

Poster presentation

## High plasma Amyloid $\beta$ 42 and P-tau in mild cognitive impairment as risk factors of the disease

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### Background

Patients with mild cognitive impairment (MCI) is reported to develop Alzheimer's disease (AD) at the rate of 12% per year, greatly exceeding the 1% to 2% incidence of normal controls. Several studies have shown an increase in plasma A $\beta$ 42 in MCI compared to normal and AD patients. The efficiency of A $\beta$  peptides elimination in earlier stages of AD has proven in animal models. We found no study measuring phospho-tau (p-tau) level in plasma.

### Materials and methods

We measured the plasma level of A $\beta$ 42 and p-tau181 in 7 patients with MCI, 29 AD and 16 normal controls who had also underwent brain SPECT imaging.

### Results

Plasma levels of A $\beta$ 42 and p-tau were significantly higher in MCI (57.9 $\pm$ 33.3 pg/ml) (44.5 $\pm$  91.5pg/ml) comparing AD (16.3 $\pm$ 15.5pg/ml) (3.4 $\pm$ 10.7pg/ml) and normal group (12 $\pm$ 7.7pg/ml) (00 pg/ml) (p<0.000) (p<0.010) respectively.

P-tau was not detectable in normal group but p-tau was detectable in (57%) (4/7) of patients with MCI and 4 patients with AD. 3 patients with MCI who had high plasma A $\beta$ 42 and detectable p-tau too, had shown bilateral

Posterior temporoparietal hypoperfusion and one showed not-characteristic perfusion defects in SPECT.

### Conclusions

Since high plasma A $\beta$ 42 and p-tau in our patients with MCI were accompanied by perfusion defect characteristic of AD which is said to be a sign of the progression of MCI to AD, we suggest the evaluation of plasma A $\beta$ 42 and p-tau as the risk factors of the disease in patients with MCI.