

Blood Pressure Correlates With Glucose Levels

BY ERIK GOLDMAN
Contributing Writer

MADRID — Fasting blood glucose levels appear to be higher in diabetic patients with poorly controlled blood pressure than in those with well controlled pressure, said Dr. Miroslav Soucek, at the annual meeting of the European Society of Hypertension.

This observation was based on a survey of more than 2,200 patients from 150 primary care practices throughout the Czech Republic. The primary objective of the study was to determine the prevalence of hypertension in the Czech population, and the extent to which physicians there are able to help their patients achieve pressure control targets as outlined in current ESH guidelines, said Dr. Soucek, who presented the findings in a poster.

Each participating physician recorded thorough case data from 15 consecutive patients aged at least 45 years, irrespective of the reason for each patient's visit. The idea was to get a representative sampling of the health status of all comers to primary care offices. The investigators defined hypertension as pressures above 140/90 mm Hg. Dr. Soucek and his colleagues obtained data from 2,211 patients with a mean age of 62 years.

Of the entire cohort, 78% of the patients were defined as hypertensive; of the 403 patients with diabetes, 75% had hypertension. Only 18% of all patients being treated for hypertension were considered well controlled (pressures under 130/80 mm Hg); the rate for diabetics was 6%.

Dr. Soucek noted that blood pressure was uncontrolled in almost 30% of the diabetic patients with hypertension even though they were on at least three anti-hypertensive drugs.

But the most striking finding of this study, one that surprised the investigators themselves, was the correlation between poor pressure control and increased fasting blood glucose. "The average fasting blood glucose showed a gradual increase, with increasing blood pressure, from 7.98 mmol/L in diabetics with blood pressure under 130/80 mm Hg to 9.44 in diabetic patients with blood pressures greater than 180/110 mm Hg," reported Dr. Soucek of the department of internal medicine, St.

Anne University Hospital, Brno, Czech Republic.

The mechanism underlying this connection is not known, and it is too soon to tell if there is a causal connection.

The clinical implication, however, is clear: Uncontrolled pressure in a diabetic patient may be a signal for uncontrolled glucose as well. These patients need even closer attention than nondiabetic hypertensives or diabetics who are not hypertensive, he said. ■

Lipid-Lowering Drug Reduces Glucose Levels

WASHINGTON — Colesevelam (Wel-Chol), approved in the United States since 2000 for lowering lipid levels, also appears to reduce postprandial glucose in patients with type 2 diabetes, Dr. Franklin Zieve and his associates reported in a poster at the annual scientific sessions of the American Diabetes Association.

The incidental observation prompted Daiichi Sankyo Inc. to sponsor a prospective study of the glucose-lowering effects of its bile acid sequestrant drug, Dr. Zieve of the Hunter Holmes McGuire Veterans Affairs Medical Center, Richmond, Va., told this newspaper at the meeting.

A total of 65 patients with type 2 diabetes with hemoglobin A_{1c} levels of 7.0% or above were randomized to receive 3.75 g/day of colesevelam (6 tablets/day) or placebo for 12 weeks, following a 4-week placebo run-in period. Patients continued taking their existing antidiabetic medications. Thirty-two colesevelam and 27 placebo subjects completed the trial.

At 12 weeks, mean postprandial glucose levels were reduced by a significant 18 mg/dL (from 269 to 251) in the colesevelam group, compared with an insignificant gain of 3 mg/dL (285 to 288) in the placebo group. Hemoglobin A_{1c} levels dropped by approximately 0.3 percentage points from baseline with colesevelam, resulting in a 0.5 percentage-point difference from placebo at 12 weeks.

—Miriam E. Tucker

Nonproliferative Diabetic Retinopathy

Hyperglycemia-induced
PKC β
Overactivation

↓

Microvascular Leakage

↓

Macular Edema

↓

Vision Loss

PKC β overactivation is a major threat to vision in patients with diabetes

In diabetes, PKC β overactivation* is one of several mechanisms that can lead to endothelial dysfunction, retinal microvascular damage, and increased vascular permeability.^{1,2} The resulting microvascular leakage may lead to macular edema, a primary cause of vision loss in nonproliferative diabetic retinopathy.³

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* Protein Kinase C β (PKC β), an intracellular enzyme involved in vascular function, can be overactivated by hyperglycemia in patients with diabetes.

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