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Vascular Disorders of the Kidney

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INTRODUCTION

The spectrum of vascular disorders of the kidneys is broad as a result of high renal blood flow and the intimate relationship between blood supply and fundamental glomerular and tubular functions. In this chapter, emphasis is placed on the clinical manifestations of hypertension, chronic kidney disease (CKD), end-stage kidney failure, and the many other causes of acute kidney injury (AKI).

RENAL VASCULAR ANATOMY

The renal arteries arise directly from the aorta and enter the renal hilum. The right renal artery passes anterior to the inferior vena cava (IVC) and is longer than the left renal artery. In up to 30% of the population, accessory renal arteries arise from the aorta to provide blood to portions of one or both kidneys, which may become important when evaluating patients for renovascular hypertension.

The renal arteries give rise to segmental, interlobar, and arcuate arteries (Fig. 30-1). Arcuate arteries course along the corticomedullary junction and give rise to interlobular arterioles, which extend outward into the cortex before branching into afferent arterioles, from which the glomerular capillary tufts arise. The postglomerular efferent arterioles from more superficial glomeruli form a capillary network in the renal cortex, and those extending from glomeruli nearer the cortical-medullary junction (i.e., juxtamedullary glomeruli) form capillaries that extend deeper into the medulla in association with thin, descending and ascending loops of Henle as the vasa recta. The vasa recta provide the sole blood supply for the renal medulla, making this portion of the kidney particularly susceptible to ischemic injury. Venules from the ascending vasa recta and the cortical capillary network empty into the renal veins.

The left renal vein returns to the IVC anterior to the aorta and inferior to the inferior mesenteric artery, which may rarely cause compression of this vein. The left gonadal vein also empties into the left renal vein, and a left varicocele may be evident if the renal vein is occluded by thrombosis or tumor involvement. The right renal vein is much shorter and empties directly into the IVC. The right gonadal vein empties directly into the IVC rather than into the right renal vein.

RENOVASCULAR DISEASE

Any process that narrows the lumen of the main or branch renal arteries sufficiently can elicit a humoral response mediated by increased renin release from the ipsilateral kidney, which leads to increases in circulating angiotensin II and aldosterone levels. Activation of the renin-angiotensin-aldosterone system increases systemic blood pressure and renal arterial perfusion pressure, renal blood flow, and the glomerular filtration rate (GFR) beyond the stenosis. Hemodynamically significant renal artery stenosis (RAS) requires a reduction in lumen diameter of at least 50% to 60%. The resulting renin-angiotensin-aldosterone system activation leads initially to systemic vasoconstriction mediated by angiotensin II and increased renal sodium and fluid reabsorption, resulting in elevation of systemic blood pressure. If the contralateral kidney has no stenosis, the increased systemic blood pressure increases sodium excretion by that kidney (i.e., pressure natriuresis).

Because hypertension in this setting is maintained by increased vasoconstriction due to angiotensin II, treatment is aimed at blocking the synthesis or effect of the elevated angiotensin II levels with an angiotensin-converting enzyme (ACE) inhibitor

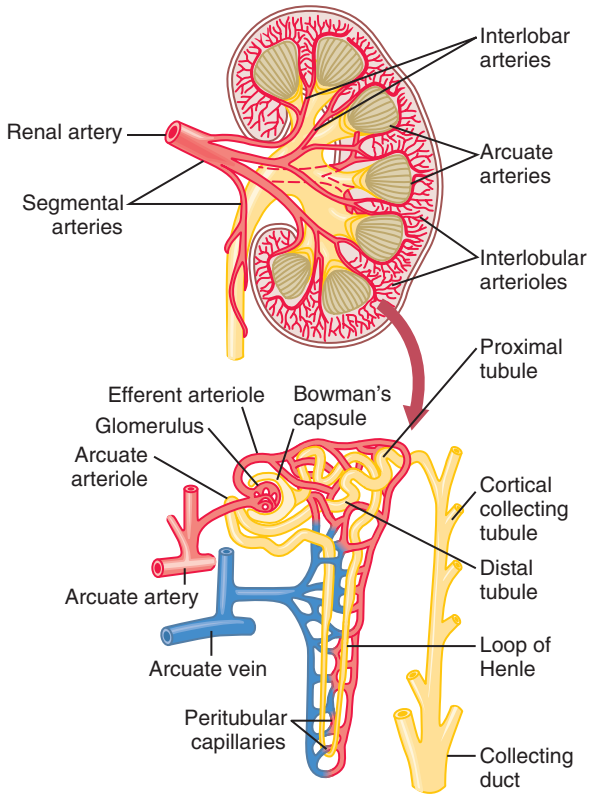

FIGURE 30-1 Section of the human kidney shows the renal arteries and a schematic of the microcirculation of each nephron. (From Guyton AC, Hall JE: Textbook of medical physiology, ed 11, Philadelphia, 2006, Saunders, p 309.)