Renal changes in the diabetic kidney

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Diabetic nephropathy, i.e. albuminuria and decreasing glomerular filtration rate—often associated with increased blood pressure—is closely associated with structural lesions in the kidney. In advanced diabetic nephropathy all compartments of the kidney show abnormalities. The glomerular changes are rather characteristic. By light-microscopy the most conspicuous change of diabetic glomerulopathy is expansion of the mesangial regions (Figure 1). Electron-microscopy shows that the key abnormality is increased amounts of extracellular material: Thickening of the basement membrane in the capillary walls (Figure 3,p) and expansion of the mesangial matrix. The individual matrix profiles are enlarged and convex (Figures 2, 3*). Capsular drops (Figure 4,d): oblong, prominent thickening of the basement membrane of Bowman’s capsule are frequently observed. In advanced stages glomerular obsolescence may be a dominant feature. Concurrently with the glomerulopathy changes are present in the juxtaglomerular arterioles, in the afferent (a) as well efferent (e) vessels (Figure 1). The pattern of the arteriolar hyalinization is patchy: Some profiles may be maximally affected while neighbouring ones show a practically normal appearance (Figure 4, three profiles of afferent arterioles marked *). Electron-microscopy shows accumulation of extracellular matrix, sometimes with a more electron-dense appearance than that of the normal matrix (Figure 5). In some segments of arterioles the smooth muscle cells (Figure 5, m) constitute only a small part of the vessel wall. Tubular degeneration may be apparent in advanced stages of renal lesions, probably as a consequence of glomerular occlusion and/or local vascular insufficiency, which may also cause widening of the interstitium. However, a diffuse widening of the interstitial space is demonstrable also in the earliest stage of nephropathy, clinically showing as microalbuminuria (Figure 6).

The interplay among the structural lesions in the various compartments are determinant for the course of renal disease in the diabetic patient.

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