Diabetic Retinopathy Predicts Ischemic Stroke

BY MARY ANN MOON

Contributing Writer

iabetic retinopathy is an independent risk factor for ischemic stroke, reported Dr. Ning Cheung of the University of Melbourne's Centre for Eye Research, and associates.

Since retinopathy is a common microvascular manifestation of diabetes, this finding indicates that microvascular disease is an important pathway for stroke in

patients with diabetes. In contrast, largevessel disease plays the central role in ischemic stroke in nondiabetic patients, the researchers said.

They used data from the Atherosclerosis Risk in Communities (ARIC) study to examine the relationship between diabetic retinopathy and ischemic stroke. Two previous population-based studies of the issue were inconclusive, "and few other data are available," Dr. Cheung and associates noted (Stroke 2007 January [Epub

doi:10.1161/01.STR.0000254547.91276.50]).

The ARIC study was a prospective, population-based, cohort study. Subjects were recruited during 1987-1989 and were aged 45-64 years at entry. A subset of 1,617 participants who had retinal photographs taken in the early 1990s accounted for the subjects for Dr. Cheung's study.

After the data were adjusted to account for several potentially confounding factors such as subject age, gender, blood pressure, and smoking status, those who had dia-

betic retinopathy were significantly more likely to develop ischemic stroke during 7 years of follow-up than were subjects who did not have diabetic retinopathy.

This association remained significant after the data were further adjusted to account for plasma fibrinogen levels and white blood cell count.

There was no dose-dependent association between the severity of retinopathy and the risk of ischemic stroke, the investigators added.

Duloxetine Has 'Modest' Effect on Glycemic Control

Treatment with duloxetine for diabetic peripheral neuropathy has only a "modest" impact on glycemic control, Dr. Thomas Hardy of Eli Lilly & Co. and colleagues reported using data from three randomized controlled trials.

The researchers noted, however, that the studies they used to look at duloxetine's impact on glycemic control were not specifically designed to answer that question. "The current study does not allow definitive conclusion about the metabolic effects of duloxetine or other medications utilized in these studies," they wrote (Diabetes Care 2007:30;21-6).

The authors pooled data from three randomized, controlled trials of adults with diabetes and diabetic peripheral neuropathic pain (DPNP). In the acute phase of the study, 1,024 participants were randomized to 60 mg duloxetine once a day or twice a day or to placebo for 12 weeks. In the study's extension phase, a subset of 867 participants was rerandomized to duloxetine or usual care in an open-label mode for another 52 weeks.

Patients with HbA_{1c} values greater than 12% and those with major depressive disorder were excluded from the study. The authors looked at data on fasting plasma glucose (FPG), HbA_{1c} , lipid levels, weight, and pain severity in both the acute and extension phases of the study.

The results showed that in the acute-phase studies, duloxetine-treated patients experienced an average increase in fasting glucose of 0.50 mmol/L, compared with a decrease of 0.11 mmol/L in the placebo group. In the extension studies, FPG again increased in the duloxetine-treated group, compared with the routine care group (0.67 mmol/L vs. -0.64 mmol/L).

 ${\rm HbA_{1c}}$ levels dropped slightly more in the duloxetine-treated group, compared with the placebo group during the acute phase, but the difference was not statistically significant. The longer-term studies showed an increase in ${\rm HbA_{1c}}$ in both the duloxetine group and the group receiving routine care, with the duloxetine-treated group having a significantly larger increase (0.52 % vs. 0.19%).

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