Mini-Lecture 11

Exploring the Renal Vasculature-Black Box of Kidney Disease

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The renal vasculature has unique structure and function. Each kidney receives a branch from the abdominal aorta, the renal artery. The renal artery gives rise to interlobar arteries which enter the renal parenchyme at the corticomedullary junction. The interlobar arteries undergo further divisions into the arcuate artery. Small laboratory animals such as mice, rats and rabbits have a unilobar kidney and the renal artery gives rise to the arcuate artery. The arcuate artery yields the interlobular arteries which ascend through the cortex, and then give rise to tiny glomerular branches, the afferent arterioles. The afferent arterioles divide into the glomerular capillaries and rejoin to form the efferent arterioles instead of a vein, creating an arterial portal system. While the efferent arterioles of superficial glomeruli drain into the peritubular capillary network, the efferent arterioles of juxtamedullary medullary glomeruli become long, straight capillaries, the vasa recta that extend deep into the medulla.

One of the unique functions of renal vasculature is the glomerular filtration. The glomerular capillaries contain numerous pores that are highly permeable to water and small solutes but much less permeable to albumin-size and larger proteins. Unlike most fenestrated capillary endothelial cells, adult glomerular endothelial cells are generally thought to lack diaphragms at their fenestrae. A recent, careful study demonstrated that only 2% of glomerular capillary cross sections from mature rat glomeruli contain diaphragmed fenestrations. However, during normal development and under some pathological conditions, most glomerular endothelial cells form diaphragms. Therefore, the reappearance of diaphragmed fenestrae may serve as an important and novel marker of glomerular disease with proteinuria.

Another unique function of renal vasculature is to regulate urine concentration. The two limbs of vasa recta play a critical role in promoting countercurrent exchange of solutes and water. The descending vasa recta have a continuous endothelium with tight junctions, whereas the ascending vasa recta have a highly fenestrated endothelium. Specific transporters for water (AQP1) and urea (UT-B) are expressed in the descending vasa recta but not in the ascending vasa recta. The descending vasa recta wall is also characterized by smooth muscle-like cells, pericytes. When the descending vasa recta are isolated and examined in vitro, the vessels respond to numerous vasoconstrictors and vasodilators. Some evidence suggests that alterations in vasa recta endothelial cells may contribute to the development of hypertension as well as defect in urine concentration.

The structural changes of renal vasculature can be seen in many conditions including diabetes and hypertension. Although some investigators believe that the abnormalities are nonspecific and entirely secondary, novel alterations in renal microvessels, such as glomerular capillaries and vasa recta, may contribute to detecting kidney diseases and understanding the pathophysiology.

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