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Vascular alterations in the canine kidney following obstruction of the urinary tract

A SEM investigation of corrosion casts

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Summary. Changes in the vasculature of the canine kidney following four weeks obstruction of the ureter via double ligature is described on the basis of SEM investigation. Three significant alterations were observed: 1) A two-thirds reduction in the depth of the renal cortex as compared to controls. 2) Rarification of the entire cortical capillary bed. 3) Reduction in both the number and diameter of the glomeruli. The rarification of the post-glomerular capillaries is interpreted to be a pressure atrophy, whereas the reduction in the number of renal glomeruli and the concommitant diminuation of their capillary loops is thought to represent a functional atrophy.

Key words: Ureter obstruction – Canine kidney – Blood vessel architecture – SEM – Corrosion casts

Introduction

Total blockage of the urinary tract is known to lead to an irreversible loss of function in the affected kidney once a certain period of time has elapsed. Earlier studies on dogs [7, 8] demonstrated a nearly complete restoration of renal function following removal of the unilateral blockade and concommitant nephrectomy of the contra-lateral kidney as long as the obstruction was of less than two week's duration. Function was restored only partially if the obstruction continued over a longer period of time (three weeks) and corresponded to about fifty per cent of the normal value. Four weeks of total obstruction resulted in irreversible damage and permanent loss of function.

The goal of the present study was to utilize the scanning electron microscope (SEM) for the investigation of changes in the vascular architecture and microvascularization of the canine kidney following four weeks of total blockage to the ureter.

Materials and methods

A total of five beagle kidneys were utilized for the purposes of this investigation. Obstruction was achieved via double ligation of one of the ureters on each of two dogs and maintained over a period of four weeks. In addition to these, the kidneys of two control animals were available, as were the contra-lateral kidneys of the obstructed dogs. Corrosion casts were prepared immediately after death via injection of low viscosity Tardoplast® resin into the renal artery. The infusion was continued until pure polymer continued to flow from the renal vein. Then the kidneys were divided by sagittal section. Additional sagittal and transverse cuts served to reduce one of each of the halves into numerous smaller sections (edge length ca. 1–2 cm), as previously described [1, 2]. Once the injection material was completely hardened, the sections were macerated in a series of 5, 10, 15 and 20% potassium hydroxide, rinsed with copious amounts of distilled water and allowed to dry in air.

To examine the alterations of the size and number of glomeruli in obstructed kidneys, the diameter of glomeruli from different parts of the cortex were measured and compared with controls ($N = 100$). The number of glomeruli were counted per fields of 1,00 mm^2 ($N = 25$ each) on sections of corrosions casts. Evaluations were carried out on a Philips 500 scanning electron microscope. Statistical evaluations included standard errors and analyses of variance and were compiled with a Macintosh Plus® microcomputer and the statview 512$^+$ (Brainpower Inc., Calabasas, California) statistical package. The second kidney half was examined in toto with a Stereomagnifier.

Results

An extensive restructuring of the vascular bed was evident in the area of the renal cortex following four weeks of total obstruction. The reduction in depth of the renal cortex to roughly one-third of that seen in comparable controls was particularly striking. Simultaneous changes in the vascular architecture were also apparent in this area. Sections of the corrosion casts revealed that the interlobular arteries of the obstructed kidneys were no longer organized in an orderly fashion, as was the case for untreated kidneys. One practical effect of the reduction in the depth of the renal cortex was an increase in the number of glomeruli per unit of surface area (Fig. 5), but a significant reduction

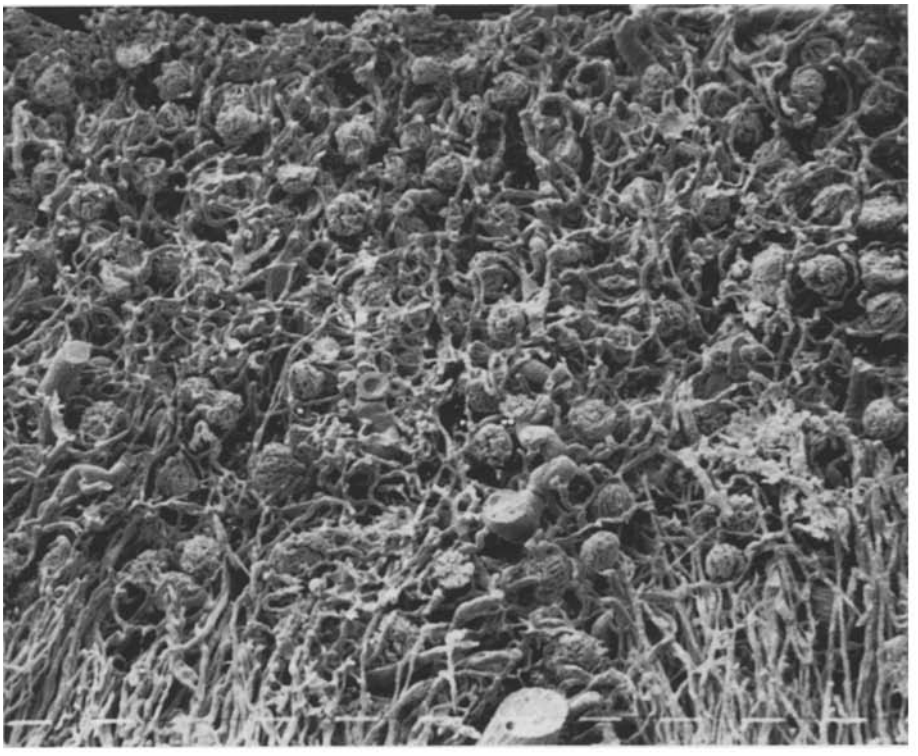

Fig. 1. SEM overview of a kidney, the ureter of which had been totally obstructed for a period of four weeks. The depth of the cortex is only about one-third that of the controls. Scale length = 100 μm

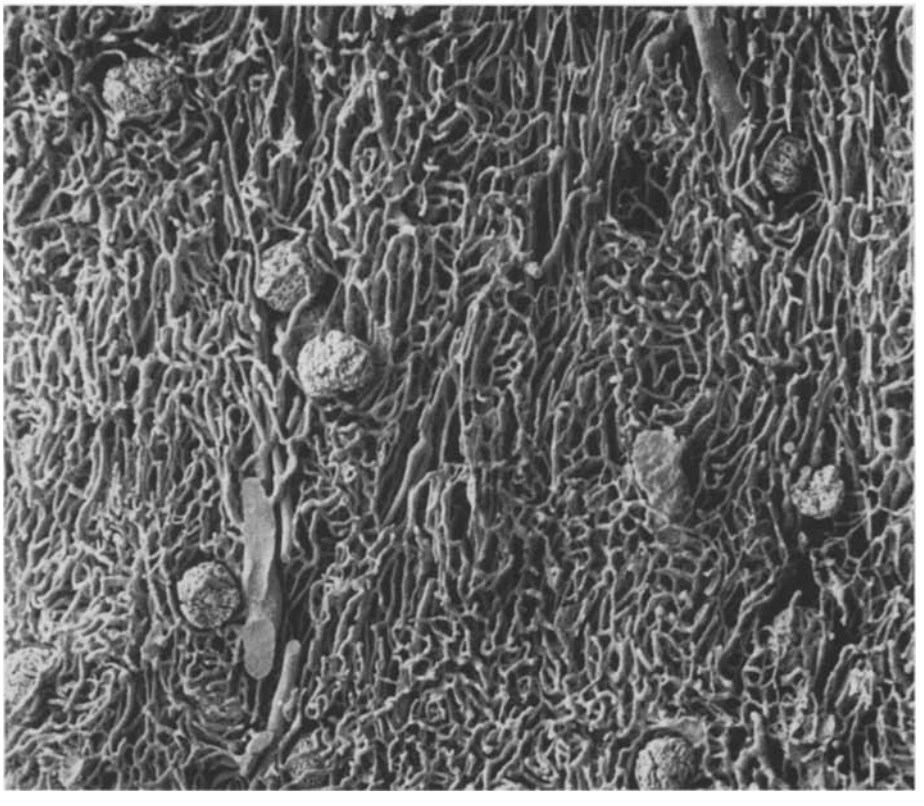

Fig. 2. SEM overview of the cortex of a control kidney, which clearly demonstrates the overwhelming capillarisation in this region. Scale length = 100 μm

in the absolute number of glomeruli was also evident when the shrinkage in size of the whole kidney was taken in account.

In addition to the vascular loops of the glomeruli, capillary arrangement and density were also noticeably affected by the restructuring process. The capillarisa-

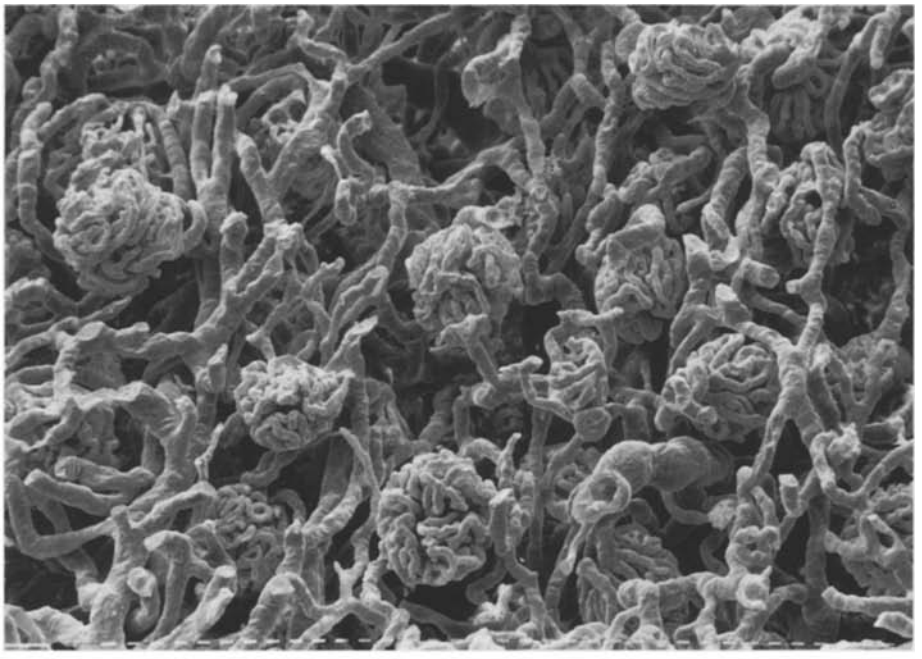

Fig. 3. Glomerula form a kidney, the ureter of which has been totally obstructed for a period of four weeks. Scale length = 10 μm

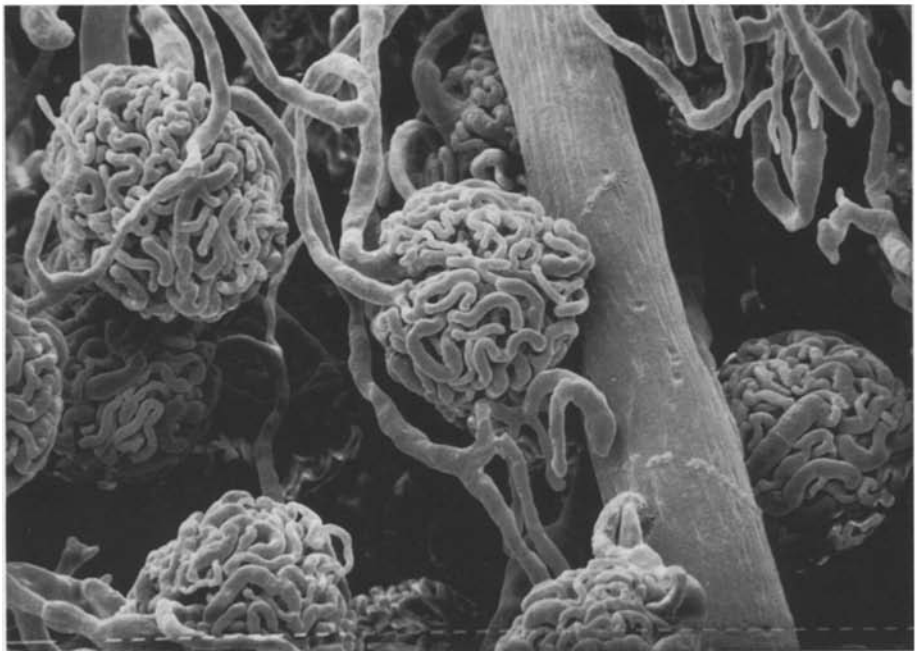

Fig. 4. Glomerula from a control kidney. Scale length = 10 μm

tion of the nephrons was reduced to minimum, so that it was no longer possible to differentiate between the capillaries of the cortical labyrinth and those of the medullary rays, as was possible in control kidneys (Fig. 1 and 2). The entire post-glomerular capillary bed appeared to be rarified and disordered. The basic construction of the vascular loops was retained, despite the considerate restructuring processes described for the vascular architecture. The number of vascular loops was significantly reduced, however, and the mean diameter of the glomeruli has decreased from ca. 190 μm to 100–120 μm (Fig. 6). The density of the glomerular capillary loops was likewise diminished (Fig. 3), and the socalled "secondary branches", which appeared regularly in control kidneys (Fig. 4), became sparse.

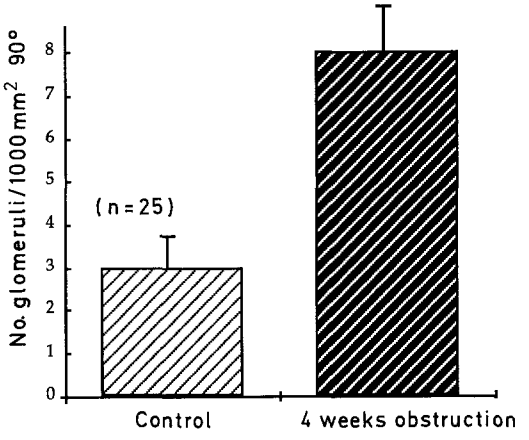

Fig. 5. Number of Glomeruli/1000 mm^2 ($N = 25$)

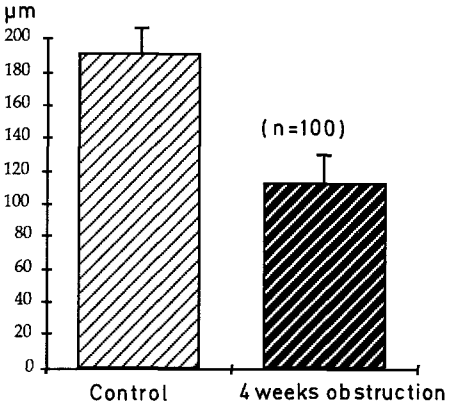

Fig. 6. Diameter of Glomeruli in µm ($N = 100$)

Discussion

Substantial restructuring processes occured in the vascular system of kidneys, whose ureters were totally obstructed for a period of four weeks. Three characteristic changes were observed:

1) A two-thirds reduction in the depth of the renal cortex as compared to controls

2) Rarifiction of the entire cortical capillary bed

3) Reduction in both the number and diameter of the glomeruli

The chronic fibrosis observed in the interstitium of the renal cortex as a consequence of obstructive nephropathies [4], would suggest that the significant reduction in the number of post-glomerular capillaries seen in the present study was the result of the pressure atrophy which was created [4], i.e., the destruction of the capillaries in the crowded interstitium [9]. The simultaneous decrease in the number of renal glomeruli and the concommitant reduction of their capillary loops, however, was more likely to be a functional atrophy. In 1986, Gillenwater investigated the influence of obstructing the renal blood vessels, and noticed the initial response to be an increased renal blood flow, which continued for approximately one or two hours [6]. The blood flow was decreased after this period of time, which was caused by an increase in post-glomerular resistance, as a result of the blocked ureter. A further reduction in renal blood flow and diminished pressure within the ureter are characteristic of the final, chronic phase. This is assumed to be the result of a preglomerular vasoconstriction, which is unreceptive to the influence of pharmaceuticals. Such vasoconstriction even appears in response to the obstruction of a single nephron [3]. We interpret the shrinkage of the glomeruli and the corresponding diminuation of their capillary bed to be a functional atrophy, which in turn might be the result of chronically reduced perfusion. This is in agreement with, the results of Weinberger et al. [10], who also demonstrated a reduction in the diameter of the renal glomeruli following the administration of elevated doses of angiotensin II.

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