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Dietary Salt Associated With MS Activity

Sodium intake was positively correlated with risk of increased disease activity in patients with multiple sclerosis, according to a small study reported here.

Each gram of estimated daily sodium intake above the average in a 52-patient sample was associated with an increase of 3.65 in MRI lesion counts, said Mauricio Farez, MD, PhD, of Fundación para la Lucha contra las Enfermedades Neurológicas de la Infancia in Buenos Aires.

Also, patients with estimated salt intake classified as high -- more than 4.8 g daily -- showed relapse rates that were 3.95 times greater (95% CI 1.39-11.21) than those with intakes less than 2 g/day, he told attendees at the European Committee for Treatment and Research in Multiple Sclerosis annual meeting.

Farez emphasized repeatedly that the findings did not prove that high salt intake caused the increased disease activity. He acknowledged that, if there is a causal relationship, it possibly could go in the reverse direction -- that patients with highly active MS may increase their salt intake as a result. But he said he did not view that as very likely.

The study in a total of 122 patients with relapsing-remitting MS grew out of previous research connecting salt intake with vitamin D levels and body mass index, he said. Numerous studies have indicated an association between vitamin D status and MS risk -- including one reported minutes earlier at the Copenhagen meeting -- and it seemed logical to examine whether sodium may share a similar association, Farez explained.

He and colleagues initially recruited 70 patients for a first phase of the observational study. They underwent a baseline MRI scan in November 2010, followed by MRI scans and analysis of urinary sodium excretion as a means of estimating sodium intake 1 year later. Finally, in November 2013, relapse rates for the preceding 2 years were calculated.

During this first phase, the MRI analyses included "combined unique activity" counts -- the total of new T2 lesions and new gadolinium-enhancing T1 lesions since the baseline scan.

A second group of 52 patients was examined in June 2013 with MRI scans and urinary sodium testing to provide replication data for the association between sodium intake and MRI lesion activity. Because this group had only a single scan and no follow-up, Farez and colleagues could only calculate T2 lesion loads, not the combined unique activity lesion counts nor relapse rates.

Farez acknowledged that an important limitation of the study was that it did not measure urinary sodium excretion with 24-hour urine collections, which he said were impractical since they require participants to carry a large container to capture all their urine for a whole day and night.

Instead, his team relied on spot urine collections and a published formula to estimate daily sodium excretion and, from that, daily sodium intake.

He reported that, in the first group of patients, not only those with high sodium intake (more than 4.8 g/day) but also those with "average" consumption showed increased risk of relapse. Participants with estimated daily intake of 2.0 to 4.8 g/day had relapse rates that were 2.75 times that of the low-intake group (95% CI 1.30-5.81).

Comparing both the average- and high-intake groups, the trend was significant at P=0.001, he reported.

Also in the first group, combined unique activity lesion counts were approximately three times greater in both the average- and high-intake groups compared with the low-intake group (P not reported), Farez said.

And, T2 lesion counts were similar in the first cohort's low- and average-intake groups at an average of about 6, but they reached a mean of approximately 14 in the high-intake group (P<0.05).

In the replication set of 52 patients, similar results were seen, Farez said. He and his colleagues calculated that each increment in intake of 1 g above the cohort average was associated with 3.65-lesion increase in T2 count (SD 0.77, P<0.001).

He did not specify the cohort average, but he said that the national average in Argentina has been measured at nearly 5 g/day, well above the U.S. mean of 3.4 g/day.

The World Health Organization has recommended a maximum daily intake of 2 g/day. In the U.S., several government agencies have jointly called for a maximum of 1.5 g/day -- but earlier this year, the Institute of Medicine complained that the scientific evidence did not a support such a low figure, instead backing an older standard of 2.3 g/day.

Farez and colleagues also measured serum sodium but found no relationship between it and clinical or MRI activity. It was also not significantly associated with estimated sodium intake, with an R^2 value of just 0.0082.

Asked by the session moderator what a causal mechanism might be, Farez said previous studies had suggested that high salt levels can promote increased inflammatory activity throughout the body. Also, he said, it may increase permeability in the blood-brain barrier, which could contribute to inflammation in the central nervous system.

Whatever such a mechanism may be, he said, "it does not seem to occur in peripheral blood."