Response to Metabolic Dysfunction in Primary Aldosteronism

We appreciate the editorial comments by Pimenta and Calhoun\(^1\) and the correspondence by Sechi et al\(^2\) on our work. Attempts to clarify the discrepancies between available studies on the metabolic effects of hyperaldosteronism are welcome. Indeed, our results challenge the prevailing hypothesis that primary aldosteronism (PA) is associated with clinically meaningful metabolic consequences.\(^3\) Pimenta and Calhoun\(^4\) appropriately point out several limitations that may threaten the validity of our study. On the other hand and in keeping with our data, Sechi et al\(^2\) summarize evidence indicating similar levels of insulin resistance in PA and essential hypertension (EH).

Pimenta and Calhoun\(^1\) call attention to the retrospective nature of our study. However, it is unlikely to have biased our results, because we analyzed quantitative variables that were objectively and prospectively measured during routine clinical care with very few missing data. As with other studies on the metabolic effects of hyperaldosteronism, however, some issues arise regarding the definition of cases with PA and the choice of controls.

In our institution, we waive suppression tests to confirm PA in patients with a high aldosterone:renin ratio and instead rely on high concentrations of plasma or urine aldosterone. The rationale of this approach has been discussed recently.\(^4\) Despite guidelines advocacy, the diagnostic value of suppression tests is not well established, and the best studies only indicate moderate accuracy.\(^5\) Moreover, our results were also negative in the large subgroup of patients with lateralized aldosterone hypersecretion, in whom the diagnosis of PA is well established.

The only controls available for our study were patients with EH. Therefore, we cautiously concluded that there was no clinically apparent excess of metabolic disturbances in PA compared with EH. The lack of change in plasma glucose and serum lipids after adrenalectomy in patients with lateralized PA makes an additional point, although the follow-up interval was rather short (median time: 28 weeks).

Taken with the evidence mentioned by Sechi et al,\(^2\) our results argue against a clinically significant difference in insulin resistance between patients with PA and otherwise similar patients with EH. We agree that these findings do not definitely preclude metabolic effects of aldosterone. Such effects are still suggested by the different metabolic profile of patients with PA and of normotensive subjects and by some studies showing an association between higher levels of aldosterone and metabolic disturbances in patients with PA or in patients with EH. However, association is not causation, and it remains to be elucidated why large differences in aldosterone levels do not translate into metabolic differences between patients with PA and patients with EH.

Disclosures

None.

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Hypertension. published online April 27, 2009;
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

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http://hyper.ahajournals.org/content/early/2009/04/27/HYPERTENSIONAHA.109.133058.citation

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