OP-07

CORRELATION OF AGGREGATED BETA AMYLOID LEVEL IN PLASMA WITH MoCA AND MMSE AMONG PATIENTS WITH TYPE 2 DIABETES WITH DEMENTIA

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INTRODUCTION
Type 2 DM as a risk factor for Alzheimer's disease (AD) has been studied in recent years; however, no clear evidence of association has been found. As potential biomarker for AD, plasma beta amyloid is likewise under study by researchers. We examined the correlation between plasma beta amyloid levels and cognitive function among type 2 DM patients with dementia as indicated by their neurocognitive assessment scores. This study hopes to devise a less invasive early detection of AD among patients with diabetes.

METHODOLOGY
In this cross-sectional study, 100 patients with type 2 DM and dementia underwent plain cranial CT scan, plasma beta amyloid, MMSE and MoCA. Patients were categorized as having vascular dementia using the NINDS-AIREN Criteria. Elevated plasma beta amyloid was used as biomarker for AD.

RESULTS
Among type 2 DM patients with dementia, there is an increased prevalence of AD (46.7%) as shown by the elevated beta amyloid level. The prevalence of vascular dementia is 6%. Among patients with non-vascular dementia, 51.3% have elevated beta amyloid. There is no significant correlation between both MMSE score and beta amyloid (r=-0.0192, p=0.8557), and between MoCA score and beta amyloid (r=0.0939, p=0.3731). The results do not show significant correlation between MMSE and MoCA scores with beta amyloid level among patients with AD.

CONCLUSION
Using the beta amyloid as biomarker, the study suggests a link between AD and type 2 DM, however, we recommend further researches to ascertain the use of plasma beta amyloid as a less invasive screening for AD among patients with diabetes.

KEY WORDS
diabetes mellitus, dementia, aggregated beta amyloid, Alzheimer's disease

OP-08

ROLE OF HYPOXIA-INDUCIBLE FACTOR 1A (HIF1A) ON INTERMITTENT HYPOXIA-INDUCED ADIPOSE TISSUE DYSFUNCTION IN TYPE 2 DIABETES MELLITUS

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INTRODUCTION
Obstructive sleep apnea (OSA) commonly coexists in type 2 diabetes mellitus (T2DM) patients, but the mechanism for this overlapping epidemic remains unclear. We hypothesized that the intermittent hypoxia (IH) in OSA leads to upregulation of hypoxia-inducible factor 1a (HIF1A) in adipose tissue (AT), leading to local fibrosis, inflammation, and macrophage infiltration. These contribute to insulin resistance and glucose intolerance in T2DM.

METHODOLOGY
We employed a combination of in vitro and in vivo approaches to investigate the role of HIF1A in OSA and T2DM. Cell and animal models were exposed to IH to simulate the hypoxic stress in OSA. The role of HIF1A was investigated through treatment with PX-478, a known HIF1A inhibitor.

RESULTS
IH exposure resulted in IL6-mediated inflammation in adipocytes and macrophage co-culture that was reversed by pre-treatment with PX-478. Further, TallyHo mice treated with PX-478 had markedly improved insulin sensitivity and glucose tolerance after IH challenge. These metabolic improvements were associated with decreased AT fibrosis, inflammation and macrophage infiltration. Trichrome stain indicated that collagen deposition was significantly reduced in AT of PX-478-treated TallyHo mice exposed to IH. We also found that the inflammatory markers IL6, TNFa and MCP1 were decreased in AT of PX-478-treated mice. Consistent with these, immunohistochemical staining confirmed lower frequency of macrophage infiltration in the PX-478 group.