Opinion Article

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"Hypertension and Renal Arterial Disease"

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Introduction

Renovascular hypertension has been known for almost 80 years, since seminal experimental research showed that gradual obstruction of the renal arteries causes a rise in systemic arterial pressure. These findings established the kidney as a key regulator of blood pressure and provided one of the most well researched models of "angiotensin-dependent" hypertension . Although progressive decline in renal blood flow eventually leads to other disruptions, including impaired volume management, circulatory congestion, and eventually irreversible kidney injury, this can occur at levels of renal pressure above those that impair kidney function. As a result, occlusive renovascular disease (RVD) encompasses a wide range of diseases, from small to major. In Western countries, atherosclerotic renal artery stenosis is the most common cause of RVD (at least 85 percent) (ARAS). This is usually the result of systemic atherosclerosis, which affects several arterial beds, including the coronary, cerebral, and peripheral vessels. According to community-based studies, up to 6.8% of people over the age of 65 have ARAS blockage of more than 60% . Screening studies show that the prevalence of detectable ARAS in hypertensive people increases with age, from 3% (years 50-59) to 25% (ages 70 and more). In elderly people with pre-existing hypertension, clinically significant atherosclerotic RVD is generally manifested by worsening or accelerating blood pressure increases. RVH can be caused by any flow-limiting vascular lesion in the renal circulation. This can be caused by a number of fibromuscular dysplasias (FMDs), such as medial fibroplasia, which appears as a "string of beads" or focal constriction of the renal artery in the middle . Up to 3% of normotensive men and women who come as potential kidney donors may be found to have some kind of FMD. The majority of people who acquire renovascular hypertension are women, with some of them being smokers. This gender disparity shows that hormonal factors influence the course of the illness and its clinical presentation. Renal trauma, arterial occlusion due to dissection or thrombosis, and embolic blockage of the renal artery are all causes of RVH . Especially in Asia. The renin-angiotensin-aldosterone system is activated, which causes RVH to develop. Angiotensin II (Ang II) has a variety of functions, including acting as a direct vasoconstrictor, stimulating aldosterone secretion from the adrenal glands, and inducing salt retention, according to studies conducted over several decades.

Additional pressor processes are activated by Ang II, including sympathetic adrenergic pathways, vascular remodelling, and prostaglandin-dependent vasodilation modification. The development of experimental RVH 8 is prevented by blocking the renin-angiotensin system or genetically knocking off AT-1 receptors. Secondary vasoconstrictor pathways can become dominant over time, making pharmacologic RAAS blockage and/or renal revascularization ineffective in entirely reversing RVH. Depending on the functional role of the remaining kidney (the non-stenotic or "contralateral" kidney), two different RVH models have been presented.When the contralateral kidney is healthy, it responds to increased systemic pressure by suppressing its own renin production and increasing "pressure natriuresis." This "2-kidney-1clip" syndrome is marked by unilateral renin release into the renal veins, increased plasma renin activity, and arterial pressure that is obviously dependent on Ang II's pressor actions. The "1-kidney-1 clip" RVD is characterised by the absence of a functional contralateral kidney or the inability to maintain ongoing pressure natriuresis. As a result, increased sodium excretion no longer offsets the rise in systemic pressure, resulting in volume expansion. A decrease in renin secretion from the stenotic kidney. Unless and until diuresis and volume contraction are achieved, these events result in lower levels for circulating plasma renin activity, loss of renal vein renin lateralization, and loss of detectable angiotensin dependence of systemic hypertension. With actuality, in 2-kidney-1-clip renovascular hypertension, the contralateral kidney is rarely normal, probably due to tissue harm from angiotensin II and/or other mechanisms. As a result, in many individuals with long-term RVH, reduced contralateral kidney function limits salt excretion. As a result, clinical laboratory symptoms in human participants differ greatly from the extremes anticipated by 1-kidney and 2-kidney experimental models. Surprisingly, investigations using blood oxygen level dependent (BOLD) MR show that blood flow

decreases (up to 35-40%) can occur without evidence of tissue hypoxia or long-term kidney fibrosis . This is due in part to the renal cortex's overperfusion as part of its filtering function, which is consistent with the discovery that less than 10% of oxygen is required to meet the kidney's energy requirements Due to energy-dependent active solute transport, the medulla is generally fed by post-glomerular arterioles with lower blood flow and has increased oxygen extraction. As a result, the kidney has a considerable cortical-medullary oxygen gradient, with deep medullary parts having significantly lower oxygen tension. As a result, moderate reductions in blood flow have only a minimal impact. Of course, renal tolerance to low blood flow has its limits. More severe and long-term reductions in blood flow endanger tissue oxygenation as well as the kidney's viability after stenosis. . Cortical hypoxia is finally linked to the activation of inflammatory pathways , according to studies of both experimental and human RVD, as shown schematically in FIGURE 3. Pro-inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-, MCP-1), indicators of injury (e.g. neutrophil gelatinase-associated lipocalin (NGAL), and the presence of t-lymphocytes and macrophages inside the tissue parenchyma, are all present. Atubular glomeruli are formed as a result of inflammatory alterations associated with severe ischemia, which result in tubule obliteration and failure to regrow intact epithelial surfaces. These methods eventually become inefficient.