MON-636: The Relationship Between Glucose Control & Cognitive Function in People with Diabetes After a Lacunar Stroke

Tali Cukierman-Yaffe. *Sheba Medical Center and Tel-Aviv University*

Tali Cukierman-Yaffe, MD, MSC[1], Leslie McClure, PhD[2], Thomas Risoli, MSC[3], Jackie Bosch, PhD[4], Hertzel C. Gerstein, MS, MD, MSC,FRCPC[5], Oscar Benavente, MD[6].
[1]Endocrinology Institute, Sheba Medical Center, Epidemiology Department, Sackler School of Medicine, Herceg Institute on Aging, Tel-Aviv university, Tel-Aviv, Israel, [2]Department of Epidemiology & Biostatistics Dornsife School of Public Health, Drexel University, Philadelphia, PA, [3]Duke CTSI Biostatistics, Epidemiology and Research Design (BERD) Methods Core Duke University School of Medicine,Durham, NC, Durham, NC, [4]School of Rehabilitation Sciences, McMaster University, Hamilton, Canada, Hamilton, ON, Canada, [5]McMaster University, Hamilton, ON, Canada, [6]Department of Medicine, Division of Neurology, University of British Columbia, Vancouver, Vancouver, BC, Canada.

Background & Objective: Both lacunar strokes and diabetes are risk factors for dementia and cognitive dysfunction. Thus, elucidating modifiable risk factors for cognitive dysfunction in people with type 2 diabetes who experienced a lacunar infarct has large public health implications. One such factor may be glycemic status, as measured by glycosylated hemoglobin (A1C). Thus, the aim of this study was to assess the relationship between A1C and cognitive function in people with diabetes after a lacunar stroke.

Research Design & Methods: The effect of baseline and follow-up A1C on the baseline and the change in Cognitive Assessment Screening Instrument (CASI) score over time among participants with a median of 2 cognitive assessments (range 1-5) was examined in of 942 individuals with diabetes and a lacunar stroke who participated in the Secondary Prevention of Small Subcortical Strokes (SPS3) trial (ClinicalTrials.gov number, NCT00059306).

Results: Every 1 % higher baseline A1C was associated with a 0.06 lower standardized CASI z-score (95% CI -0.101, -0.018). Higher baseline A1C values were associated with lower CASI z-score over time (p for interaction=0.037). A 1% increase in A1C over time, corresponded with a CASI score decrease of 0.021 (95% CI -0.0043, -0.038) during follow-up. All these remained statistically significant after adjustment for age, sex, education, race, depression, hypertension, hyperlipidemia, BMI, CVD, OSA, diabetic retinopathy, nephropathy insulin use and White Matter Abnormalities.

Conclusion: This analysis of 942 individuals with diabetes after a lacunar stroke demonstrates a relationship between A1C and change in cognitive scores over time. Intervention studies are needed in order to delineate if better glucose control could slow the rate of cognitive decline in this high risk population.