RESEARCH ARTICLE

Serum β-amyloid 1–42 Levels as Alternative Non-invasive Screening Biomarker for Alzheimer's Disease and Vascular Dementia in Indonesian Elderly Population

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Abstract

BACKGROUND: Alzheimer's disease (AD) and vascular dementia (VaD) impose a substantial public health burden in Indonesia; however, accessible blood-based biomarkers for early screening remain limited. Although cerebrospinal fluid β-amyloid 1–42 is an established biomarker, its invasive nature restricts its use for population-level screening. Therefore, it is necessary to have locally-produced serum β-amyloid 1–42 ELISA kit that is specifically designed for Indonesian elderly population. In this study, a locally-produced β-amyloid 1–42 ELISA kit was validated and used for the screening of AD, VaD and mild cognitive impairment (MCI) Indonesian population.

METHODS: A cross-sectional study including 166 subjects: 31 AD, 34 VaD, 34 MCI patients, and 67 cognitively normal controls was conducted. All participants underwent cognitive assessments including Mini Mental State Examination (MMSE) and Montreal Cognitive Assessment-Indonesian version (MoCA-Ina), as well as brain magnetic resonance imaging (MRI) 3-Tesla for the assessment of medial temporal atrophy/white matter changes. Fasting venous blood sampling was taken from each subjects for the measurement of serum β-amyloid 1-42 measurement using locally-produced ELISA kit.

RESULTS: Median serum β-amyloid 1–42 levels were 11.03, 10.99, and 10.99 pg/mL for the AD, VaD, and MCI subjects, respectively. The β-amyloid 1–42 levels were correlated with MMSE scores in all group (AD: r=−0.455, p=0.010; VaD: r=−0.419, p=0.014; MCI: r=−0.412, p=0.015). The validity analysis of the locally-produced serum β-amyloid 1–42 ELISA kit, showed sensitivity of 94.12% (95% CI: 87.3–97.9), specificity of 80.36% (95% CI: 72.4–86.8), and diagnostic accuracy of 83.56% (95% CI: 77.2–88.5).

CONCLUSION: Serum β-amyloid 1–42 levels are lower in AD and VaD subjects compared to MCI and control subjects. Serum β-amyloid 1-42 is inversely correlated with cognitive function across all groups based on MMSE score. Additionally, the locally-produced β-amyloid 1-42 ELISA kit demonstrated sensitivity of 94.12% and specificity of 80.36%, meeting Global CEO Initiative Consensus for pre-screening tools, supporting its potential as a scalable, non-invasive screening biomarker in Indonesian primary care settings.

KEYWORDS: G-banding karyotyping, next generation sequencing, non-invasive prenatal testing

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Introduction

Alzheimer's disease (AD) and vascular dementia (VaD) constitute the majority causes of dementia in elderly populations, imposing considerable public health burden globally and in Indonesia.(1-3) Around 60–70% of the people suffering from neurodegenerative diseases are having AD.(4) Despite their distinct etiologies, AD and VaD frequently coexist, particularly in elderly populations, leading to overlapping clinical manifestations that complicate accurate diagnosis.(1,2) Mild cognitive impairment (MCI) represents an intermediate clinical stage between normal aging and dementia, characterized by measurable cognitive decline with preserved functional independence.(4) Importantly, individuals with MCI are at increased risk of progression to AD or VaD, highlighting the need for reliable biomarkers to support early detection and differential diagnosis.(5-7)

AD is characterized by progressive neurodegeneration, primarily driven by β-amyloid plaque accumulation and tau pathology (8), whereas VaD results from cerebrovascular damage that disrupts cerebral blood flow and neural networks. Research attempting to establish the reliability of β-Amyloid and tau as biomarkers has culminated in an amalgamation of contradictory results and theories regarding the biomarker concentrations necessary for an accurate diagnosis.(1,9-13) β-Amyloid 1-42, a cleavage product of amyloid precursor protein (APP), is central in AD pathology.(14) The β-Amyloid 1-42 level is traditionally measured via cerebrospinal fluid (CSF), which is highly accurate, but also very invasive, hence restricting its use for population-level screening. Therefore, it is necessary to have blood-based assessment, particularly with cost-efficient, for more scalable population screening.

Some studies in various country reported that serum/plasma β-Amyloid 1-42 could be used as a core biomarker in progressive cognitive impairment, though local validation is needed to ensure clinical translation.(1,15-17) Despite the established role of β-amyloid biomarkers in AD pathology, several critical gaps remain for Indonesian populations. Since most validation studies performed in Indonesia utilize expensive commercial enzyme-linked immunosorbent assay (ELISA) kits (such as Simoa™ or Lumipulse™), making it inaccessible for routine screening in low-resource settings. In addition, serum/plasma β-amyloid level might varies across ethnicities due to genetic polymorphisms (ApoE genotype), dietary patterns, and comorbidity profiles, hence population-specific validation is necessary.(15-17)

In our prior study, we tried to develop a locally-produced β-Amyloid 1-42 ELISA kit to bridge the gap mentioned above. The ELISA kit was developed following standard immunoassay development protocols and tailored significantly for Indonesian populations.(18) Therefore, in this study, the locally-produced β-amyloid 1–42 ELISA kit was used for comparing the serum levels across AD, VaD, and MCI patients to elucidate biomarker performance in mixed dementia pathology in Indonesian elderly; and was validated for benchmarking diagnostic accuracy against Global CEO Initiative Consensus Criteria (≥90% sensitivity, ≥75% specificity) for pre-screening tools.(19)

Methods

Study Design

A cross-sectional study was conducted involving 99 elderly patients (including 31 AD, 34 VaD, and 34 MCI patients) from the National Brain Center Hospital, Jakarta and 67 cognitively normal elderly as controls that were enrolled from two primary care centers in Pasar Minggu and Matraman, Jakarta. All subjects included aged ≥50 years old. AD subjects were included if they have cognitive deficit defined by Mini Mental State Examination (MMSE) score <24 or Montreal Cognitive Assessment-Indonesian version (MoCA-Ina) scores <18, as well as if the magnetic resonance imaging (MRI) confirmation showed medial temporal and hippocampal atrophy.(20) VaD subjects were included if they have cognitive deficit defined by MMSE score <25 or MoCA-Ina scores <23, as well as MRI confirmation showed white matter change. Meanwhile, MCI subjects were included if they have subjective memory complaint, had objective cognitive impairment on neuropsychological testing based on MMSE score of 24–26 or MoCA-Ina of 18–25, preserved activities of daily living; and absence of dementia based on DSM-5 criteria. Subjects were excluded if they had neuropsychiatric disorders, unstable medical condition, or medication interfering with cognition. All subjects provided informed consent to take part in the study, and the study protocol was approved by the Institutional Review Board of the National Brain Center Hospital (Approval No. DP.04.03/D. XXIII.9/130A/2023).

MMSE and Moca-Ina Assessment

The cognitive deficit of each subject was assessed using validated Indonesian version of MMSE and MoCA-Ina (adjusted for education). Both tests assessed cognitive

function, particularly in predicting AD, VaD, and MCI based on particular score as mentioned in previous sub-section. MMSE consists of 30 questions and evaluated five cognitive domains, including orientation, registration, attention and calculation, recall, as well as language and visuospatial ability. Meanwhile, Moca-Ina assessed 7 cognitive dimensions including visuospatial and executive function, naming, attention, language, abstraction, delayed recall (memory), and orientation with maximum score of 30. The level of education were grouped into three group, low level for elementary school, middle group for junior high school and senior high school and high group for minimum bachelor degree. An education adjustment of +1 point was applied for individuals with ≤12 years of formal education.

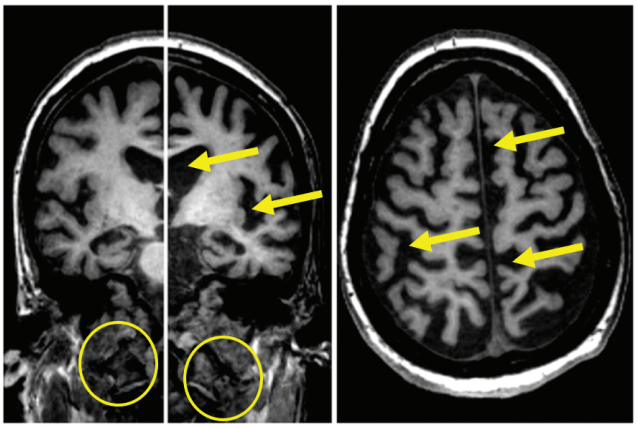

Figure 1. Brain MRI of AD subjects. Weighted sequences assessing medial temporal and hippocampal atrophy in Alzheimer's disease. Yellow arrow: medial temporal atrophy. Yellow circle: hippocampal atrophy.

Brain MRI Confirmation

To complete the neurobehavioral test, other than cognitive function assessment brain neuroimaging was also performed by three-tesla (3T) brain MRI to exclude structural lesions and assess early neurodegenerative changes. In this current study, the confirmation of AD was determined if medial temporal and hippocampal atrophy was assessed by the 3T MRI scan T1-weighted (Figure 1). Meanwhile, the confirmation of VaD was determined white matter hyperintensities/changes was assessed 3T MRI scan T1-flair (Figure 2).

β-Amyloid 1-42 Measurement

Five mL of fasting blood samples were collected from each subject via venipuncture, allowed to clot 30-60 minutes, centrifuged at 2,000 g for 10 min, aliquoted and then stored at −80°C. A maximum of two freeze-thaw cycles were permitted. Using the collected blood sample, the measurement of serum levels of β-Amyloid 1-42 was performed using our previously developed locally-produced β-amyloid 1–42 ELISA kit (Catalog No. PSP-AB42-001, Pusat Studi Satwa Primata IPB University, Bogor, Indonesia), which was developed following standard immunoassay development protocols. As an of β-Amyloid 1-42 peptide detection kit, this ELISA kit had been compared for its detection capabilities with several commercial of β-Amyloid 1-42 ELISA kits, and have been reported to show no statistically significant differences compared to either the of β-Amyloid 1-42 standard solution or the results of previous samples.(18)

The microplate wells was coated with capture antibodies specific for β-Amyloid 1-42, and incubated with blood samples. After incubated, the wells were washed to remove unbound components, and enzyme-conjugated detection antibodies were added. A substrate solution was also added to produce a colorimetric reaction proportional to the β-Amyloid 1-42 concentration. After the reaction stopped, the optical density (OD) was measured at the specified wavelength using the RT-6500 Microplate Reader (Rayto, Shenzhen, China). All procedures adhered strictly to the manufacturer's protocol. Calibration curves and quality controls were used to quantify the β-Amyloid-42 levels in serum samples accurately. The standard curve exhibited excellent linearity ($R^2 = 0.993$, $y = 277.46x + 0.7019$) across the concentration range of 0.5–25 pg/mL, with coefficients of variation for standard replicates consistently below 10%. Intra-assay precision was confirmed by duplicate measurements yielding mean CV values under 15%, while inter-assay reproducibility was maintained with pooled serum controls resulting in inter-plate CV below 20%.

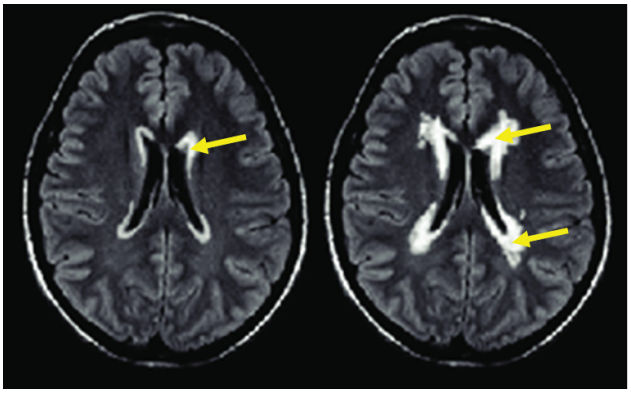

Figure 2. Brain MRI of VaD subjects. Flair sequences evaluating white matter hyperintensity in VaD subjects. Yellow arrow: white matter hyperintensities.

Statistical Analysis

Statistical analysis was performed using R v4.4.1 (R Core Team, Vienna, Austria). Mean comparison between groups were analyzed with T-test or ANOVA depends on the normality of the variables. Diagnostic accuracy (sensitivity, specificity, positive/negative predictive value, likelihood ratios) of the locally-produced β-amyloid 1–42 ELISA kit was also calculated and confirmed by ROC curve with AUC and optimal cut-off (Youden index). A $p<0.05$ was deemed statistically significant.

Results

Subjects' Characteristics

A total of 166 subjects were included in this current study, including 31 subjects with AD, 34 subjects with VaD, 34 subjects with MCI, as well as 67 cognitively normal subjects as controls. There were significant differences in the age between subjects, with MCI subjects whose had the lowest median of 53 years old, followed by subjects with AD (62 years old), VaD (68 years old), and control subjects (69 years old). Eventhough the age requirement for this study was ≥50 years old, however the age range for the MCI subjects group was lower, causing lower median age for the MCI subjects. Subjects with higher education dominating the MC, AD, and control subjects; while in VaD subjects most subjects had middle level education ($p=0.028$). Cognitive performance, assessed by MMSE and MoCA-Ina, showed expected severity gradients, with AD subjects exhibiting the lowest scores in MMSE score, while VaD subjects exhibiting the lowest scores in MoA-Ina score (Table 1).

Serum β-Amyloid 1–42 Levels Were Lower in AD and VaD Subjects

Serum β-amyloid 1–42 level was significantly different among all group ($p<0.001$) with lowest median found in VaD and AD subjects, followed by higher median found in MCI subjects. Meanwhile, the control subjects demonstrated highest median serum β-amyloid 1–42 levelthan the other three groups, showing that β-Amyloid 1-42 was higher in normal elderly compared to elderly with cognitive impairment (Table 2). The overall range of β-amyloid 1–42 level across all groups was 1.32–23.71 pg/mL, with an overall mean of 11.15±4.83 pg/mL. These values demonstrate a clear downward shift in β-amyloid 1–42 level among AD and VaD compared to MCI and control groups.

Further analysis of serum β-amyloid 1–42 levels based on gender, age group, and education level was performed on the AD, VaD, and MCI subjects cumulatively. However, there were no significant different between male and female, also among the age group ($p>0.05$). However, the serum β-amyloid 1–42 levels was rising as the higher education level, with median level of 2.66, 10.99, and 11.20 pg/mL for low, middle, and high education, respectively (Table 3). These demographic patterns suggest that education level might serve as important covariates in interpreting serum β-amyloid 1–42 levels and should be considered in clinical screening applications.

Correlation between β-amyloid 1–42 Levels and Cognitive Scores

Among the AD, VaD, and MCI subjects, β-amyloid 1–42 levels showed significant negative correlations with MMSE scores, with correlation coefficients of –0.455 for AD subjects, –0.419 for VaD subjects, and –0.412 for

Table 1. Baseline demographic and clinical characteristics.

Characteristic	AD (n=31)	VaD (n=34)	MCI (n=34)	Controls (n=67)	*p* -value
Demographics					
Age (years), median (IQR)	62 (61-80)	68 (61-80)	53 (50-80)	69 (65-75)	<0.001*
Female, n (%)	17 (54.8)	25 (73.5)	21 (61.8)	42 (62.7)	0.345
Education Level, n (%)					0.028*
Low (elementary)	5 (16.1)	1 (3.0)	0 (0.0)	3 (4.5)	
Middle (junior/senior high)	9 (29.0)	19 (55.9)	8 (23.5)	18 (26.9)	
High (≥bachelor)	17 (54.8)	15 (44.1)	26 (76.5)	46 (68.7)	
Cognitive Assessment					
MMSE, median (range)	16 (11-21)	20 (11-21)	23 (11-24)	26 (25-27)	<0.001*
MoCA-Ina, median (range)	22 (11-24)	17 (11-21)	20 (11-21)	N/A	0.002*
Memory decline, n (%)	26 (83.9)	26 (76.5)	26 (76.5)	0 (0.0)	<0.001*

Tested with ANOVA, *$p<0.05$ considered significant.

Table 2. β-amyloid 1–42 levels among all groups.

β-Amyloid 1-42 (pg/mL)	AD (n=31)	VaD (n=34)	MCI (n=34)	Controls (n=67)	*p* -value
Mean±SD	9.78±4.44	9.95±4.12	13.59±4.98	15.24±3.85	<0.001*
Median	11.03	10.99	13.72	15.30	

Tested with ANOVA, *p<0.05 considered significant.

MCI subjects. In contrast, no significant correlations were observed between β-amyloid 1–42 levels and MoCA-Ina scores in AD, VaD, or MCI groups (Table 4).

Logistic Regression Analysis for Risk Prediction of AD and VaD

To further assess whether serum β-amyloid 1–42 could independently predicts AD and VaD after accounting for demographic and cognitive factors, a multivariable logistic regression analysis was performed to compare dementia (AD + VaD) *vs.* non-dementia (MCI + controls). After adjustment for age, sex, and education level, lower β-amyloid 1–42 concentrations remained significantly associated with higher odds of dementia (AD and VaD subjects). Age and MMSE score were also strong predictors, while gender did not contribute significantly (Table 5). Even after adjusting for age and education, lower β-amyloid 1–42 remained a significant predictor of dementia, supporting its validity as a biomarker beyond demographic confounders.

Diagnostic Accuracy

The locally-produced β-amyloid 1–42 ELISA kit demonstrated strong diagnostic performance, with a sensitivity of 94.12% (95% CI: 87.3–97.9), specificity of 80.36% (95% CI: 72.4–86.8), and an overall accuracy of 83.56% (95% CI: 77.2–88.5). These metrics meet the performance thresholds recommended by Global CEO Initiative Consensus Criteria that mentioned dementia biomarker should have ≥90% sensitivity, ≥75% specificity for a screening tool, indicating that serum β-amyloid measurement using this assay has clinically acceptable discriminatory capability.

Table 3. β-amyloid 1–42 levels based on gender, age group, and education level on AD, VaD, and MCI subjects.

Category	n	Median (Min-Max)
Gender		
Male	63	11.05 (1.86-23.71)
Female	36	11.01 (1.32-19.38)
Age Group		
<65 years	50	11.05 (2.66-22.33)
65–74 years	28	11.04 (1.86-23.71)
75–84 years	12	11.01 (1.32-14.01)
≥85 years	9	10.48 (2.66-15.39)
Education Level		
Low	5	2.66 (1.32-19.38)
Middle	36	10.99 (1.86-22.34)
High	58	11.20 (2.40-23.71)

Discussion

This study evaluated the diagnostic utility of a locally produced β-amyloid 1–42 ELISA kit for distinguishing dementia (AD and VaD) from MCI and cognitively normal older adults in Indonesia. Consistent with established pathological models, serum β-amyloid 1–42 levels were markedly lower in AD and VaD groups compared with MCI and controls, supporting its relevance as a peripheral biomarker reflective of underlying neurodegenerative processes.(14-17) Despite greater biological variability in serum compared with CSF analytes, the current findings indicate that serum β-amyloid 1–42 retains discriminatory value across diagnostic categories.(11-13)

In current study, it was found that the median β-amyloid 1–42 level in AD group was 11.03 pg/mL and in VaD group was 10.99 pg/mL. These values are comparable with international reports, which generally describe mean serum β-amyloid 1–42 concentrations of approximately 10–15 pg/mL in cognitively normal older adults and 8–12 pg/mL in patients with AD (11-13), suggesting that the observed levels are consistent with prior studies.

An additional finding was the differential association between serum β-amyloid 1–42 and cognitive measures. Inverse significant correlations were observed with MMSE, but not with MoCA-Ina. Although MoCA-Ina is more sensitive to early cognitive impairment, particularly in executive and visuospatial domains, MMSE may better capture global cognitive decline related to amyloid-driven neurodegeneration, especially memory and orientation

Table 4. Correlation analysis between β-Amyloid 1-42 levels and cognitive test scores in AD, VaD, and MCI subjects.

Group	(r, *p* -value)	
	β-Amyloid 1-42 *vs* MMSE	β-Amyloid 1-42 *vs* MoCA-Ina
AD	r=-0.455, *p* =0.010	r=-0.201, *p* =0.278
VaD	r=-0.419, *p* =0.014	r=-0.137, *p* =0.440
MCI	r=-0.412, *p* =0.015	r=-0.000, *p* =0.999

Tested with Spearman's Rho (rank correlation), *$p<0.05$ considered significant.

deficits characteristic of AD.(4,21) Since MMSE predominantly measures global cognitive impairment and memory-related functions, which are more directly associated with amyloid-driven neurodegeneration. Conversely, MoCA-Ina places greater emphasis on executive function, visuospatial ability, and abstraction, domains that are influenced by heterogeneous mechanisms beyond the amyloid pathology, including vascular changes and educational effects. Therefore, serum β-amyloid 1–42 levels may correlate more strongly with MMSE than with MoCA-Ina, particularly in Indonesian elderly populations.

The assay demonstrated strong diagnostic performance, with a sensitivity of 94.12% (95% CI: 87.3–97.9), specificity of 80.36% (95% CI: 72.4–86.8), and accuracy of 83.56% (95% CI: 77.2–88.5). These metrics meet international performance thresholds for first-tier screening biomarkers.(19) While CSF β-amyloid 1–42 remains the gold-standard biomarker, with established cut-off values such as <192 pg/mL for AD positivity (2), serum testing might offers important practical advantages. Venipuncture is minimally invasive, widely accessible, and substantially less costly than lumbar puncture or positron emission tomography (PET) imaging, making serum β-amyloid 1–42 particularly suitable for large-scale or primary-care, based screening in Indonesia.(15-17)

Nevertheless, since serum β-amyloid 1–42 is influenced by peripheral metabolism, blood–brain barrier transport, and non-neuronal sources such as platelets, this may reduce brain specificity and contribute to inter-individual variability.(22,23) Measuring the β-amyloid 1–42/β-amyloid 1–40 ratio may improve diagnostic precision by normalizing total amyloid production and should be considered in future studies.(15-17,19) Despite these limitations, the diagnostic accuracy achieved in this study is comparable to that reported for CSF biomarkers in some cohort studies (2,10-12,22), supporting the potential clinical utility of serum β-amyloid 1–42 as a pre-screening tool.

Eventhough in our previous study, this locally-produced serum β-amyloid 1–42 ELISA kit was compared to 3 commercially available ELISA kits (18), however in current study there are lack of direct comparison with other ELISA kits or confirmatory CSF or PET biomarkers due some constraints. Potential confounders, including ApoE genotype, comorbidities, medications, and inflammatory markers, were also not assessed. Therefore, a longitudinal study incorporating expanded biomarker panels, such as the β-amyloid 1–42 /β-amyloid 1–40 ratio and the β-amyloid 1–42 /β-amyloid 1–40 ratio, and consideration of various confounders are needed to establish the predictive and monitoring value of serum Aβ1–42 in Indonesian populations.(19,21)

Conclusion

Serum β-amyloid 1–42 levels are lower in AD and VaD subjects compared to MCI and cognitively normal elderly subjects. Serum β-amyloid 1-42 is inversely correlated with cognitive function across all groups based on MMSE score. Additionally, the locally-produced β-amyloid 1-42 ELISA kit demonstrated sensitivity of 94.12% and specificity of 80.36%, meeting Global CEO Initiative Consensus for pre-

Table 5. Multivariable logistic regression for dementia vs. MCI/controls.

Predictor	Odds Ratio (OR)	95% CI	*p* -value
β-amyloid 1–42 (per 1 pg/mL decrease)	1.18	1.08–1.29	0.001*
Age (per 10-year increase)	2.34	1.56–3.51	<0.001*
Female gender	1.45	0.68–3.09	0.335
High education (ref: low/middle)	0.42	0.19–0.93	0.032
MMSE score (per 1-point decrease)	1.25	1.12–1.39	<0.001*

*$p<0.05$ considered significant.

screening tools, supporting its potential as a scalable, non-invasive screening biomarker in Indonesian primary care settings.

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Authors Contribution

IAP contributed to study conception, design, data collection, data analysis, and manuscript drafting. HSD contributed to methodology and critical manuscript revision. MH and PP contributed to data collection, statistical support, and manuscript drafting. JN contributed to neuroimaging interpretation and critical revision. BU and YK contributed to methodology. All authors reviewed and approved the final version of the manuscript.

Conflict of Interest

The authors declare no conflicts of interest or competing interests related to the content of this manuscript.

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