Functional MRI Reveals Brain Damage in Lupus

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TORONTO — Cognitive impairment in patients who have childhood-onset systemic lupus erythematosus can be identified with functional magnetic resonance imaging, reported Svetlana Lvovich, D.O., in a poster presentation at the annual meeting of the Pediatric Academic Societies.

The pilot study, which was done by Dr. Lvovich and her associates, included 10 patients who had systemic lupus erythematosus (SLE). Five of the patients had cognitive impairment (CI), and five of them did not have CI.

Functional magnetic resonance imaging (fMRI) showed differences between the patients with CI and those without.

"They [those with CI] need to recruit more neurons to do the task," she said in an interview. The three tasks that patients were required to perform during fMRI were specially designed to evaluate attention, working memory, and language processing.

Neuropsychiatric testing is the preferred method for diagnosing CI in SLE patients. However, it is time consuming and it requires extensive training to be able to administer it, said Dr. Lvovich of the Cincinnati Children's Hospital Medical Center.

In contrast, fMRI is quick and easy to perform.

There also were subtle fMRI differences seen between SLE patients with normal cognition, compared with healthy controls, which suggested that it is SLE-specific processes that contribute to CI rather than the long-term use of steroids," she noted.

"However, to determine this, we will need to look at patients who are on steroids but don't have SLE," she said.

"We think that fMRI may be useful to investigate and identify cortical roots of cognitive impairment in children with SLE," she concluded. ■

Family History, Smoking Promote Cartilage Loss

Smoking seems to contribute to the development of knee cartilage loss and defects in those with a family history of knee osteoarthritis (OA), according to results reported by Dr. Changhai Ding and associates of the University of Tasmania in Australia.

In this study, 345 relatively young individuals (average age, 45 years) were measured at baseline and again 2.3 years later. Of the 162 persons with at least one parent with severe primary knee OA, 40 current smokers had greater loss in medial and lateral tibial cartilage volumes (beta = –2.20% and –1.45%. respectively) than did 47 former smokers and 75 never-smokers, after adjusting for confounding factors in a logistic regression analysis. Pack-years of smoking were also significantly associated with changes in cartilage volume (Arthritis Rheum. 2007;56:1521-8).

The authors did not find a similar relationship between smoking status and knee OA measures in 163 individuals with no family history of knee OA. The only factor significantly associated with smoking status in control individuals was change in lateral tibiofemoral cartilage volume, providing evidence for a "gene-environment interaction in the etiology of knee OA."

Among those with a family history of knee OA, being a current smoker increased the risk of developing medial tibiofemoral cartilage defects by nearly fivefold during the study period and increased the risk of lateral tibiofemoral cartilage defects by threefold. The risk increases in heavy smokers (at least 20 pack-years) versus never-smokers were 10-fold and 13-fold, respectively.

The interaction in smoking status, family history of knee OA, and cartilage effects remained significant in regards to change in medial tibial cartilage volume and increases in cartilage defects, both medial and lateral, after adjusting for confounding factors. In the overall group, the prevalence of knee pain was higher in current smokers (41%) than in former smokers or never-smokers (33%), regardless of family history. However, there was no overall association between smoking status and baseline tibial cartilage volume or prevalent tibiofemoral cartilage defects.

—Melinda Tanzola