

Epidemiology of Hypertensive Kidney Disease: Diagnosis

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Hypertension is a chronic disease that is increasing in prevalence worldwide and spans race and gender lines. The prevalence of hypertension, chronic kidney disease (CKD) and end-stage renal disease (ESRD) attributable to hypertension continues to rise worldwide. Identifying the precise prevalence of CKD attributable to hypertension is difficult owing to the absence of uniform criteria to establish a diagnosis of hypertensive nephropathy. Despite the increasing prevalence of CKD-associated hypertension, awareness of hypertension among individuals with CKD remains suboptimal and rates of blood-pressure control remain poor. Future investigations identifying early signs of hypertension-related CKD, increasing awareness of the effects of hypertension in CKD and determining optimal therapeutic interventions might help reduce the incidence of hypertensive nephropathy.

Hypertension-associated renal dysfunction is manifested primarily by increases in serum creatinine level. However, persistent increases in serum creatinine levels reflect substantial renal parenchymal damage and some degree of irreversible kidney dysfunction. Identifying individuals with early signs of CKD might help the targeting of therapies to more effectively prevent disease progression and associated complications. Microalbuminuria has been suggested as a potential marker of early kidney dysfunction but, as noted earlier, is clinically relevant only when increases into the macroalbuminuric range (>300 mg per day) occur in the presence of appropriate blood-pressure control

(that is, <140/90 mmHg). Microalbuminuria might accurately represent vascular dysfunction and serve as a marker of cardiovascular risk rather than CKD progression. Conversely, macroalbuminuria represents true renal parenchymal damage and should continue to serve as a prognostic marker of CKD progression and a therapeutic target in CKD treatment.

Novel markers of kidney injury are currently under investigation and could be used to identify the first signs of hypertension-associated renal injury. Serum levels of cystatin C have demonstrated promise as an early marker of hypertension-associated kidney dysfunction and may accurately reflect estimated Glomerular Filtration Rate (eGFR) in various populations. Despite the association of cystatin C level with systolic blood pressure, its ability to detect early kidney injury remains limited in its inherent connection with GFR. Like serum creatinine level, the serum level of cystatin C will only increase (indicating renal dysfunction) in the presence of a fall in GFR. The propensity of the kidney to hyperfilter and preserve GFR in the setting of early kidney injury limits the ability of any GFR-based estimation of kidney injury. In addition, novel bio-markers that reflect tubular damage (for example, neutrophil gelatinase-associated lipocalin and interleukin 18) could serve as early markers of hypertension-associated kidney injury. Further investigation into biomarkers of hypertension-associated CKD is ongoing and will hopefully be fruitful.

The associations of hypertension with CKD was initially identified in observational and experimental studies in which blood pressure was

measured in the setting of a specific health-care encounter in the clinic. The cumulative effect of elevated blood pressure on end organ damage, however, might best be demonstrated via blood-pressure monitoring outside of the health-care setting: that is, with home and ambulatory monitoring of blood-pressure levels. Elevated home blood-pressure readings were the best predictors of patient prognosis and home blood-pressure readings had a stronger association with the composite outcome (ESRD or death) and each individual outcome than office blood-pressure readings. The most important value of ambulatory blood-pressure monitoring, especially in patients with CKD, is the evaluation of circadian variations in blood pressure. During sleep, blood pressure should decrease by at least 10%, a concept called nocturnal dipping. The absence of this decrease, referred to as nondipping, is associated with increased severity of kidney disease and cardiovascular disease in patients with CKD. Ab-

sence of nocturnal dipping also seems to be associated with an increased risk of cardiovascular disease in individuals with and without CKD.

In conclusion, hypertension can lead to kidney disease or exist as a comorbid condition of kidney disease and can contribute to disease progression. The rates of hypertension-associated CKD and ESRD continue to rise and have a substantial influence on public health and health-care financing. Despite increasing rates of disease and evidence supporting the role of hypertension in CKD progression, patient awareness and control of hypertension remain suboptimal. Lifestyle and pharmacological interventions have both been demonstrated to be effective in lowering blood pressure. Continued vigilance by primary care physicians, nephrologists and other physicians that routinely care for these individuals is required to increase the rates of individuals with CKD and hypertension achieve target blood pressure levels.



Comment on a paper:

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