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NEWS

LONGITUDINAL TRAJECTORIES OF AMYLOID BURDEN AND PLASMA BIOMARKERS IN ALZHEIMER'S DISEASE: A LONGITUDINAL ADNI STUDY

PURPOSE

Obesity is a risk factor for Alzheimer's disease (AD), but its impact on the trajectories of amyloid burden and blood biomarkers (BBMs) of AD is still scarce. This study investigates how baseline obesity relates to the trajectory of amyloid burden alongside corresponding changes in BBM.

METHODS AND MATERIALS

Plasma samples from the Alzheimer's Disease Neuroimaging Initiative (ADNI) were assayed using six leading commercial tests. Amyloid positron emission tomography (PET) scans were used to assess amyloid burden. Spearman's partial correlation was used to assess the baseline cross-sectional association between body mass index (BMI) and BBM levels. Linear mixed-effects regression models were used to study three-way interaction between baseline BBM, time, and obesity.

RESULTS

At the baseline cross-sectional level, BMI was not significantly associated with whole brain amyloid burden. However, longitudinal analysis revealed that participants with baseline obesity exhibited a significantly greater rate of amyloid accumulation over time compared to non-obese individuals. At the baseline cross-sectional level, BMI showed no significant correlation with ptau217 levels measured by C2N or Janssen but had significant negative associations with measurements by Fuji, AlzPath, and the C2N ptau217 ratio. BMI was negatively correlated with Roche and QX plasma NfL and GFAP levels. At the longitudinal level, baseline obesity was not directly associated with longitudinal levels of plasma ptau217 and ptau217 ratio. However, all participants showed increases over time, with those who were obese experiencing significantly faster rates of increase compared to non-obese individuals. In the case of plasma NfL, baseline obesity was not consistently associated with longitudinal changes across different measurement methods. Lastly, baseline obesity status had no significant effect on the longitudinal trajectories of plasma GFAP levels.

CONCLUSIONS

individuals with obesity show faster amyloid accumulation over time despite similar baseline levels. Plasma p-tau217 and its ratio were especially sensitive to these changes. This study provides valuable insights for interpreting amyloid burden BBM levels in response to obesity in AD.

CLINICAL RELEVANCE/APPLICATIONS

The impact of obesity on trajectories of changes in amyloid burden and BBM of Alzheimer's disease is an important aspect to consider implementing these into clinical practice.