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COMMENT

Accumulating evidence suggests the potential of selective adrenal artery embolization as a standard treatment for primary aldosteronism

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Primary aldosteronism (PA) is a leading cause of secondary hypertension. Various studies to date have revealed that PA had cardiovascular and renal risk independent of blood pressure and that specific treatment could improve the cardiovascular prognosis [1]. Typically, the patients with unilateral PA undergo adrenalectomy and those with bilateral PA receive mineralocorticoid receptor (MR) antagonists, such as spironolactone and eplerenone. In addition, radiofrequency ablation of adrenal tumors has been shown to have comparable effect to surgery in the treatment of PA [2]. Another alternative, selective adrenal artery embolization (SAAE), has also been reported to have significant treatment effect on hyperaldosteronism. Inoue et al. reported the first case of aldosterone producing adenoma who received SAAE and achieved a significant reduction in blood pressure [3]. Furthermore, this approach has been applied to bilateral PA in recent years (Table 1). Zhang et al. and Dong et al. reported that SAAE safely improved plasma aldosterone level, serum potassium level, and blood pressure in small prospective cohorts of patients with bilateral PA [4, 5]. Besides, Zhou et al. conducted a controlled trial to compare the effect of MR antagonists and SAAE in bilateral PA patients, and the improvement in blood pressure, aldosterone level, and serum potassium level was comparable between two treatment groups [6]. While MR antagonists require patients to take medication for lifelong period, SAAE can be a one-time treatment and has the advantage of no need to continue specific treatment. However, compared with other established treatments such as adrenalectomy and MR antagonists, there is insufficient evidence to determine whether SAAE provides cardiac and renal benefits to PA patients. To address this, Qiu et al. evaluated echocardiography in addition to conventional parameters and observed a significant reduction in left ventricular hypertrophy at 12 months (median) after SAAE [7]. Although the data on cardiovascular risk is still warranted, it was the first report to show the favorable effect on cardiac function. On the other hand, in the present study, Lai et al. analyzed the impact of SAAE on renal function in a prospective cohort, which is the largest cohort ($n = 182$) to evaluate SAAE [8]. They compared pre- and post-treatment clinical and biochemical data of 182 PA patients who underwent SAAE and showed that eGFR decreased by 3.5% after a median follow-up of 8 months, whereas previous studies have reported eGFR reductions of 9.9% to 19.7% after adrenalectomy and 7.6% to 13.2% after MR antagonists administration [8]. The change in eGFR after SAAE was also evaluated according to basal renal function, and the group with lower basal eGFR showed a smaller decrease in eGFR. Based on these results, they concluded that SAAE corrected glomerular hyperfiltration, especially in the basal high eGFR group, and smaller reduction of eGFR compared to other treatment might reflect less SAAE-related renal damage.

It is well known that hyperaldosteronism induces glomerular hyperfiltration, resulting in an apparent increase in eGFR. However, there is a significant difference in urinary albumin level between patients with normal renal function and those with hyperaldosteronism, which means that glomerular hyperfiltration due to hyperaldosteronism causes glomerular injury and that eGFR is not a simple indicator of renal function especially in PA patients. In the present study, they evaluated various parameters, including eGFR, blood pressure, dose of antihypertensive drug, and

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Table 1 Recent studies evaluating the therapeutic effect of SAAE on bilateral PA

Reference	Subjects	No. of patients	Follow-up period	Evaluated parameters				
				BP, Dose of drug, PAC, Renin, Serum K	eGFR	Urinary Albumin	Echocardiography
Lai et al. [8]	Unilateral and bilateral PA	182	8 months (median)	○	○	—	—
Zhou et al. [10]	Bilateral PA (vs MR antagonists)	29	6 months	○	—	—	—
Qiu et al. [7]	Bilateral PA	31	12 months (median)	○	○	—	○
Zhou et al. [6]	Unilateral and bilateral PA (vs MR antagonists)	74	12 months	○	—	—	—
Dong et al. [5]	Bilateral PA	39	12 months	○	—	—	—
Zhang et al. [4]	Unilateral and bilateral PA	36	3 months	○	—	—	—

SAAE selective adrenal artery embolization, *PA* primary aldosteronism, *MR* mineralocorticoid receptor, *BP* Blood Pressure, *PAC* plasma aldosterone concentration, *eGFR* estimated glomerular filtration rate

aldosterone and renin levels before and after SAAE. However, the data on urinary albumin levels were not collected, and it remains to be validated whether a smaller decrease in eGFR indicates less renal damage or insufficient improvement of glomerular hyperfiltration. In addition, a multicenter registry study of PA in Japan reported that a smaller acute decrease in eGFR 6 months after MR antagonist administration was associated with a greater decline of long-term eGFR at a median follow-up of 4.5 years [9]. Long-term follow-up after SAAE is needed to conclude the exact meaning of smaller eGFR reduction after SAAE.

Considering the increasing number of PA patients and the importance of a sufficient approach to hyperaldosteronism, it is preferable to have multiple options for the treatment of PA to achieve biochemical and clinical cure as much as possible, depending on the patients' conditions, such as subtypes, severity, complications, or the patients' desire. To determine whether SAAE has comparable treatment potential to adrenalectomy and MR antagonists, further long-term studies evaluating changes in urinary albumin levels will help to clarify the more detailed impact of SAAE on renal function and prognosis.

Compliance with ethical standards

Conflict of interest The authors declare no competing interests.

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