Georgian Scientists
ქართველი მეცნიერები
Vol. 8 Issue 4(1), 2026
https://doi.org/10.52340/gs.2026.08.04(1).08



THE ROLE OF ACQUIRED THROMBOPHILIA IN CARDIO-CEREBRAL COMPLICATIONS AND TREATMENT APPROACHES IN AGING

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Background: Vascular aging serves as a foundational driver for systemic thromboembolic diseases, primarily driven by persistent endothelial dysfunction, vascular stiffness, and altered hemorheological dynamics. In the geriatric cohort, the threat is heavily exacerbated by acquired thrombophilia—a hypercoagulable state triggered by external factors such as chronic low-grade systemic inflammation, malignancies, prolonged immobility, and metabolic disorders. Unlike inherited hypercoagulable conditions, acquired thrombophilias progressively compound with age, transforming subclinical vascular changes into acute, life-threatening clinical episodes. Understanding the unique mechanism by which acquired clotting risks trigger macrovascular and microvascular catastrophes is vital for designing safe prophylactic strategies in aging populations.

Objective: To systematically evaluate the clinical impact of acquired thrombophilia on the development of cardio-cerebral ischemic complications in patients aged 60 and older. Additionally, this study investigates the efficacy and safety profiles of modern personalized antithrombotic treatment algorithms designed to minimize both recurrent thromboembolic events and catastrophic bleeding risks in elderly individuals.

Methods: A comprehensive retrospective clinical evaluation was performed on elderly patients presenting with cardio-cerebral complications at the Arensia Research Center. All subjects underwent an integrated diagnostic workup including biomarkers for hypercoagulability, hyperhomocysteinemia screening, and antiphospholipid antibody profiling. Vascular evaluations were performed via advanced neuroimaging and high-resolution transthoracic echocardiography to identify subclinical thrombi and quantify endothelial strain. Longitudinal data tracked the clinical outcomes of targeted therapies using Direct Oral Anticoagulants (DOACs) versus adjusted low-dose antiplatelet regimens over a 24-month post-discharge follow-up period.

Results: Clinical assessments demonstrated that acquired thrombophilic markers—specifically elevated procoagulant factors and persistently positive antiphospholipid antibodies—significantly correlated with accelerated cardio-cerebral events in aging cohorts. In patients over

60, hypercoagulability directly multiplied the relative risk of acute ischemic strokes, transient ischemic attacks (TIAs), and multi-infarct cognitive decline. On the cardiac spectrum, acquired thrombophilia served as a critical secondary trigger for acute coronary syndrome (ACS) and mural thrombus formation, particularly in hearts already compromised by age-related atrial fibrillation. Regarding therapeutic pathways, standard antithrombotic approaches often induced unacceptably high hemorrhagic transformations due to fragile senescent vessels. Conversely, personalized antithrombotic regimens leveraging low-dose DOACs (e.g., rivaroxaban or apixaban) balanced with strict endothelial protection protocols yielded a 34% reduction in recurrent ischemic events without increasing major gastrointestinal or intracranial bleeding.

Conclusion: Acquired thrombophilia represents a major, yet under-diagnosed, driver of cardio-cerebral morbidity and mortality within the aging population. Combining targeted biomarker profiling with tailored, low-dose anticoagulant strategies offers a superior clinical methodology to mitigate ischemic risk, optimize vascular health, and ensure patient safety in geriatric medicine.

Keywords: Acquired Thrombophilia, Vascular Aging, Ischemic Stroke, Myocardial Infarction, Geriatric Cardiology, Antithrombotic Management.