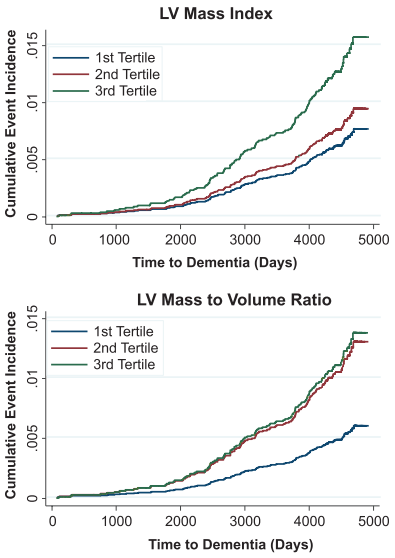
Cardiovascular Disease in Primary Aldosteronism (p 530)

Parameter	Odds ratio for CVD	95 % confidence interval	P
Serum K^+	0.735	0.503 - 1.073	0.111
Serum $K^+ \leq 3.5$ mEq/L	1.832	1.219 - 2.753	0.004*
Unilateral subtype	1.931	1.155 - 3.229	0.012*
PAC (pg/mL)	1.000	0.999 - 1.001	0.423
PAC ≥ 125 pg/mL	1.938	1.096 - 3.428	0.023*

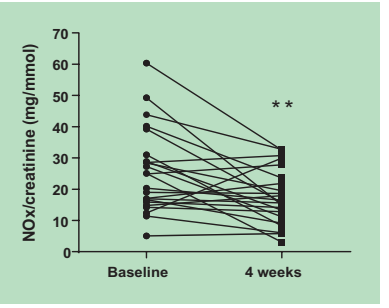
Aldosterone causes organ damage in animal models, and the prevalence of cardiovascular disease (CVD) among patients with primary aldosteronism (PA) is reportedly high. There have been several clinical studies examining the factors associated with CVD in these people; but the results are controversial. In some studies, the prevalence of CVD was distributed nearly equally across the spectrum of plasma aldosterone or serum potassium levels, whereas other studies reported that hypokalemic patients with PA had an elevated rate of CVD. Of the 2582 patients with PA studied in this study, the prevalence of CVD was higher than in age-, gender-, and blood pressure-matched patients with essential hypertension. Hypokalemia (≤3.5 mEq/L) and the unilateral subtype was associated with significantly higher adjusted odds ratios for CVD. Although plasma aldosterone concentration (PAC) was not linearly related to the adjusted odds ratios for CVD, patients with high PAC (≥125 pg/mL) had significantly higher adjusted odds ratios for CVD than patients with lower PAC (<125 pg/mL). Our result suggests PA patients with PACs higher than the upper limit of normal as well as with hypokalemia or unilateral subtype are at higher risk of developing CVD and, therefore, have a greater need for PA-specific treatment. In contrast, PA patients with normokalemia, the bilateral subtype, and PACs lower than the upper limit of normal are at lower risk of CVD and may not need immediate PA-specific treatment.

Left Ventricular Function and Cognitive Impairment (p 429)



A growing literature is revealing connections between cardiovascular disease and cognitive impairment/dementia. However, the relationships between subclinical cardiac dysfunction and cognitive performance is not been well established in the general population. In this issue of *Hypertension*, we investigated longitudinally cardiac magnetic resonance measures of left ventricular structure and function in relation to cognitive performance and incident dementia in a large multiethnic population free of cardiovascular disease at baseline. A total of 4999 participants with cardiac magnetic resonance images at baseline were followed for a median of 12 years. Left ventricular hypertrophy and remodeling were strongly associated with lower cognitive function and a higher risk of future hospitalization with dementia. Additionally, lower left ventricular sphericity (increased conicity) was associated with increased risk of future dementia. These associations were independent of demographic confounders, cardiovascular risk factors, and cardiovascular events. Measures of left ventricular function were not associated with risk of future dementia or cognitive function. This study provides 2 avenues of important insight into the link between cardiac and cerebral involvement in dementia. First, cardiac structural and functional abnormalities may be useful in the identification of individuals at risk of future cognitive impairment. Second, these data suggest that potential common pathophysiological mechanisms could underlie both cardiovascular and cerebrovascular structural remodeling (eg, microvascular disease) leading or contributing to cardiac concentric remodeling and vascular dementia.

VEGF Inhibitor–Induced Hypertension and Renal NO (p 473)



VEGF (vascular endothelial growth factor) is critically involved in tumor angiogenesis and is therefore an important target in the pharmacological treatment of many cancer diseases. Clinical use of VEGF inhibitors is associated with a preeclampsia-like syndrome characterized by rapid development of hypertension and proteinuria and has revealed an important role of VEGF in blood pressure regulation. The underlying mechanisms of VEGF inhibitor–induced hypertension are only partly understood but may include decreased systemic nitrogen oxide (NO) and increased plasma endothelin-1. Pazopanib is one of the recommended first line treatments in advanced renal cell carcinoma patients and in a prospective, single center observational study in a cohort of pazopanib-treated patients, significant hypertension developed during the first 4 weeks of treatment. Hypertension was accompanied by proteinuria, decreased plasma NO metabolites and increased endothelin-1. In addition, results showed decreased urinary excretion of NO metabolites without change in urine endothelin-1 or renal function. The kidneys are important in long-term regulation of blood pressure, and data support a significant role of reduced renal NO bioavailability in VEGF inhibitor–induced hypertension. Sufficient blood pressure control is necessary in this group of patients to prevent impaired treatment options of cancer targeted therapy. Based on mechanistic evidence, long-acting NO donors or cGMP-based drug interventions seem particularly well suited for organ protection and blood pressure reduction.

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