Pulmonary vein isolation and renal sympathetic denervation in CKD patients with refractory AF

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Mini review

Atrial fibrillation (AF) affects approximately 2% of the population worldwide, and this percentage will increase in the next 50 years [1,2]. The prevalence of AF is higher in older people, reaching up to 0.5% at 40 to 50 years of age and ranging from 5% to 15% at 80 years of age [1-5]. Men generally develop AF more frequently than do women. By 40 years of age, the lifetime risk of developing AF is almost 25% [6]. AF frequently complicates Chronic Kidney Disease (CKD) and is associated with adverse outcomes. Progression of end-stage renal disease is a major complication of CKD, and the incidence of AF is associated with a higher risk of developing the end-stage renal disease in patients with CKD [7]. The ideal approach for the treatment of AF is rhythm control, but this is sometimes very hard to accomplish [8]. Pokushalov and colleagues [9] recently reported that renal sympathetic denervation (RSD) diminishes systolic and diastolic blood pressure in drug-resistant hypertensive patients and reduces AF recurrences when combined with pulmonary vein isolation (PVI). Targeting of the pulmonary veins (PVs) and/or the PV antrum is the cornerstone for most AF ablation procedures. If the PVs are targeted, complete electrical PVI should be the goal of the procedure. For such procedures, complete isolation of all PVs is currently widely accepted as the best endpoint. A strategy using percutaneous catheter-based delivery of Radiofrequency (RF) energy was recently settled to interject the sympathetic innervation of the kidneys. This new procedure exposed no severe vascular or renal complications in the long term (up to 36 months). Our group believes that RSD can reduce AF recurrence in patients with CKD by modulation of the sympathetic hyperactivity present in this disease. As reported in the SYMPLICITY HTN-3 trial [10], no significant differences in the 24-h ABPM were observed between 6 and 12 months in the denervation and crossover subjects. Ambulatory data were available for only 20 of 70 (29%) non-crossover subjects at 12 months, given that ABPM was not protocol-mandated for these subjects at this time point. However, in these 20 subjects, a pattern similar to that of office readings was observed, showing a larger 24-h ABPM reduction at 6 than 12 months (−11.0 ± 19.5 vs. −6.1 ± 14.4 mmHg at 6 and 12 months, respectively; P = 0.272) [10].

Recent data suggesting that the combined number of complete and incomplete ablation runs (i.e., the overall number of ablation attempts) is related to greater blood pressure reductions [11]. Sympathetic activation is a hallmark of the essential hypertensive state occurring early in the clinical course of the disease [12-14]. In CKD, the sympathetic overactivity looks to be expressed at the earliest clinical phase of this condition, being directly related to the severity of the renal failure [15-18]. In both hypertension and renal failure, the mechanisms of the hyper adrenergic state are manifold and include reflex and neurohumoral pathways [12,13,17]. The adrenergic activation has an adverse impact on cardiovascular morbidity and, in the case of renal failure, also on cardiovascular mortality [12,13,18,19]. We believe that this over activity from the essential hypertensive state is in part controlled by anti-hypertensive drugs because patients maintain a normotensive state, leaving only sympathetic hyperactivity due to CKD.

The association of RSD with PVI may be a positive impact on AF recurrence. Once PVI was achieved, the dominant initiating source was eliminated. However, in patients with substantial pathology in the atrial substrate, additional intervention might be required to maximize the antiarrhythmic response. Additionally, ablation of afferent renal nervous input decreases central sympathetic output [15], which might attenuate autonomic triggers of AF and offer the potential for an antiarrhythmic effect superior to medications.

References


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