Renal Arterial Disease and Hypertension

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KEYWORDS

• Renovascular • Renal artery stenosis • Hypertension • Angiotensin • Kidney
• Ischemic nephropathy

KEY POINTS

- Renal artery disease produces a spectrum of progressive clinical manifestations ranging from minor degrees of hypertension to circulatory congestion and kidney failure.
- Moderate reductions in renal blood flow do not induce tissue hypoxia or damage, making medical therapy for renovascular hypertension feasible for many patients.
- Several prospective trials indicate that optimized medical therapy using agents that block the renin-angiotensin system should be the initial management.
- Evidence of progressive disease and/or treatment failure should allow recognition of high-risk subsets that benefit from renal revascularization.
- Severe reductions in kidney blood flow ultimately activate inflammatory pathways that do not reverse with restoring blood flow alone.

Renovascular hypertension has been recognized for more than 80 years, since seminal experimental studies showed that progressive occlusion of the renal vessels produces an increase in systemic arterial pressure. These data established a central role of the kidney in blood pressure regulation and provided one of the most widely studied models of angiotensin-dependent hypertension.[1] This condition can occur at levels of renal pressure greater than those that impair kidney function, although progressive reduction in renal blood flow leads to additional disturbances, including impaired volume control, circulatory congestion, and ultimately irreversible kidney injury. Hence, occlusive renovascular disease (RVD) comprises a spectrum of disorders ranging from incidental, minor disease to incipient occlusion with tissue ischemia, as shown in **Fig. 1**.

EPIDEMIOLOGY

The dominant cause (at least 85%) of RVD in Western countries is atherosclerotic renal artery stenosis (ARAS). This condition often develops as part of systemic atherosclerotic disease affecting multiple vascular beds, including coronary, cerebral, and

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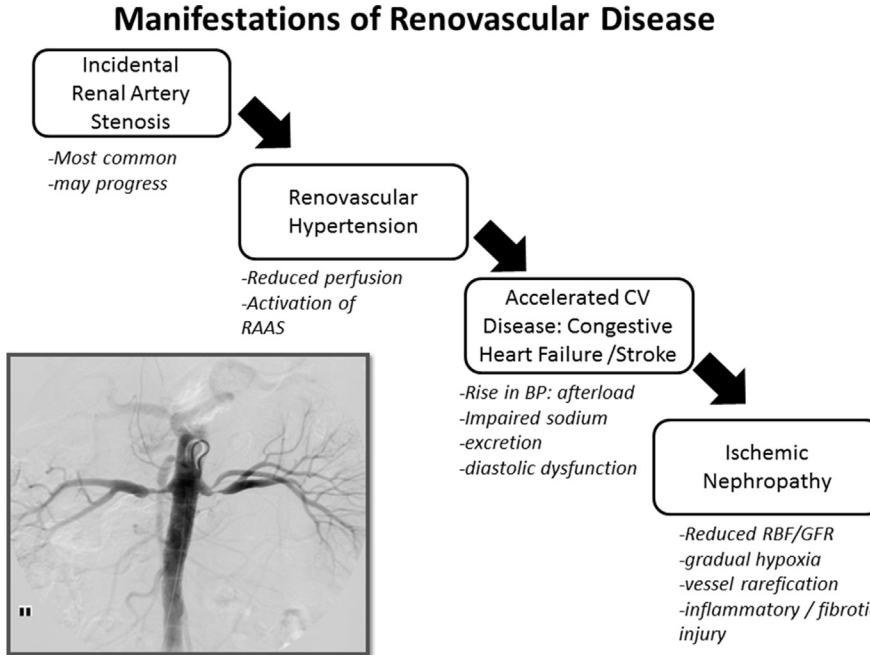

Fig. 1. Progressively more severe clinical manifestations associated with occlusive RVD. Minor degrees of lumen obstruction manifest as incidental lesions of minimal hemodynamic importance. As obstruction leads to reduced pressures and flow beyond the lesion, renovascular hypertension and acceleration of cardiovascular events ensue, particularly when associated with impaired sodium excretion. Ultimately, severe and long-standing RVD activates injury pathways within the kidney parenchyma that may no longer depend primarily on hemodynamic effects of stenosis and respond only partially to restoring vessel patency. BP, blood pressure; CV, cardiovascular; GFR, glomerular filtration rate; RAAS, renin-angiotensin-aldosterone system; RBF, renal blood flow.

peripheral vessels. Community-based studies suggest that up to 6.8% of individuals older than 65 years have ARAS with more than 60% occlusion.[2] Screening studies indicate an increasing prevalence of detectable ARAS in hypertensive subjects, from 3% (ages 50–59 years) to 25% (ages >70 years).[3] Clinically significant atherosclerotic RVD often is manifest by worsening or accelerating blood pressure increases in older individuals with preexisting hypertension.

Any flow-limiting vascular lesion within the renal circulation can produce renovascular hypertension (RVH). This can arise from a variety of fibromuscular dysplasias (FMDs), such as medial fibroplasia, which typically presents the appearance of a string-of-beads, or focal narrowing in the midportion of the renal artery[4] (**Fig. 2**). Some form of FMD may be detected incidentally in up to 3% of normotensive men or women presenting as potential kidney donors.[5] Those who progress to develop renovascular hypertension are predominantly women, some of whom are smokers. This gender predominance suggests that hormonal factors modulate the progression of this disorder and its clinical phenotype. Other disorders that produce RVH include renal trauma, arterial occlusion from dissection or thrombosis, and embolic occlusion of the renal artery (**Box 1**). Particularly in Asia, inflammatory vascular disorders such as Takayasu arteritis commonly affect the renal circulation. An emerging iatrogenic form of RVD includes occlusion of