amyloid had poorer longitudinal memory performance than the nonincreasing group. These effects, though subtle, may represent the earliest consequences of amyloid deposition that are detectable prior to the onset of amyloid positivity and any clinically-relevant cognitive dysfunction.

IC-P-014 PREDICTION OF BETA-AMYLOID POSITIVITY IN MILD COGNITIVE IMPAIRMENT WITH DATA OBTAINED FROM ROUTINE MEMORY CLINIC PRACTICE

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Background: Although recent advances in neuroimaging techniques have enabled the detection of in vivo Alzheimer's disease (AD) pathology in human, only a limited number of memory clinics are able to utilize it in clinical practice due to high cost and low accessibility. This study, therefore, aimed to develop prediction models for the beta-amyloid (Aβ) positivity on amyloid positron emission tomography (PET) in mild cognitive impairment (MCI) individuals with data that are routinely obtained in memory clinic setting. **Methods:** Sixty seven MCI patients were included in this study. All subjects received clinical and neuropsychological assessments, laboratory evaluations for blood sample, magnetic resonance imaging, and ^{11}C-labelled Pittsburgh Compound B (PiB) PET, For the development of Aβ positivity on PiB PET prediction models, all the variables were first categorized into four groups: clinical (C), neuropsychological (N), laboratory (L), and imaging (I) groups. In each group, the variables that showed significant or trend level association with Aβ positivity in univariate analyses were selected for further analyses. The selected variables of each group were combined sequentially in the prediction models, and logit values were calculated. Finally, with the logit values, the receiver operating characteristic (ROC) analyses were performed for each model to calculate the area under the curve (AUC). **Results:** For each group, following variables were selected: Total scores of geriatric depression scale, subjective memory complaint questionnaire, trait anxiety, blessed dementia scale-activities of daily living and history of hypertension for group C; raw scores of word list recall and recognition, and constructional recall for group N; triiodothyronine, high-density lipoprotein cholesterol, erythrocyte sedimentation rate, and APOE 4 positivity for group L; adjusted hippocampal volume for group I. In the ROC analyses, AUC for the prediction models were as follows: 0.793 for group C, 0.894 for combined group C+N, 0.956 for combined group C+N+L, and 0.944 for combined group C+N+L+I. **Conclusions:** The findings suggest that the systematic combinations of data obtained from routine clinical practice may be successfully used to predict Aβ positivity in MCI individuals.

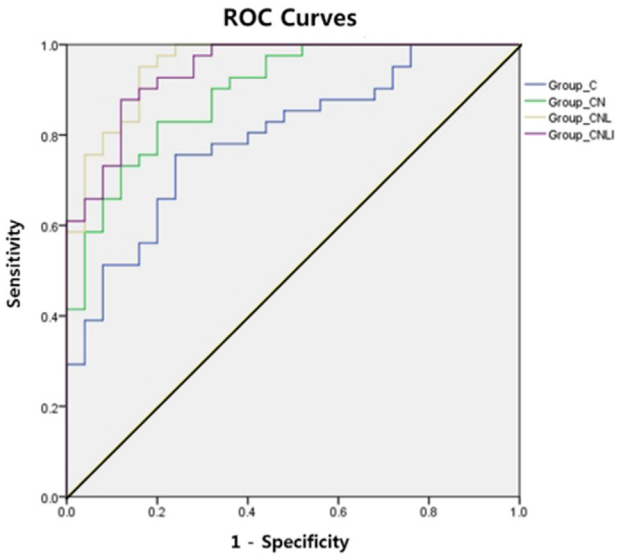

Figure 1. The receiver operating characteristic (ROC) curves of each prediction model for amyloid positivity.

Table 1
Comparison of the area under the ROC curves of each prediction model

Model	AUC	SD	p	95% CI
Group C	0.793	0.055	<0.001	0.685-0.901
Group C+N	0.894	0.039	<0.001	0.818-0.969
Group C+N+L	0.956	0.023	<0.001	0.910-1.000
Group C+N+L+I	0.944	0.026	<0.001	0.593-0.996

Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; SD, standard deviation; CI, confidence interval.

IC-P-015 ASSOCIATION OF PAST AND CURRENT BODY MASS INDEX WITH BRAIN AMYLOID DEPOSITION AND NEURODEGENERATION IN COGNITIVELY NORMAL ELDERLY

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Background: Both low and high body mass index(BMI) has been associated with cognitive impairment and Alzheimer disease(AD) dementia. Nevertheless, very little information is available for the association between BMI over life course and brain amyloid beta(Aβ) burden and AD-specific neurodegeneration. In this study, we examined the relationship of past and current BMI with in vivo cerebral Aβ deposition and AD-signature region cortical thickness in cognitively normal elderly population. **Methods:** Two-hundred twelve cognitively normal elderly subjects aged