Double-edged blinde, hemorrhagic or cardioembolic cognitive impairment

Levent Cerit*, Hatice Kemal, Aziz Günsel, Hamza Duygu

Department of Cardiology, Near East University, Nicosia, Cyprus

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To the Editor

I have read the article entitled “Cognitive function and adherence to anticoagulation treatment in patients with atrial fibrillation” by Jankowska-Polańska, et al.[1] with great interest. The investigators reported that cognitive impairment is an independent determinant of compliance with pharmacological therapy in elderly patients with atrial fibrillation (AF). Lower adherence, beyond the assessment of cognitive function, is related to the age of patients.[1]

AF is associated with late-life dementia.[2] This association is not only seen in vascular dementia, but also in Alzheimer’s disease (AD). It has been reported that AF may be associated with higher risk of cognitive function impairment. One important possible explanation is the increased incidence of stroke. Ott, et al.[3] have demonstrated that even among AF patients without stroke, AF is still associated with dementia and AD. Both vascular dementia and AD had been reported to be associated with AF.

CHADS2 score (congestive heart failure, hypertension, age ≥ 75 years, diabetes mellitus, stroke) has been reported to be of used for risk stratification regarding stroke recurrence in patients with AF, and also to predict AF recurrence after ablation and predict AF complications among AF patients.[4] Chou, et al.[5] reported that CHADS2 score is a useful predictor for the development of vascular dementia as well as AD in patients with AF.

In this context, AF is associated with an increased risk of impaired cognitive function. In this study, adherence to anticoagulation therapy and relationship between cognitive functions are evaluated and correlation of results with CHADS2 score may be beneficial.

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*Correspondence to: drcerit@hotmail.com

Authors’ reply

Beata Jankowska-Polańska1, Katarzyna Lomper2, Izabella Uchmanowicz3, Krzysztof Dudek2, Joanna Jaroch3

1Department of Clinical Nursing, Wrocław Medical University, Bartla, Wrocław, Poland
2Department of Logistic and Transport Systems, University of Technology, Wybórzew Stanisława Wyspiańskiego, Wrocław, Poland
3Emergency Medicine Centre, Cardiology Unit, Marcinick Lower-Silesian Specialist Hospital, Fieldorfa, Wrocław, Poland

We appreciated much for Dr. Cerit’s comments on our article. Evidence from meta-analysis conducted by Kalan-
tarian, et al.,[1] has suggested that AF is associated with a high risk of cognitive impairment (CI) and dementia (D), with or without a history of clinical stroke. Several mechanisms have been considered for the relationship of AF and CI. One explanation is the presence of the same risk factors such as hypertension, heart failure, diabetes mellitus in the both of conditions. Another potential mechanism is that AF comprises all the components of Virchow’s triade (hyper-coagulable state in AF, stasis of blood in remodeled left atrium and structural injury of the heart) that leads to thrombus formation in left atrium/left atrial appendage and finally to clinical and subclinical strokes. Other potential mechanisms include: brain hypo-perfusion due to beat-to-beat variability in the length of the cardiac cycle and reduced cardiac output, the pro-inflammatory state in AF and peri-ventricular white matter lesions.

Also, the results of the ONTARGET and TRANSCEND studies proved that cognitive and functional decline are important consequences of AF, even in the absence of overt stroke.[2] AF is associated with an increased risk of covert cerebral infarction, which is reported in about one-quarter of patients with AF who undergo magnetic resonance imaging (MRI) of the brain. Epidemiologic studies assessing the relationship of AF and cognitive impairment to brain lesions have been inconsistent. In a study by Farina, et al.,[3] which excluded patients with a history of stroke by detailed imaging, an association between AF and cognitive impairment was shown.

CHAD2DS2-VASc scale [cardiac failure, hypertension, age ≥ 75 (doubled), diabetes, stroke (doubled)-vascular disease, age 65–74 and sex category (female)] is the most-widely used scoring system to assess the thromboembolic risk in patients with AF. For the answer to the questions being asked to our study on the relationship of CHAD2DS2-VASc score and cognitive impairment, we have performed an additional analysis of our study. As shown in Figure 1, with the increasing CHAD2DS2-VASc score, the more advanced forms of cognitive impairment reflected by the decreasing results of Mini Mental State Examination (MMSE) have been observed. Also, CHAD2DS2-VASc score was significantly correlated to MMSE (r = -0.258, Figure 2). In our study, subjects with a history of stroke have been excluded. The question arises to what extent for cognitive impairment occurrence the silent ischaemic brain changes are responsible for? Unfortunately, there is a lack of MRI performance in our study group. Probably, MRI might be of a significant clinical value in patients with AF and cognitive impairment, especially with the higher scores of CHAD2DS2-VASc scale. We propose to consider MRI in subjects with AF, the higher

![Figure 1. Comparison of assessment of the risk of thromboembolic events in patients with atrial fibrillation (scale CHA2DS2-VASc) in groups with different levels of cognitive impairment (MMSE questionnaire) and the result of Kruskal-Wallis. MMSE: Mini Mental State Examination.](http://www.jgc301.com; jgc@mail.sciencep.com | Journal of Geriatric Cardiology)

![Figure 2. Correlation between the risk of thromboembolic events in patients with atrial fibrillation (CHA2DS2-VASc) and the level of cognitive impairment (MMSE questionnaire) and the value of the correlation coefficient Spearman. MMSE: Mini Mental State Examination.](http://www.jgc301.com; jgc@mail.sciencep.com | Journal of Geriatric Cardiology)
age of the subjects. Further studies are required to elucidate the etiology of cognitive impairment in patients with atrial fibrillation.

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