Pigmentation of Renal Cortical Tubules in Horses

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Diffuse pigmentary nephrosis is relatively frequent in cattle (so-called fuscin nephrosis) [3] and has been seen in goats (so-called “cloisonné kidney”) [1, 2]. The equine case we describe (adult male horse) is apparently without antecedent in the literature. After slaughtering, the internal organs appeared normal except that the kidneys had a black pigmentation of the cortex (fig. 1). About two hours postmortem, the kidneys were sent to our laboratory for optical and electron microscopy.

Histologically, the convoluted portion of the proximal tubules had a granular brown pigmentation of the epithelium and an abnormal continuous thickening of the basement membrane (fig. 2) which appeared brown in the unstained sections as well.

Histochromically, methods [4] for the identification of organic pigments showed both pigmented structures (tubular cells and the surrounding basement membranes) to be negative for melanin. The thickened basement membranes reacted positively for lipofuscin, and the granules of the epithelium for lipofuscin containing iron (liposiderin) (fig. 3).

Electron microscopy (fig. 4) showed the thickened and pigmented membrane to be constituted, from the inside to the outside, of a moderately thickened basement membrane of the tubule and a superimposed stratum of irregular thickness (1–6 μm) formed by moderately electron-dense granules having a diameter of 50 to 200 nm.

This external layer contained residues of cytoplasmic degenerating structures (fibroblasts?) from which it probably originated. The ultrastructure of the granules corresponded neither to melanin nor to hemosiderin, but rather to the granules present in the residual bodies of cells (fig. 5). Therefore, the above described pigment appears ultrastructurally analogous to lipofuscin.

Although the unusual pigmented thickening of the basement membrane morphologically resembles that of the so-called “cloisonné kidney” of goats, the absence of ferritin in the basement membrane excludes the pathogenetic interpretation given in those cases; that is, deposition of pigment on basement membranes as a result of tubular hemosiderosis due to hemolysis [1, 5]. Nevertheless, the tubular epithelial cells and basement membrane changes in this horse seem to be related, and the distinct pigmentation of the kidney corresponds to histochemical and ultrastructural findings of lipofuscinosis.

References


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Fig. 1: Cut surface of kidney. Dark pigmentation of cortex.
Fig. 2: Thick basement membrane of tubules. Periodic acid–Schiff.
Fig. 3: Thickened and pigmented basement membranes. Hueck’s method for lipofuscin.
Fig. 4: Pigmented thickened basement membranes containing deposits of small particles. Cell debris in an indentation of basement membrane (arrow).
Fig. 5: Residual body of cellular origin (top), thickened basement membrane (tbm) with granular material, and part of cytoplasm of tubular cell (double arrow).