the presence of ApoE4 positive status were adjusted for the comparison. **Results:** Compared with non-diabetes group, executive function (Trail Making Test B) is significant impaired in both the diabetes group and pre-diabetes group. Delayed memory (Rey Auditory Verbal Learning Test) is significantly impaired in the diabetes group. There was significant difference between the three groups in the total left hippocampal volume \( (F=3.257, P=0.046) \) and the left hippocampal tail volume \( (F=4.623, P=0.014) \). There was a significant correlation between the A1c and the volume of the left hippocampal tail \( (R=-0.334, P=0.009) \), bilateral subiculum \( (R=-0.291, P=0.024; R=0.271, P=0.036) \) and bilateral molecular layer \( (R=-0.307, P=0.017; R=-0.283, P=0.028) \) after adjusting for covariants. **Conclusions:** These results support the view that for diabetes, the left posterior hippocampus, especially the hippocampal tail, may be an earlier affected region and then the atrophy process gradually spread to other hippocampal subfields, causing the executive function loss. More clinical attention should be paid in adults with pre-diabetes status for the earlier cognitive impairment prevention.

**P2-418** PRESUBICULUM VOLUME LOSS MAY MEDIATE VASCULAR COGNITIVE IMPAIRMENT IN PRECLINICAL STAGE

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**Background:** Hippocampus subfield volume loss is linked with ageing and Alzheimer’s Disease process. However, less is known for younger adults suffering from vascular risk factors (VRFs) in preclinical dementia and CVD stage. The aim of study is to investigate the effect of total VRFs on hippocampus subfield volume change and clinical cognition impairment. **Methods:** The total burden of VRFs in non-dementia and CVD free middle-aged subjects was assessed by Framingham Cardiovascular risk factor scale and then grouped into low-risk (<10%), medium-risk (10%-20%) and high-risk (>20%) group. A battery of cognitive tests was used to assess various domains of cognitive function. Volumetric analysis was performed using 3D T1-weighted MPRAGE image. Freesurfer 6.0 was used for hippocampus parcellation. Hippocampal subfield volumes were adjusted by intracranial volume. Cognitive tests were transformed to Z-scores. Partial Correlation and Linear Regression Model were used, with age, gender, education years, ApoE4 positive status as covariants. **Results:** After excluding 2 badly segmented subjects, 91 (aged 57.89±5.04) were included in this study. Total Framingham score was significant in the left presubiculum \( (B=-.284, p=.033) \). When specifically looking into the gender, men tended to suffer hippocampus subfield loss in CA1 \( (B=-.399, p=.048) \) and fissure \( (B=-.432, p=.027) \). Low-risk group is significantly better than high-risk group in working memory \( (p=.016) \) and better than medium-risk group in visual attention ability \( (TrailMakingB, p=.042) \) after Bonferroni correction for multiple comparison. **Conclusions:** Presubiculum was significantly impaired with the accumulation of VRFs. Working memory and visual attention were two affected cognitive domains in our younger cohort. Vascular cognitive impairment may suffer another pathology process but still share some common features with Alzheimer’s Disease in preclinical stage.

**P2-419** PROJECTION TO LATENT SPACES DISENTANGLES SPECIFIC CEREBRAL MORPHOMETRIC PATTERNS ASSOCIATED WITH AGING AND PRECLINICAL AD

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**Background:** Partial Least Squares (PLS) is a mathematical technique that relates two sets of observable variables by means of a few latent explanatory factors. The aim of this study was to use PLS to discover the associations between CSF biomarkers and structural brain imaging in preclinical AD and to disentangle their specific contribution from confounding demographic factors. **Methods:** Magnetic Resonance Imaging (MRI-T13D) was acquired from a subset of the ADNI cohort comprising 321 asymptomatic subjects (185 controls, 136 preclinical) for which CSF biomarkers was made publicly available. Each image was preprocessed using FreeSurfer to derive cortical thickness and grey matter volumes in the cortical regions of the Desikan-Killiany parcellation as response variables, and age, education, number of APOE4 alleles and CSF biomarkers as predictors. PLS was used to find the latent space that best describes the variation of predictors, responses and their covariance. The latent space dimension \( (L) \) was chosen as a compromise between the total variance explained and the cross-validated total r-squared coefficient. **Results:** Predictors accounted for a lower amount of variance for cortical thickness vs. grey matter volume, resulting in a lower dimensional latent space in the former \( (L=3) \) and \( (L=5) \). Latent variables showed significant correlation with age and CSF biomarkers, especially with tau measures. Figures 1 and 2 illustrate, respectively, latent processes related to cortical thickness and grey matter volume by coloring brain structures according to their importance. Patterns related to preclinical AD show a combination of cortical shrinkage and expansion. Age-related latent variables show global cortical reduction and atrophy. **Conclusions:** PLS was able to disentangle the cerebral morphometric patterns associated to preclinical AD stages from other demographic factors. Results with both cortical thickness and volumetric data present significant overlap, thus showing the robustness of this approach. Interestingly, volumetric data showed more significant correlations with CSF Aβeta than cortical thickness.

![Figure 1](image.png)

**Figure 1.** Pattern found for each latent variable when using cortical thickness. Latent variable 1, 2 and 3 correspond to (a), (b) and (c) respectively. High positive/negative values are represented in cyan/yellow respectively.
Background: The most common cause of dementia is Alzheimer’s disease (AD) which pathologic process appears as measurable changes in both cerebrospinal fluid (CSF) and imaging biomarkers even a quarter of a century before clinical symptoms. The aim of our study was to assess the usefulness of CSF biomarkers, inter alia Aβ42, tau and p-tau, and imaging biomarkers, such as volumetric atrophy, in detecting the disease in the subclinical phase. Methods: Our study consisted of 191 subjects (94 men) with mean age of 65.7 years (range 42-84) that were selected in neurological examinations, including both CSF biomarkers and MRI examinations, conducted between years 2004-2011 in Kuopio University Hospital. Results: In our cohort 55 subjects were diagnosed with AD after baseline state examination (MMSE) as a tool for screening of cognitive impairment. Application of easy Z score imaging system (eZIS) on BPS images for quantitative evaluation of rCBF has been introduced in Bangladesh in 2017. This study attempted to explore association of MMSE scoring and Z score in patients with clinical likelihood of having Alzheimer’s disease (AD). Methods: Patients with clinical likelihood of having AD on basis of MMSE score who underwent BPS using Tc-99m ethyl cysteinate dimer (ECD) from February to December 2017 were included in this study. eZIS version 3 (Mastuda et al.) was applied to BPS image DICOM data to quantify rCBF. Patients were divided into two groups on basis of MMSE score (group A, mild stage of AD = 20-26 and group B, moderate stage of AD = 10-19). Results: BPS was performed on 34 patients (M/F = 26/8) with clinically suspected AD with mean age of 61.2±10.4 years (41-90) and mean duration since clinical onset of 10.3±4.7 months (6-24). Mean MMSE and mean Z scores were 22.9±1.11 and 1.01±0.35 in group A (n = 18) while 17.56±0.96 and 2.62±0.53 in group B (n = 16). There was negative correlation between MMSE and eZIS score; r = -0.69 in group A (p = 0.002); r = -0.58 in group B (p = 0.019) and r = -0.93 overall (p = 0.000). Conclusions: Quantification rCBF with eZIS score was correlated with clinical MMSE scoring in patients with clinical likelihood of having AD. We propose use of eZIS as an objective adjunct to MMSE for clinicians in major institutes of Bangladesh to aid initiation of appropriate pharmacotherapy in patients with suspected AD.