Metabolic Dysfunction in Primary Aldosteronism

To the Editor:

The study by Matrozova et al. analyzed retrospectively the metabolic parameters of a large French cohort of patients with primary aldosteronism (PA) who were compared with matched patients with primary hypertension (PH). In these 2 groups of patients, fasting plasma glucose levels, as well as the prevalence of hyperglycemia, were comparable, supporting the contention that abnormal carbohydrate metabolism and the metabolic syndrome are equally frequent in PA and PH. Also, metabolic parameters were found to be comparable in patients with different subtypes of PA.

As discussed by the authors of the article and in an accompanying editorial, these findings are somehow deviant from those of previous studies that were conducted in smaller groups of patients and in which PA was found to be associated with greater impairment of insulin sensitivity and more frequent metabolic syndrome than PH. Although many reasons could explain disparity of findings, the methodology used to assess carbohydrate metabolism is of primary importance. Because this methodology is complex, the majority of these studies have used fasting plasma glucose and insulin values rather than the gold standard, the euglycemic-hyperinsulinemic clamp. In a prospective study, we compared patients with PA with both primary hypertensive and normotensive controls by use of the hyperinsulinemic clamp, reporting insulin resistance in both hypertensive groups in comparison with normotensive subjects. In agreement with Matrozova et al., we found no difference in the fasting glucose and lipid levels between the 2 hypertensive groups but observed a greater impairment of insulin sensitivity in terms of metabolic clearance rate of glucose in PH compared with PA. Also, consistent with the findings of Matrozova et al., we did not observe differences in glucose metabolic clearance rate between patients with adrenal adenoma or idiopathic PA. Thus, our results, together with those of the French authors, would suggest that hypertensive states are associated with insulin resistance independent of their etiology.

This conclusion does not preclude the possibility that aldosterone contributes, among other factors, to the determination of insulin resistance in hypertensive patients. In the 5.7-year follow-up of our patients with PA, we found that altered parameters of glucose metabolism were significantly, although not completely, corrected after both adrenalectomy and administration of mineralocorticoid receptor antagonists. Furthermore, in a recent cross-sectional study that was conducted with use of the hyperinsulinemic clamp in patients with PH, we have reported a strong association between elevated plasma aldosterone levels and decreased sensitivity to insulin. Therefore, the role of aldosterone in causing alterations in glucose homeostasis might be relevant not only in patients with PA but also in patients with lesser degrees of aldosterone excess.

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Disclosures

None.

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