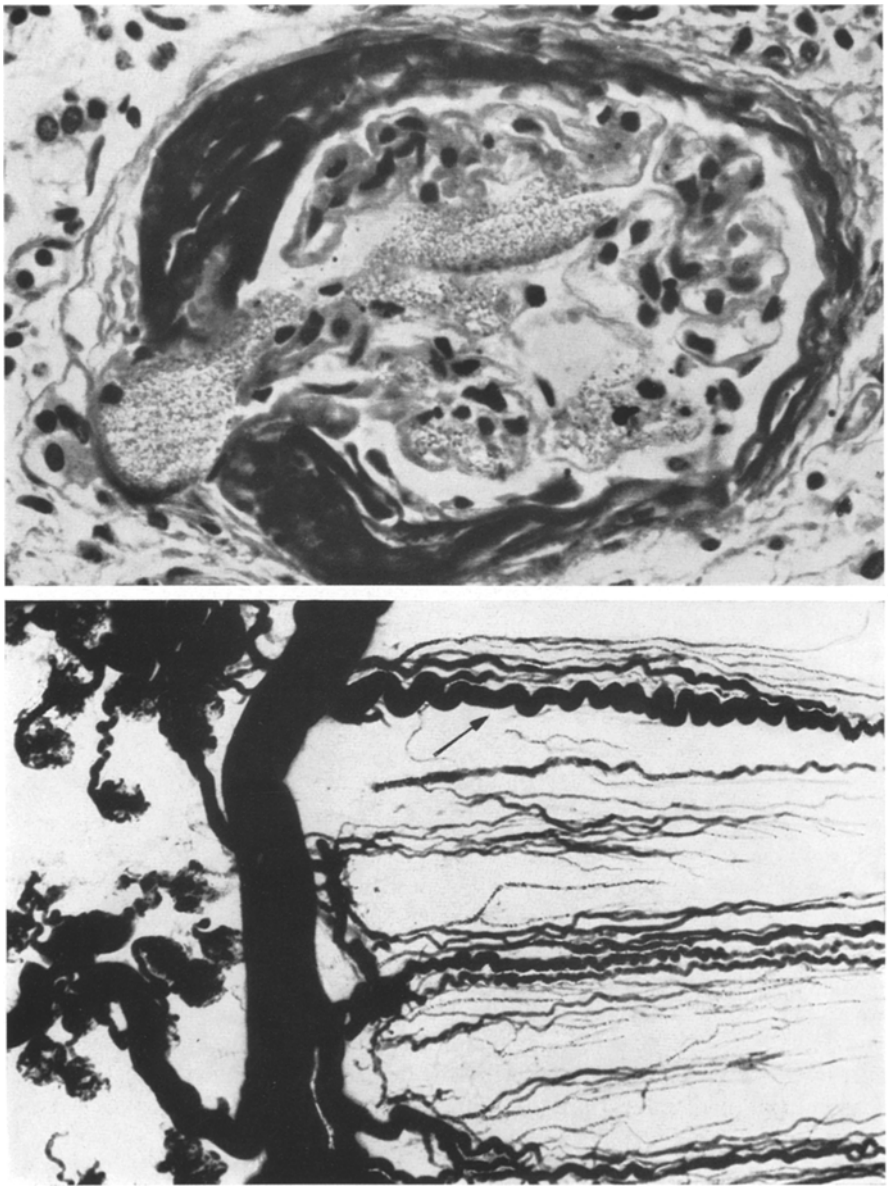

Fig. 1. a Micro-angiogram showing the juxtamedullary zone and the inner zone of the medulla. Many juxtamedullary glomeruli are devoid of efferent arterioles and the medullary vessels seen are largely of arteriolae rectae verae type. Most of them are normal but some are very wide and intensely spiralling (arrow). Case 4. ×35. b Glomerulus from the juxtamedullary zone in Fig. 1a, displaying periglomerular fibrosis with cushion-like swelling around the vascular pole. van Gieson. ×560.

Type 2. This type differed from the foregoing chiefly in respect of pronounced periglomerular fibrosis, which involved all glomeruli in the area. This fibrosis was particularly marked around the vascular pole of the glomerulus where it was seen as a cushion-like swelling (Fig. 1b). A few glomeruli were

completely fibrotic or hyalinized. Both in cortex and medulla the interstitial tissue was richer in collagen and inflammatory cells than in type 1 (Fig. 3b), and in some kidneys the cellular infiltration had the character of lymph follicles. Over the involved regions of the cortex the renal capsule displayed fibrotic thickening. In some cases deposits of mineral salts were observed in a few collecting ducts. In two kidneys (cases 4 and 10) the papillary region of some medullary pyramids contained small areas of necrosis, with few inflammatory cells and not involving the mucosa of the renal pelvis.

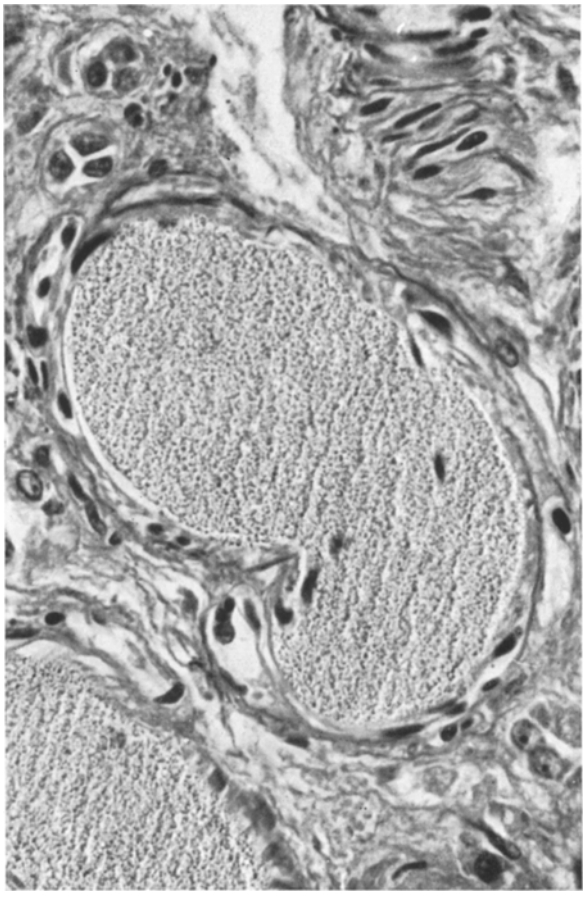

Fig. 1 c. The medullary arteriole indicated in Fig. 1a is wide with muscular hypertrophy of the wall. Van Gieson. ×560

Type 3. Regions displaying this type of change were characterized by practically total atrophy of the cortical tubules, which were replaced by a dense connective tissue. This was infiltrated by moderate numbers of lymphocytes and plasma cells. The glomeruli in these areas were as a rule totally degenerated, but some contained open capillaries and showed advanced periglomerular fibrosis. The renal capsule over the corresponding region usually displayed marked fibrotic thickening. The interstitial fibrosis and cellular infiltration extended into the medulla where the tubules were atrophic. A few collecting ducts contained mineral salt deposits.

Type 4. In contrast to type 3, extensive glomerular degeneration was combined with closely distributed, small dilated tubules with flat epithelium, which were filled with a colloid-like substance. In addition to the glomerular scars, the interstitial tissue of these thyroid-like areas displayed moderate fibrosis with local infiltration by chronic inflammatory cells. The thyroid-like change extended into the juxtacortical segment of the medulla. Towards the pelvis the medulla showed prominent interstitial fibrosis with some hyalinization and infiltration by lymphocytes and plasma cells. The renal capsule covering the thyroid-like cortical tissue was fibrotically thickened.

Type 5. As in type 4, the renal tissue had a thyroid-like appearance but the colloid-filled tubules were as a rule considerably larger, and the interstitial tissue sparser with glomeruli in different stages of degeneration. How-

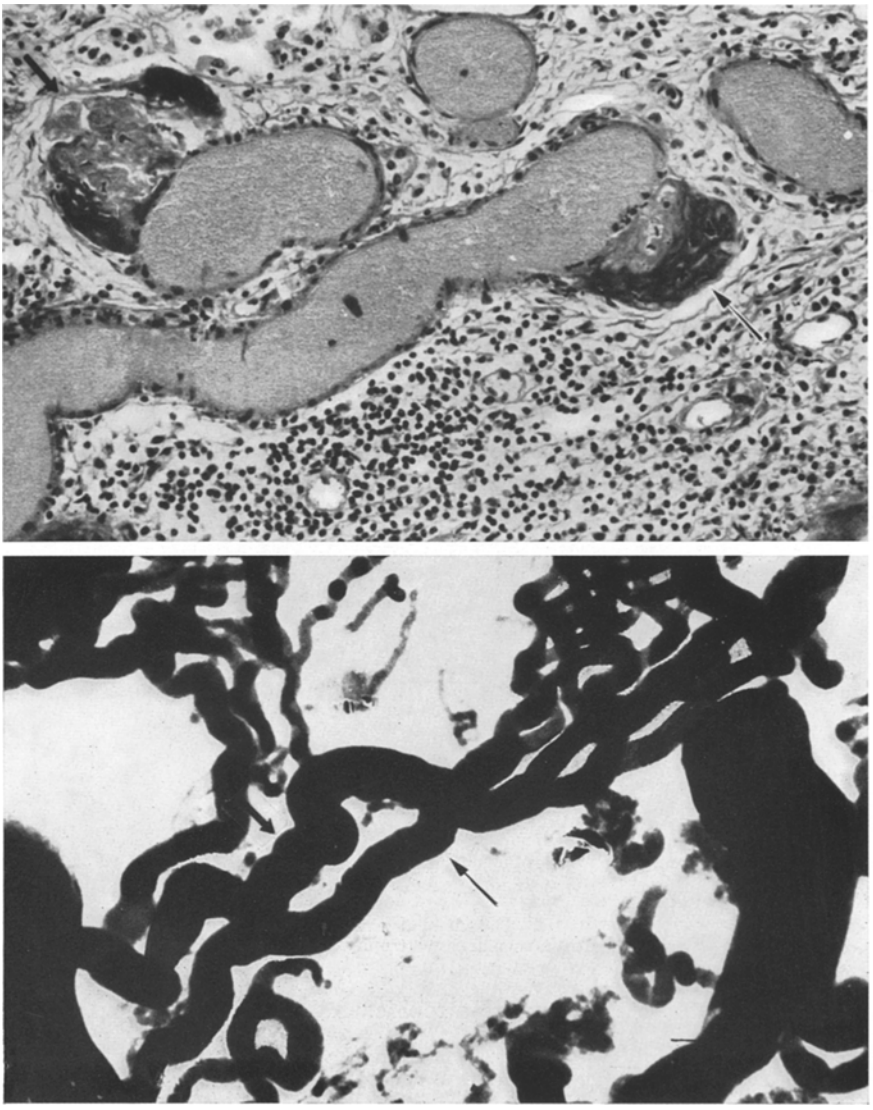

Fig. 2. a Micro-angiogram from the juxtamedullary zone of a normotensive kidney. Two aglomerular arterioles (arrows) split up into arteriolae rectae verae. Case 10. ×88. b The aglomerular arterioles indicated in Fig. 2a pass tangentially to their degenerated glomeruli (arrows). Chronic inflammatory cells are numerous and there is tubular atrophy. The vessel walls are normal. Van Gieson. ×225

ever, even completely degenerated glomeruli had an open capillary loop in the vascular pole (Fig. 5b). In the medulla the dilatation of the tubules was less marked than in the cortex, and the interstitial tissue was severely fibrotic, with some hyalinization and pronounced infiltration by chronic inflammatory cells, even intratubularly. The renal capsule displayed fibrotic thickening over the altered regions.

Additional findings. In all the kidneys the wall of the renal pelvis displayed inflammation. In those where changes of types 1—3 dominated these signs

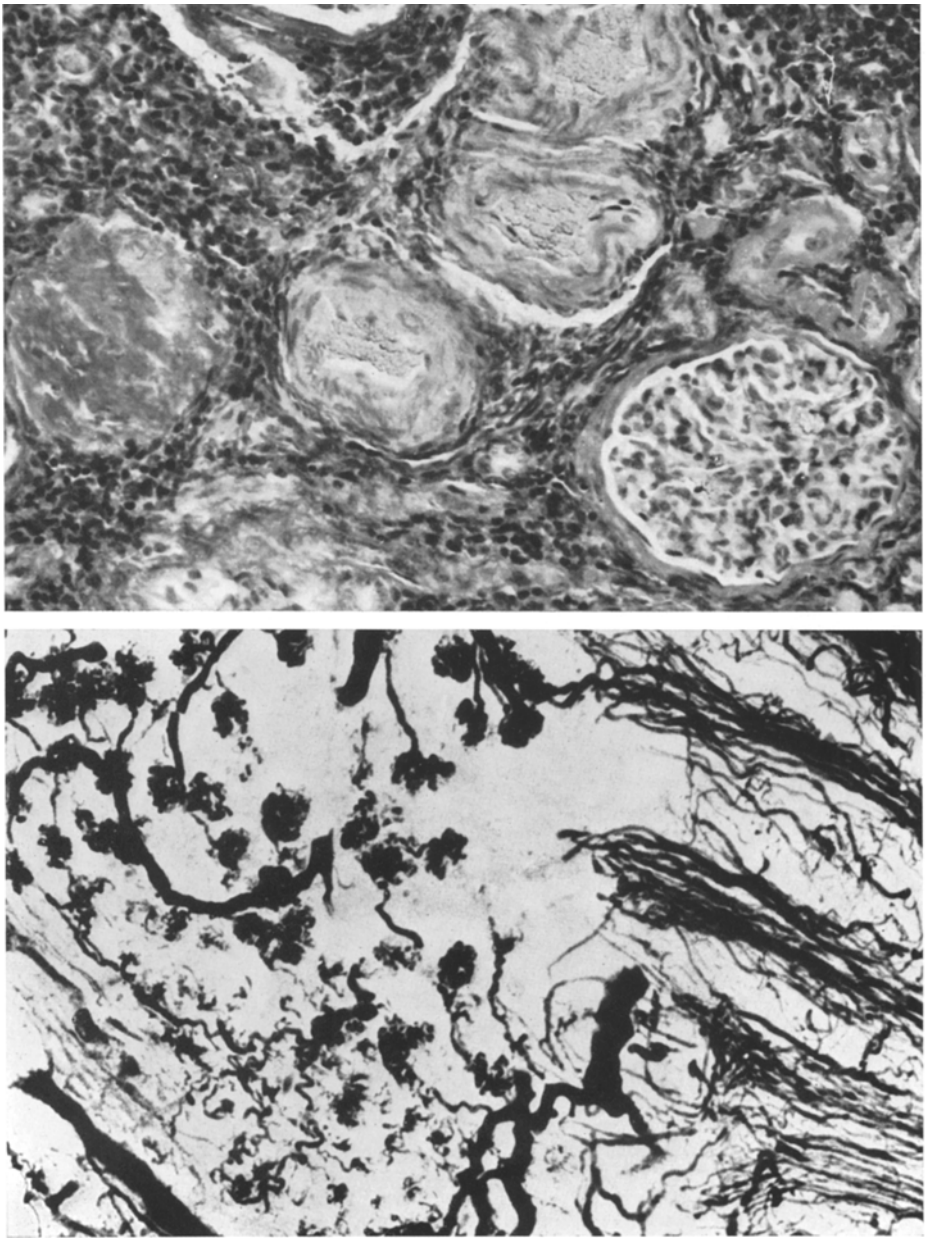

Fig. 3. a Micro-angiogram from the cortex and the juxtamedullary zone of a normotensive kidney. Above right, a few capsular vessels. Below, normal cortical pattern. Center and above, extremely thin and spiralling preglomerular vessels and small glomeruli. Case 6. ×35. b The affected part of the cortex in Fig. 3a displays a dense infiltration of chronic inflammatory cells, tubular atrophy and periglomerular fibrosis. The walls of the preglomerular vessels are greatly thickened and the lumina narrowed. Van Gieson. ×288

of pyelitis consisted in a mild subepithelial fibrosis and a more or less intense infiltration by lymphocytes and plasma cells, sometimes assuming the appearance of lymph follicles. In kidneys with extensive changes of type 4 or 5 there was

as a rule pronounced subepithelial fibrosis in the pelvis wall, and mild or moderate infiltration by inflammatory cells.

In the hydronephrotic specimens the alterations in the wall of the renal pelvis were largely as described above. The medullary changes of the respective types included, however, a larger component of fibrosis.

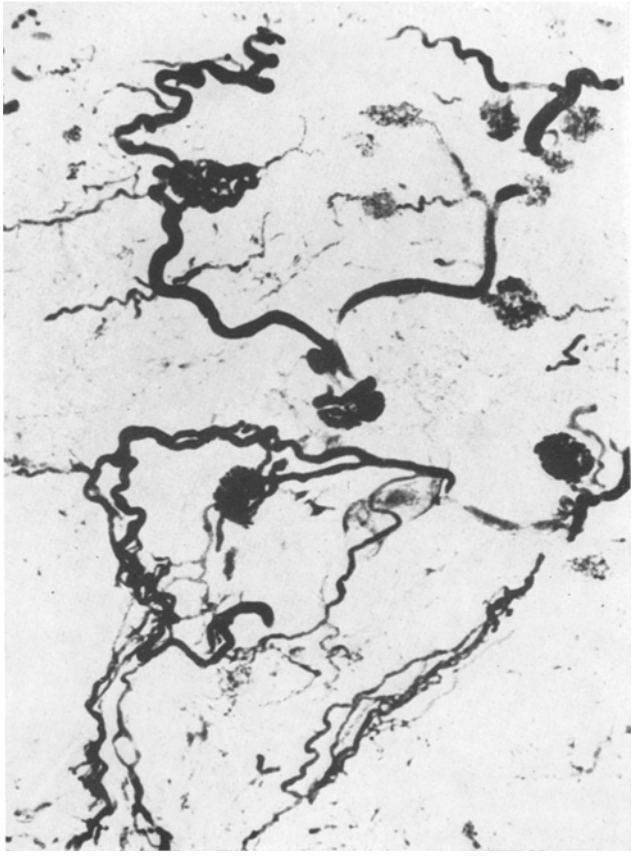

Fig. 4. Micro-angiogram from a hypertensive kidney showing narrow and slender cortical vessels and medullary vessels of the arteriolae rectae verae type. Most cortical arterioles end blindly. The cortical area has histologically a thyroid-like appearance of type 4, the vessel walls are thickened by a cellular proliferation. Case 8. ×35

Vascular morphology

Type 1. The micro-angiograms showed mostly the interlobular arteries and afferent arterioles to be of normal width, with intense spiralling, and to follow a largely normal direction. The larger of these vessels displayed mild intimal fibrosis and the smaller ones were histologically normal. In some cases the preglomerular vessels were remarkably narrow and the histologic sections revealed advanced wall changes. In the arcuate and interlobular arteries these consisted of intimal fibrosis rich in elastic fibres, and in the afferent arterioles, of alternate cellular and hyaline thickening.

Most of the glomeruli were normal in appearance, while others were small, with fine capillaries and mild periglomerular fibrosis. Some arterioles ended blindly (Fig. 4), and the histologic sections showed hyalinization of their walls and total degeneration of their glomeruli. Most of the visualized cortical glomeruli

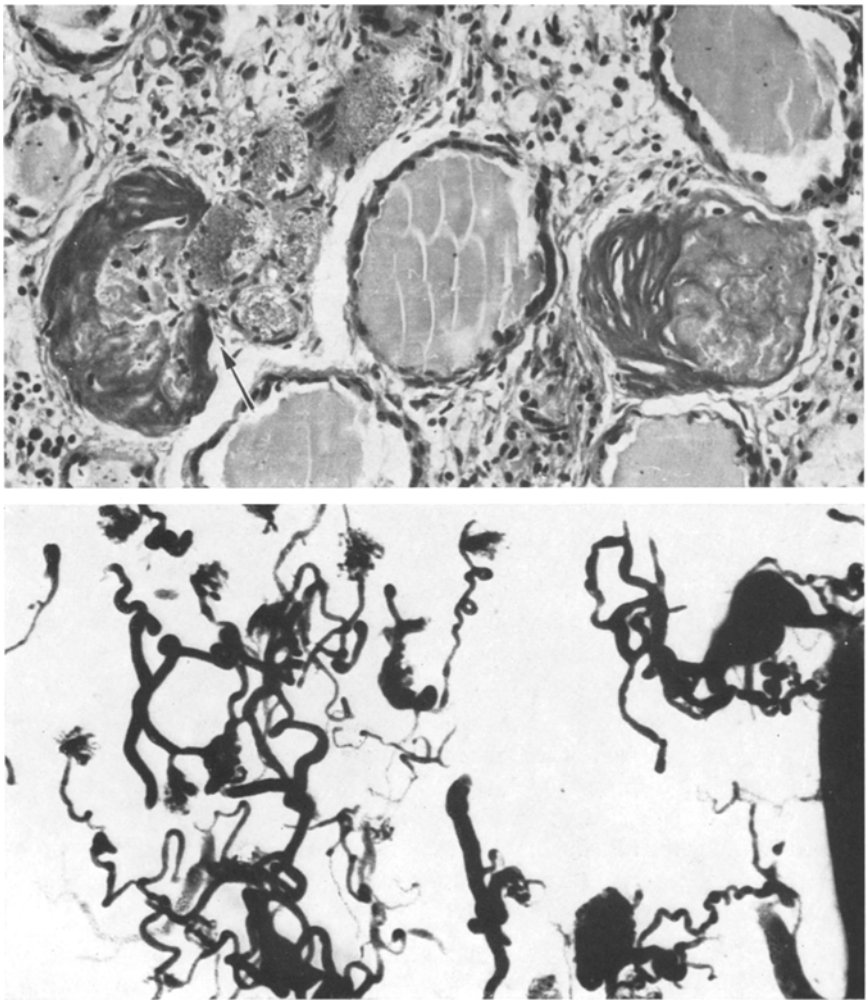

Fig. 5. a Micro-angiogram showing vessels of uniform calibre coursing in irregular loops through the cortex, thereby forming the typical vascular network of type 5. Case 13. ×35. b Degenerated cortical glomeruli from the area depicted in Fig. 5a. There is direct continuity between the afferent and efferent arterioles through the vascular pole of one of the glomeruli (arrow). The tissue has a thyroidlike appearance. Van Gieson. ×225

gave off an efferent arteriole, which was, however, abnormally narrow as were the peritubular capillaries. These post-glomerular vessels were devoid of wall changes.

The micro-angiographic pattern of the medulla was unremarkable and the distribution of arteriolae rectae spuriae and verae was normal for the respective ages. The former emerged from the efferent arterioles of juxtamedullary glomeruli, while the latter constituted ramifications of arterioles that passed through totally degenerated juxtamedullary glomeruli (Figs. 2a and b).

Type 2. In these regions the pre-glomerular vessels in some of the kidneys were of normal width and displayed only minor wall changes (Fig. 2), while in others they were extremely narrow (Fig. 3). The walls of the latter vessels were markedly thickened; the larger branches displayed cellular intimal fibrosis and the smaller ones cellular thickening of the whole cross-section, resulting in an onion-peel appearance. Whatever their calibre, the vessels displayed intense spiralling (Figs. 1a and 3a). The renal capsule covering these areas contained numerous wide and tortuous vessels, the walls of which were thin, resembling those of capillaries.

In the cortex the number of blindly ending, hyalinized vessels leading to degenerated glomeruli was larger than in type 1. Most glomeruli visualized were small and their capillaries were fine. These glomeruli displayed the typical periglomerular fibrosis, with cushion-like swelling around the vascular pole (Fig. 1b). The efferent arteriole was often not visualized, and as regards the juxtamedullary zone, this feature was particularly marked in those renal lobes the medullary pyramids of which contained areas of necrosis. In these pyramids some bundles of otherwise normal arteriolae rectae contained one or more vessels that were remarkably wide, and displayed intense spiralling and muscular hypertrophy of their walls (Figs. 1a and c).

Type 3. The cortical vessels in this type of change displayed less intense spiralling than in the foregoing ones, and they often deviated from their normal direction in irregular kinks. All were narrow, and the thickened walls resembled in appearance those of the narrow vessels of type 2. Most of the arterioles were aglomerular, the cortical ones ending blindly and the juxtamedullary ones forming thin arteriolae rectae verae. The few glomeruli visualized were small and no post-glomerular vessels were evident. Vessels of the thickened renal capsule resembling those of type 2 were observed.

Type 4. The vessels visualized in these areas displayed irregular spiralling, and, often, wide departures from their normal direction (Fig. 4). These deviations, however, were less abrupt than in type 3. All the pre-glomerular vessels were thin and the thickening of the walls was marked. In the larger branches this was due to intimal fibrosis and in the smaller ones to cellular proliferation. Most of the visualized glomeruli gave off efferent vessels, devoid of wall changes. The medulla was mainly supplied by arteriolae rectae verae, but these were rather sparse and as a rule abnormally thin.

Type 5. This type was characterized micro-angiographically by networks of vessels of uniform, arteriolar calibre, which coursed in irregular loops and gentle curves through the cortex (Fig. 5a). Occasional glomeruli either interrupted the course of these vessels or were attached to off-shoots. In the serial histologic sections many of the vessels were followed through the vascular poles of totally degenerated glomeruli (Fig. 5b). The networks thus were found to be composed of interlobular arteries, afferent and efferent arterioles and dilated loops of peritubular capillaries. The walls of the interlobular arteries were greatly thickened by intimal fibrosis, and those of the arterioles and capillaries displayed moderate hyaline thickening. As in type 4, the medulla was supplied mainly by arteriolae rectae verae, which were rather sparse and thin.

Additional findings. In the ten kidneys obtained from normotensive patients, the uninflamed areas were normal for the respective ages, whereas in the five hypertensive kidneys they displayed micro-angiographic and histologic changes resembling those in essential hypertension (LJUNGQVIST). In the inflamed areas, on the other hand, the vascular patterns were qualitatively similar in the normotensive and hypertensive kidneys.

In all the specimens the arterioles were constricted at their site of origin from the arcuate and interlobular arteries. As in the kidney of essential hypertension (LJUNGQVIST), they were most marked in areas of severe vascular change, but they were also seen in intact tissue, a feature that is normal for the kidney (MORISON 1926, DÒMINI 1959, LJUNGQVIST and LAGERGREN 1962).

In the six kidneys where there was also hydronephrosis the changes in the micro-angiographic pattern that were due to this condition were easily distinguished, for they occurred throughout the organ, even in the regions devoid of inflammation, and were characterized by the stretching of those vessels that ran parallel to the surface of the kidney, while the vessels perpendicular thereto deviated from their normal direction. This feature was particularly marked in the case of the arteriolae rectae of the medulla. These findings are in agreement with those reported in experimental hydronephrosis (DOMINI 1959).

Discussion

It is evident that the intrarenal arterial pattern in chronic pyelonephritis has certain characteristic features which distinguish it from that in normal aging and essential hypertension. Whereas the latter patterns were uniform throughout a particular kidney, the pattern of chronic pyelonephritis displayed sharply demarcated regions of altered vessels corresponding to the pyelonephritic foci. In some of the kidneys these foci were more or less normally vascularized and the vessel walls were without pathologic features. This is in contradiction to the theory presented by KINCAID-SMITH (1955) that the contraction of the kidney and the inflammation are due to ischaemia resulting from primary vascular damage. The findings are more consistent with the view of HEPTINSTALL et al. (1960) that pyelonephritic scarring is not ascribable in the main to ischaemia caused by narrowing of the arteries, but is rather a manifestation of the inflammation per se.

Of the various alterations of the renal parenchyma that may occur in chronic pyelonephritis, the thyroid-like tissue has received particular attention. This tissue is generally regarded as a late manifestation of the disease, but it has been suggested that it may not necessarily be of inflammatory origin but rather constitute a congenital malformation of the renal tubules (ASK-UPMARK 1929, FAHR 1938, VOTH 1949). The present findings suggest that the thyroid-like tissue may originate in two different ways — either as the result of pyelonephritic damage to normal renal tissue (type 4), or as the result of such damage to abnormal tissue (type 5). In the latter case the change was characterized by a higher degree of tubular dilatation and by an entirely different vascular pattern. In type 4 this pattern bore a close resemblance to that of types 1—3, whereas in type 5 it included networks in the cortex not found in the other types. These networks were formed by direct continuity between the afferent and efferent arterioles

through the vascular pole of degenerated glomeruli throughout the cortex. Aglomerular arterioles formed in this way are normally found only in the juxtamedullary zone as a result of a degeneration of its glomeruli (LJUNGQVIST and LAGERGREN 1962). Such aglomerular juxtamedullary arterioles were also noted in the present material. The occurrence of aglomerular arterioles of juxtamedullary type also at other levels of the cortex would seem to indicate that juxtamedullary tissue occurred there ectopically, and the type 5 changes may therefore be regarded as a malformation. As the continuity is maintained throughout the degeneration of the glomeruli, these may function until they are completely destroyed. This persistent functioning of large numbers of degenerating glomeruli would explain the pronounced dilatation of the tubules, which were obstructed through the parallel fibrosis of the medulla. In type 4, on the other hand, there was no structural evidence of such a continuation of the glomerular function in degeneration of the arteriole-glomerular units, and the obstructed tubules were consequently less dilated.

In areas of pyelonephritis the pre-glomerular vessels displayed considerably more intense spiralling than under normal conditions of aging (LJUNGQVIST and LAGERGREN 1962) and in essential hypertension (LJUNGQVIST). In the previous studies the spiralling has been tentatively ascribed to three factors: (i) growth of the vessels during their adaptation as collaterals to occluded vessels, (ii) shrinkage of the interstitial tissue, which shortens the distance between the ends of the vessels, and (iii) disturbed flow through the spiralled vessels which retains the spiralling and, if the intravasal pressure is high, possibly accentuates it. Many pyelonephritic regions were well supplied by interlobular arteries and arterioles having no evidence of wall changes, but the spiralling was no less marked. This suggests that the spiralling was not a feature of collateral adaptation. Since the normo- and hypertensive kidneys displayed no clear difference in the degree of spiralling of the vessels in pyelonephritic areas, it is unlikely that hypertension had any significant bearing on this change. The considerably more intense spiralling of the intrarenal arteries in chronic pyelonephritis than under normal conditions of aging and in essential hypertension is therefore probably to be attributed mainly to the more intense contraction of the interstitial tissue.

The micro-angiographic picture was characterized, moreover, by absence of post-glomerular vessels even when the glomeruli were apparently normal. Histologic examination of the micro-angiographed specimens established that this feature was not due to artefactually incomplete filling of the vessels by contrast medium. These specimens disclosed a typical periglomerular fibrosis, which was particularly marked around the vascular pole. PFEIFFER (1932) considered that this thickening could lead to obstruction of the arterioles in the pole. It would be expected that the efferent arteriole would be closed off first, since it has a lower intravasal pressure and a thinner wall than the afferent vessel. The present findings suggest that this mechanism is the main cause of the loss of the post-glomerular vessels. This might, however, also be due to direct inflammatory damage, but such a mechanism would probably be of secondary significance since in pyelonephritic tissue with no evidence of periglomerular fibrosis, as in type 1, the post-glomerular vessels were clearly visualized.

In two kidneys there were small areas of necrosis in the papillary region of some medullary pyramids. Since the medulla is unusually susceptible to inflammatory agents (FREEDMAN and BEESON 1958, GUZE 1960), a bacterial or toxic genesis of the necrosis cannot be ruled out. However, the inflammatory reaction in the necrotic areas was very mild and there were definite signs of severely disturbed blood supply to these areas, which indicated that ischaemia played an essential role in the formation of the necrosis. Thus, the renal lobes involved displayed periglomerular fibrosis of the juxtamedullary glomeruli, a large number of which had no post-glomerular vessels. Of the arteriolae rectae visualized some were remarkably wide with hypertrophic walls and intense spiralling. Since such vessels occurred in bundles of normal arteriolae rectae, the spiralling could not be due to shrinkage of the surrounding tissue. The vascular change is, however, identical with that observed by WEYRAUCH and DE GARIS (1937) in areas of vascular occlusion where unoccluded vessels undergo collateral adaptation. It is probable that the formation of collateral vessels in the medulla was due to the considerable loss of post-glomerular juxtamedullary vessels and that the necrosis was a manifestation of the inadequacy of this adaptation. That necrosis occurred only in the medulla in spite of a loss of post-glomerular vessels also in the cortex seems to indicate that a reduced blood supply is more deleterious to the medulla than to the cortex. This is consistent with the findings by PAPPENHEIMER and KINTER (1956) and LILLIENFIELD et al. (1958) that the haematocrit value of the papillary region is considerably less than that of the cortex. In parallel micro-angiographic and histologic studies of renal papillary necrosis in cases of pyelonephritis, evidence of collateral vascular adaptation was noted in pyramids having necrotic papillae (LAGERGREN and LJUNGQVIST). It is probable that also in this condition local medullary ischaemia resulting from loss of post-glomerular juxtamedullary vessels plays an essential rôle in the formation of the necrosis. The medullary necrosis observed in the present material perhaps represents an early stage of renal papillary necrosis, beginning centrally in the papillary region.

It seems to be generally accepted that renal hypertension is caused by cortical ischaemia. From the aspect of vascular morphology, this can be due to vascular changes that will either reduce the cortical, and/or improve the medullary circulation. It has been shown that in advanced stages of essential hypertension there is an anatomic basis for both these mechanisms, namely a narrowing of the cortical vessels and an increase in arteriolae rectae verae (LJUNGQVIST 1962). Whether these vascular changes are, in fact, a cause or an effect of the hypertension is obscure. Largely similar vascular features were observed in the pyelonephritic regions in the kidneys of the present material. Since pyelonephritis precedes the hypertension and probably gives rise to the vascular changes occurring in this condition it is conceivable that these are responsible for the hypertension. Whether or not cortical areas affected by pyelonephritis will be ischaemic, with consequent development of hypertension, will depend on the degree of impairment of the cortical vessels, and the extent to which juxtamedullary arteriole-glomerular units in the area are converted arteriolae rectae verae. The severity of the hypertension would then depend on the balance between cortical and medullary circulation in the areas affected by pyelonephritis

and the amount of cortical tissue thereby rendered ischaemic, and not solely on the degree of thickening of the walls of the cortical vessels as WEISS and PARKER (1939) and KINCAID-SMITH (1955) supposed. This argument is supported by the fact that in the present material, as in ones previously reported (BROD 1957, KLEEMAN et al. 1960), there were cases with severe changes in the vessel walls and no hypertension, and also cases with hypertension where the wall changes were relatively mild.

Summary

Comparative micro-angiographic and histologic studies have been performed on 15 kidneys with chronic pyelonephritis.

The vascular changes included intense spiralling of the pre-glomerular cortical vessels and loss of the post-glomerular ones. The spiralling was due mainly to the damage to and consequent contraction of the interstitial tissue, the loss of post-glomerular vessels to their occlusion at the vascular pole of the glomeruli by periglomerular fibrosis. Two kidneys displayed medullary necrosis, which was ascribed to the loss of post-glomerular juxtamedullary vessels with consequent ischeamia of the medulla.

The walls of the pre-glomerular cortical vessels were often greatly thickened and the lumina narrowed. These changes were not correlated to hypertension. In the pyelonephritic areas some juxtamedullary arteriole-glomerular units were converted to aglomerular arterioles forming arteriolae rectae verae. Whether or not cortical ischaemia will appear in a pyelonephritic area, with consequent development of hypertension will depend on the balance between the cortical circulation, hampered by the wall changes, and the medullary circulation, enhanced by the formation of arteriolae rectae verae.

Zusammenfassung

Vergleichende mikro-angiographische und histologische Untersuchungen wurden an 15 menschlichen Nieren mit chronischer Pyelonephritis durchgeführt. Gemeinsam ist allen pyelonephritischen Veränderungen eine intensive Spiralisierung der präglomerulären Rindengefäße und ein Verlust der postglomerulären Rindengefäße: Die Spiralisierung geht hauptsächlich zurück auf eine Schädigung mit nachfolgender Schrumpfung des interstitiellen Gewebes, der Verlust der postglomerulären Gefäße auf einen Verschluß am Gefäßpol der Glomeruli durch periglomeruläre Fibrose. In zwei Nieren fanden sich kleine Marknekrosen, die auf diesen Verlust der postglomerulären Gefäße mit nachfolgender Ischämie des Markes zurückgeführt wird.

Die Wand der präglomerulären Rindengefäße ist oft stark verdickt, die Lichtung eingeengt. Diese Veränderungen zeigten keine Beziehung zur Hypertension. In den pyelonephritisch veränderten Gebieten waren einzelne juxtamedulläre Arteriolen mit zugehörigen Glomeruli zu aglomerulären Arteriolen umgewandelt, die dann in dem Mark in Arteriolae rectae verae übergehen. Ob eine Ischämie der Rinde und damit Hypertension sich einstellt oder nicht, wird abhängen von dem Verhältnis zwischen der durch die Gefäßwandveränderungen beeinträchtigten Zirkulation der Rinde und der durch die Bildung der Arteriolae rectae verae verstärkten Zirkulation im Mark.

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