Georgian Scientists
ქართველი მეცნიერები
Vol. 8 Issue 4(1), 2026
https://doi.org/10.52340/gs.2026.08.04(1).05



# THE USE OF CARDIOVASCULAR COMPLICATION MARKERS IN THE URGENT CARE MANAGEMENT OF LONG COVID

**Nona Kakauridze**

Internal Medicine Department, Tbilisi State Medical University (TSMU), Tbilisi, Georgia

**Introduction:** According to contemporary epidemiological data from the Global Burden of Disease (GBD), the absolute rate of cardiovascular (CV) accidents in the elderly population (60+) is increasing sharply. This dynamic is critically accelerated by post-COVID-19 syndrome (Long COVID). Diagnosing Long COVID in older adults using the American 12-point RECOVER index is frequently challenging due to an atypical clinical presentation and overlapping symptoms.

**Aim of the Study:** The aim of this paper is to conduct an in-depth analysis of current scientific literature to identify specific clinical markers against the background of established pathomechanisms and biomarkers. Identifying these markers will enable early detection of the negative impact of post-COVID-19 syndrome in the elderly, thereby preventing subsequent cardio-cerebral catastrophes and reducing mortality rates in this vulnerable population.

**Pathophysiology:** This paper reviews the link between the molecular cascade of post-COVID-19 systemic thromboinflammation, macro- and microvascular dysfunction (ANOCA/INOCA), neurodegeneration, and accelerated senescence in the elderly. The pathophysiology of COVID-19 is conceptualized as a biphasic—slowly and rapidly progressing—connective tissue, immune, and thromboinflammatory condition driven by the prolonged tissue persistence of viral antigens (spike and nucleocapsid proteins). This persistence induces premature lymphocyte aging (immunosenescence) and telomere decondensation, undermining cellular regeneration. Because the endothelial response is systemic, CV dysfunction occurs synchronously with cerebral small vessel disease (CSVD), programming premature biological aging.

**Key Molecular and Clinical Markers:** Since identifying every single marker is impractical in routine clinical practice, this study focuses on core biomarkers that simultaneously reflect myocardial and cerebral injury at the micro-level, serving as essential tools for clinical monitoring:

- Apolipoprotein A1 (ApoA-1) Decline and Elevated Homocysteine: A decrease in ApoA-1 dismantles the natural anti-inflammatory shield, triggering lipid peroxidation. Concurrently, hyperhomocysteinemia directly damages the endothelium and exacerbates atherothrombotic processes.
- IL-18: Functions as a master mediator of the pro-inflammatory cascade, activating inflammasome, amplifying vascular inflammation, and contributing to atherosclerotic plaque instability.
- Elevated P-Selectin: Drives endothelial adhesion, leading to microvascular ischemia in the heart and blood-brain barrier disruption.
- Chronic D-Dimer Elevation: Confirms ongoing, accelerated microthrombosis, significantly increasing ischemic stroke risks in elderly individuals.
- Nuclear Factor kappa B (NF-κB): Acts as a molecular switch triggering neuroinflammation and activating tau protein hyperphosphorylation, leading to the pathological assembly of neurofibrillary tangles.

**Conclusion:** This study confirms that Long COVID acts as a biological catalyst for accelerated senescence, manifested through telomere decondensation and immune cell exhaustion. Coronary microvascular pathology and neurodegenerative brain injury represent two distinct manifestations of the same systemic endotheliitis. Early monitoring of these dual-action clinical markers (including Homocysteine and IL-18) is vital for preventing premature dementia, fatal strokes, and early mortality in the elderly population.

**Keywords:** Long COVID, Gerontology, Thromboinflammation, CVD