Response to Effectiveness of Adrenalectomy and Aldosterone Antagonists for Long-Term Treatment of Primary Aldosteronism

Our data demonstrated the efficacy of mineralocorticoid receptor (MR) blockers for the treatment of primary aldosteronism (PA).1 However, by no means did we interpret our findings to show the superiority of adrenalectomy versus MR blocker therapy. The higher rate of cure of hypertension seen with surgery was intrinsic with the definition of cure: 42% of our adrenalectomized versus none of our medically treated patients could withdraw pharmacological treatment at long term. Hence, when guided by results of adrenal vein sampling, adrenalectomy provides long-term cure of hypertension and allows tapering, or withdrawal, of the antihypertensive medications.

Comparing adrenalectomy and MR blockade would require randomization of PA patients to either treatment. Neither the study of Reincke et al.2 nor our study used this design. We assigned patients to treatment based on adrenal vein sampling results. Moreover, when quoting us, Kaplan excerpted our full sentences: “This larger series of PA patients... allowed us to document a prominent and persistent decrease in LV mass index in the adrenalectomized patients, and a borderline significant fall in the medically treated PA patients, which might suggest the superiority of adrenalectomy over medical treatment in regressing LVH. However, the smaller size of this group with ensuing lower power might also account for the lack of statistical significance.” Hence, as our findings in the medically treated PA patients could derive from a type 2 (ß) statistical error,1 any conclusions on the superiority of either treatment are inappropriate.

At follow-up, left ventricular hypertrophy remained inappropriate for the decreased pre- and after load in both treatment groups, which might keep patients at higher risk. As we stated, the lack of left ventricular regression to an entirely normal condition emphasizes the need for an earlier diagnosis of PA followed by institution of specific treatment to achieve prevention, rather than regression, of the left ventricular changes.

The data on atrial fibrillation (AF) were obviously misinterpreted. Figure 1 shows that the excess of AF was because of persistence of the arrhythmia in the PA patients who had AF at baseline. Figure 3 illustrates a lower AF-free survival in the PA patients during follow-up from the initial diagnosis of PA and not from allocation to treatment. Hence, although PA implies a higher risk of AF, which is being tested in the Prospective Appraisal of the Prevalence of Primary aldosteronism in Hypertensive patients presenting with atrial flutter or fibrillation (PAPPHY) Study,3 these results do not allow to conclude that “continued increases in atrial fibrillation in the treated patients suggest that relief of autonomous hyperaldosteronism does not provide protection against this bothersome complication” as Kaplan did.

We fully support the view of Funder4 that MR blockade is valuable whenever the PA subtype cannot be diagnosed. However, our medically treated PA patients required multiple drugs to achieve blood pressure control, rather than a cheap MR antagonist. Moreover, their need for drugs increased (about 2.73, on average) during follow-up. Hence, even not considering the expenses for periodic biochemical testing and follow-up visits, Kaplan underestimated the total yearly drug cost. Sodium restriction, which lowers blood pressure and target organ damage in the presence of inappropriate aldosterone secretion, is another cheap and broadly appropriate option.

Disclosures

None.

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