

Racial Disparities Seen in Parkinsonism Severity

Diagnosis, accessibility, and economic factors may point to African Americans' higher UPDRS scores.

BY MARY ANN MOON

FROM ARCHIVES OF NEUROLOGY

African Americans with parkinsonism had more severe symptoms, more disability, and poorer symptom management than did whites during a 5-year assessment at a single center.

These disparities cannot be attributed to differences between the races in age, cognitive function, or disease duration since these factors were comparable between blacks and whites in this single-center study. It appears that the racial disparities "may be explained by delayed diagnosis, referral patterns, access to care, economic factors, or a combination of all of these," wrote Dr. J. Patrick Hemming and his associates at the University of Maryland, Baltimore (Arch. Neurol. 2010 Dec. 13 [doi: 10.1001/archneurol.2010.326]).

Independently of race, both lower socioeconomic status and lower education level also were associated with more severe signs and symptoms, greater disability, and poorer management of parkinsonism in this study, which the investigators described as "the first to show health disparities in disease severity and disability in parkinsonism."

"Studies in different patient populations

and geographic locations are necessary to confirm these findings," they noted.

In their study, Dr. Hemming and his colleagues evaluated 1,090 patients with parkinsonism who were participating in a quality of life assessment at the uni-

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Major Finding: African Americans scored an average of 10 points higher on the Unified Parkinson's Disease Rating Scale than did whites.

Data Source: Single-center, observational cohort study of 1,024 white and 66 black patients with parkinsonism.

Disclosures: Dr. Hemming's associates reported ties to numerous manufacturers of drugs for Parkinson's disease.

versity's movement disorders center between 2003 and 2008. A total of 66 patients were African American and the rest were white; the researchers were unable to assess patients of other racial or ethnic backgrounds.

African Americans scored an average of 10 points higher on the Unified Parkinson's Disease Rating Scale than did whites (53 vs. 42.8). The investigators called this finding a "striking difference that may influence mortality" because a previous study found that a one-point in-

crease on the UPDRS scale was associated with a relative risk ratio of 1.1 for death within 7 years.

African Americans also had worse scores than did whites on a modified version of the Older Americans Resource and Services (OARS) disability subscale, which measures the level of difficulty in performing 14 daily activities.

Significantly fewer African Americans were prescribed antiparkinson medications than were whites (62% vs. 78%), and fewer African Americans also were receiving new dopaminergic agents (21% vs. 41%). In contrast, significantly more black patients used antipsychotic medications than did whites (13% vs. 6%).

When the data were analyzed by income and education level after controlling for race, UPDRS and OARS scores were significantly higher among patients who earned less than \$30,000 per year than among those who earned more than \$70,000 per year.

Similarly, low-income patients used newer dopaminergic agents significantly less often than did high-income patients (30% vs. 47%). Low-income patients, however, were more likely to be prescribed antidepressants, antipsychotics, and antimentia agents.

Newer dopamine agonists were pre-

scribed significantly less often for patients with less than a college education (35%) than for those with a college education (43%). In contrast, antipsychotics were approximately twice as likely to be prescribed for patients without a college education (8.4%) than they were for those with a college education (4.7%).

These findings reinforce the conclusion that racial disparities in the management of parkinsonism are not solely due to differences in income or education level, but that race itself is a significant independent factor, Dr. Hemming and his associates said. "We need to better understand the cause of parkinsonism and to find remedies for disparate outcomes among patients with parkinsonian disease who are of different backgrounds and means," they noted.

"Studies have shown that African Americans and other minorities may perceive common medical conditions as natural processes that do not require medical intervention," the investigators wrote.

In addition, they suggested that "physicians may be influenced by unconfirmed reports that Parkinson's disease is less common in African American populations."

Future studies should examine patient and physician attitudes and beliefs about symptoms of and therapies for parkinsonism, Dr. Hemming and his associates added. ■

Statin Use Reduced Risk of Parkinson's in Cohort Study

BY SUSAN LONDON

FROM THE ANNUAL MEETING OF THE NORTH AMERICAN PRIMARY CARE RESEARCH GROUP

SEATTLE – Statin use was associated with protection against the development of Parkinson's disease in a population-based historical cohort study of 94,308 middle-aged and older adults in Israel.

Statin users, who accounted for one-third of the cohort, had a 27% lower risk of Parkinson's disease (PD), compared with nonusers, after adjustment for potential confounders.

"Statins, in addition to lowering cholesterol levels and reducing cardiovascular risk, may have a protective effect on the incidence" of PD, Dr. Amnon Lahad said at the meeting.

PD, a central nervous system degenerative disease, "may react like other cardiovascular diseases in responding to statins," he said, speculating that ischemia plays a role in its pathogenesis, much as it does for dementia. "It is probably at the level of the microvasculature. We don't really know how to test [for it], but it's there."

Half a dozen studies have assessed the association between statins and PD in recent years, but their conclusions have been inconsistent. Most have found that statin use confers increased risk, with a subset suggesting that this association is mediated through cholesterol levels, making statins simply a confounder, said Dr. Lahad, chairman of the department of family medicine at the Hebrew University of Jerusalem.

He and his colleagues searched the database of the largest health maintenance organization in Israel to identify all patients older than 45 years in a single administrative region during 2001-2007. They excluded patients who had PD or took statins before the study period, used neuroleptic drugs at any time, changed health insurance, or did not have a record of LDL cholesterol values.

Statin use and chronic illnesses were also ascertained from the database. Family history on PD was not available, and body mass index was not used because it was inconsistently recorded, according to Dr. Lahad.

Analyses were based on

VITALS

Major Finding: Statin users had a 27% lower risk of developing Parkinson's disease than did nonusers.

Data Source: A population-based historical cohort study of 94,308 patients from one region of Israel.

Disclosures: Dr. Lahad reported that he had no relevant conflicts of interest.

94,308 patients, he reported. The cohort was nearly equally divided by sex. About a fifth of patients had low socioeconomic status, as indicated in the database by the waiving of their co-payment for prescriptions.

Substantial proportions of the cohort smoked (20%) and had diabetes (22%), hypertension (51%), or ischemic heart disease (19%), or had previously experienced a stroke (8%).

Some 32% of the patients were classified as statin users because they filled at least six monthly statin prescriptions during a 9-month period. The rest were classified as nonusers.

Overall, 1.1% of the cohort developed PD during the study period, as ascertained from their filling of at least two monthly

prescriptions for an antiparkinsonian medication.

In a Cox regression analysis, the risk of PD was elevated for men compared with women (hazard ratio, 1.57; P less than .0001), for patients with low socioeconomic status compared with their better-off peers (HR, 1.33; P less than .0001), and for patients who had experienced a stroke compared with those who had not (HR, 1.96; P less than .0001).

"Surprisingly, the other diseases or conditions [hypertension, ischemic heart disease, diabetes, and smoking] were not related, even in a pretty big group," to PD, Dr. Lahad said. "The most surprising is smoking, which in the literature is connected."

LDL cholesterol level at baseline was not significantly associated with the risk of PD. However, there was a trend for an increased risk of PD with an LDL level greater than 100 mg/dL, and a lower risk of the disease in those with LDL levels greater than about 160 mg/dL.

When statin use was added to the analysis, statin users had a one-fourth reduction in the risk of PD relative to nonusers (HR, 0.73; $P = .001$), and the other significant associations persisted.

However, when the investigators accounted for statin use, the individuals with the highest LDL cholesterol levels no longer had a reduced risk of the disease. "It probably was the effect that this group got statins much more often, so it did protect them," Dr. Lahad speculated. "And it showed, without the statin, purely the effect of the cholesterol."

The risk of PD fell with an increasing duration of statin use (as assessed from the number of prescriptions and months of use). But in this case, the association was weaker. "It was a trend; it wasn't by itself significant," he noted.

"Of course, it's not a randomized controlled trial," Dr. Lahad acknowledged, so it is possible that other factors explain the observed association. "But at least we don't find the alarming finding of previous studies that show that statins are connected to an increase in morbidity." ■