The Relationship of Glycemia to Lipid and BP Lowering

In recent years, glucose control has been at the center of many debates, including those about how intense control should be and the effect of intensive control on cardiovascular (CV) risk factors. But none of the many studies searching for answers to the debates have examined whether glycemia influences the ability to achieve lipid and blood pressure (BP) targets, say researchers from the Phoenix Arizona Indian Medical Center; the University of Oklahoma; the Washington Hospital Center; and the National Heart, Lung, and Blood Institute. To find out, the researchers conducted a post hoc analysis of data from the Stop Atherosclerosis in Native Diabetics Study (SANDS).

The study participants included 499 Native Americans who were randomly assigned to 1 of 2 treatment groups: aggressive or standard. In the aggressive group, the target goals were ≤ 115 mm Hg for systolic BP; ≤ 70 mg/dL for low-density lipoprotein cholesterol (LDL-C); and ≤ 100 mg/dL for non–high-density lipoprotein cholesterol (non–HDL-C). In the standard group, the goals were ≤ 130 mm Hg for systolic BP (SBP); < 100 mg/dL for LDL-C; and ≤ 130 mg/dL for non–HDL-C.

The researchers examined the association between A1C and carotid intima medial thickness (CIMT) and left ventricular mass index (LVMI). They also evaluated the potential effects of baseline A1C on achieving SBP, LDL-C, and non–HDL-C goals. They obtained A1C measurements for 491 participants at baseline and 426 at the end of the study. Change in A1C measures was available for 419 participants.

The analysis revealed no significant change between the 2 groups in A1C after 36 months. However, the likelihood of reaching target goals declined significantly in both groups with increasing tertile of baseline A1C. A1C did not influence the effects of lipid and SBP lowering on carotid atherosclerosis (ATH), which the researchers say reinforces the need to focus on lipid and BP control in diabetes regardless of glycemia status. The degree of baseline glycemia also did not significantly influence change in CIMT or LVMI over 36 months, implying that improvements in CV disease risk are possible despite the patient's level of glycemia, the researchers conclude.

SANDS is unique, the researchers say. Instead of comparing pharmacologic regimens, SANDS compared intensity of treatment. The results of this analysis are noteworthy, they add, for 2 of the following reasons: (1) by showing that the improvements in subclinical measures of ATH and cardiac function did not differ as a result of differences in glycemia control; and (2) by showing that the treatment strategies used to achieve the BP and lipid targets did not affect glycemia control, "eliminating potential negative consequences of treatment regimens." Thiazide diuretics and beta-blockers (steps 2 and 3 of the SANDS regimen for BP control) can have adverse effects on glucose management, whereas ACE inhibitors can have beneficial effects. Had intensive lipid and BP treatment worsened glucose control, the researchers say, "that finding would have complicated clinical decision making because of the known benefits of glycemia control on microvascular complications."

Their findings demonstrate that it's possible to pursue aggressive goals without endangering glucose control—supporting current guidelines that advise pharmacologic control of BP and lipids as the primary non–lifestyle-related risk reduction strategy in diabetes.

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