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<td><strong>Author(s)</strong></td>
<td>Zhou, L; Chu, LW; Kwan, JSC; Lam, KSL; Ho, PWL; Ho, JWM; Chan, KH</td>
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<tr>
<td><strong>Citation</strong></td>
<td>The 16th Medical Research Conference, Department of Medicine, The University of Hong Kong, Hong Kong, 22 January 2011. In Hong Kong Medical Journal, 2011, v. 17 suppl. 1, p. 69, abstract no. 118</td>
</tr>
<tr>
<td><strong>Issued Date</strong></td>
<td>2011</td>
</tr>
<tr>
<td><strong>URL</strong></td>
<td><a href="http://hdl.handle.net/10722/142835">http://hdl.handle.net/10722/142835</a></td>
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Plasma amyloid beta peptides and oligomers levels in Alzheimer’s disease

L Zhou1,2, LW Chu2, JSC Kwan1, KSL Lam1,2,3, PLW Ho1,2, JWM Ho1, KH Chan1,2,3
1Department of Medicine, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong
2Hong Kong University Alzheimer’s Disease Research Network, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong
3Research Centre of Heart, Brain, Hormone and Healthy Aging, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong

Introduction: Amyloid beta (Aβ) exists in different forms including Aβ peptides, oligomers, protofibrils, and fibrils. It has been believed that Aβ fibrils in Alzheimer’s disease (AD) brain contribute to AD pathogenesis, but recent evidences suggest that Aβ fibrils have stronger relationship with AD pathogenesis.

Aims: To study the plasma Aβ40, Aβ42, and Aβ oligomers levels in AD patients and non-demented age-matched controls, and the correlations between plasma Aβ oligomers levels and cognitive function.

Methods: We studied 44 AD patients and 22 controls. Cognitive functions were assessed by cognitive assessment tools: Chinese version of mini-mental state examination (MMSE), Abbreviated Metal Test (AMT), Alzheimer’s Disease Assessment Scale Cognitive Subscale (ADAS-cog), and Delayed 10-Word Recall Test (DWRT). Plasma Aβ40 and Aβ42 levels were measured by ELISA kits (Invitrogen). Plasma Aβ oligomers level was detected by ELISA, using Aβ42 oligomeric antibody as detecting antibody and Aβ N-terminal antibody (against residues 1-14) as capturing antibody.

Results: There was no difference in plasma Aβ40 and Aβ42 levels between AD patients (median Aβ40 level 145.93 pg/mL, median Aβ42 level 9.94 pg/mL) and controls (median Aβ40 level 130.34 pg/mL, median Aβ42 level 8.42 pg/mL; P=0.196 and P=0.187, respectively). In women with AD, increased plasma Aβ42 level was associated with increased MMSE (P=0.043) and decreased ADAS-cog (P=0.034) suggestive of positive correlation between plasma Aβ42 level and cognitive function. Plasma Aβ oligomers level was higher in AD patients (median 642.54 ng/mL, range 103.33-2676.93 ng/mL) than controls (median 444.18 ng/mL, range 150.19-1311.18 ng/mL; P=0.047), and was negatively correlated with cognitive function evidenced by increased plasma Aβ oligomers level associated with decreased MMSE, AMT, DWRT scores and increased ADAS-cog scores (P=0.037, P=0.043, P=0.025, P=0.036, respectively).

Conclusion: Plasma Aβ42 and Aβ40 levels are not suitable biomarkers for AD diagnosis; but plasma Aβ oligomers level may reflect severity of cognitive impairment in women with AD. Plasma Aβ oligomers level may help diagnose AD patients, but the range is wide among AD patients, making it not an ideal biomarker for AD diagnosis.

Plasma amyloid beta peptides and oligomers antibodies in Alzheimer’s disease

L Zhou1,2, LW Chu2, JSC Kwan1, KSL Lam1,2,3, PLW Ho1,2, JWM Ho1, KH Chan1,2,3
1Department of Medicine, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong
2Hong Kong University Alzheimer’s Disease Research Network, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong
3Research Centre of Heart, Brain, Hormone and Healthy Aging, LKS Faculty of Medicine, The University of Hong Kong, Hong Kong

Introduction: Various forms of amyloid beta (Aβ) including Aβ peptides, oligomers, protofibrils and fibrils are thought to be pathogenic in Alzheimer’s disease (AD). The exact pathophysiological role of endogenous Aβ autoantibodies (Ab) in healthy subjects and AD patients are uncertain. Potential protective role of Aβ Ab has been suggested.

Aims: To study the serum Aβ monomers and Aβ oligomers Ab levels in AD patients and non-demented age-matched controls, and the relationship between Aβ monomers and Aβ oligomers Ab levels and cognitive function.

Methods: A total of 44 AD patients and 22 controls were recruited. Cognitive functions were assessed by cognitive assessment tools: Chinese version of mini-mental state examination (MMSE), Abbreviated Metal Test (AMT), Alzheimer’s Disease Assessment Scale Cognitive Subscale (ADAS-cog). Aβ Ab levels were assayed by ELISA. Aβ oligomers Ab levels were assayed by ELISA. Aβ monomer and Aβ oligomers Ab levels were coated on 96-well plates for measuring Aβ monomer Ab and Aβ oligomers Ab respectively. The secondary antibody was rabbit-anti-human antibody (detecting antibody). Calibration curves were made by 2C8 (against Aβ residues1-16) and 7A1a (Aβ oligomers antibody) for Aβ monomer Ab and Aβ oligomers Ab respectively. Negative control was incubating with secondary antibody and Aβ N-terminal antibody (against residues 1-14) without incubation with patients/controls’ serum.

Results: There was no difference in serum Aβ monomer Ab level between AD patients (median 177.43 μg/mL) and controls (median 190.88 μg/mL, P=0.55). In AD patients, Aβ monomer Ab level was negatively correlated with cognitive function evidenced by increased Aβ monomer Ab level associated with decreased MMSE, AMT and increased ADAS-cog scores (P=0.004, P=0.013, P=0.005, respectively). Serum Aβ oligomers Ab level was higher in AD patients (median 42.81 μg/mL, range 11.91-241.62 μg/mL) than controls (median 24.15 μg/mL, range 2.32-329.93 μg/mL; P=0.014).

Conclusion: Serum Aβ monomer Ab is not a suitable biomarker for AD diagnosis, but may reflect the severity of AD. Serum Aβ oligomers Ab level may help in AD diagnosis, but wide range of titers among AD patients makes it not an ideal biomarker for AD diagnosis.