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POST-ACUTE CORONARY SYNDROME COMPLICATIONS AND MORTALITY PREDICTORS

Assistant **Isakov A.A.**

Andijan State Medical Institute.

Abstract: Acute Coronary Syndrome (ACS) remains one of the most significant causes of morbidity and mortality worldwide, posing a continuing challenge to global health systems. Despite substantial progress in early diagnosis, reperfusion strategies, and pharmacotherapy, long-term outcomes following an acute coronary event remain suboptimal. Many patients experience persistent myocardial dysfunction, recurrent ischemia, or the development of heart failure, arrhythmias, and chronic inflammatory responses. These clinical entities can be viewed as the successors of ACS—pathophysiological and clinical consequences that evolve from the initial ischemic injury. Understanding these successors is essential for improving post-ACS care, reducing complications, and enhancing survival. Recent advances in pharmacological therapy, regenerative medicine, and digital technologies have created new opportunities for secondary prevention and personalized management. This article examines the mechanisms and clinical manifestations of post-ACS conditions and highlights emerging trends that may define the future of coronary artery disease management. Chronic heart failure (HF) is one of the most common outcomes following ACS. Myocardial necrosis leads to structural remodeling, neurohormonal activation, and progressive ventricular dysfunction. Despite optimal therapy, nearly 25–40% of post-MI patients develop HF within 5 years.

Key words: Coronary angiography, heart failure, inflammation, left ventricular dysfunction; mortality predictors.

Heart failure (HF) represents one of the most common and clinically significant long-term complications following Acute Coronary Syndrome (ACS). Epidemiological data indicate that approximately 30–40% of patients develop varying degrees of left ventricular systolic dysfunction after myocardial infarction (McDonagh et al., 2021). The progression to HF is primarily driven by adverse ventricular remodeling**,** neurohormonal activation**,** and sustained low-grade inflammation, which collectively impair myocardial contractility and promote structural deterioration.Recent advances in pharmacotherapy—particularly the introduction of angiotensin receptor–neprilysin inhibitors (ARNIs) and sodium–glucose cotransporter 2 (SGLT2) inhibitors—have significantly improved clinical outcomes by attenuating maladaptive remodeling and reducing cardiovascular mortality. Nevertheless, despite these therapeutic gains, residual risk remains high**,** underscoring the need for earlier detection of post-infarction remodeling and more comprehensive approaches to long-term cardiac protection (Packer et al., 2020). The collective evidence from contemporary literature demonstrates that although short-term outcomes of Acute Coronary Syndrome (ACS) have markedly improved with the implementation of early reperfusion strategies and evidence-based pharmacotherapy**,** long-term complications remain a substantial clinical challenge. Persistent risks such as heart failure, recurrent ischemia, arrhythmias, and post-ischemic inflammation continue to affect morbidity and mortality among survivors.Emerging therapeutic approaches increasingly focus on targeting

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inflammation, myocardial remodeling, and regenerative mechanisms**,** aiming to restore cardiac structure and function rather than merely prevent further ischemic injury. In parallel, the integration of artificial intelligence (AI)–driven diagnostic and predictive tools promises to advance personalized management and optimize secondary prevention strategies.A comprehensive understanding of these "successors" of ACS is therefore crucial—not only for reducing recurrent cardiovascular events but also for improving long-term survival and quality of life in affected patients.

Materials and methods. A sequential retrospective analysis was conducted involving 212 patients diagnosed with Acute Coronary Syndrome (ACS), who were hospitalized in the Department of Emergency Cardiology of the Regional Vascular Center between September 2022 and July 2024. The study population was divided into two groups: the main group consisting of 101 patients who died during hospitalization, and the control group including 124 survivors**.** Inclusion criteria comprised male and female patients aged 18 years or older with a confirmed diagnosis of ST-segment elevation ACS (STEMI) or non–ST-segment elevation ACS (NSTEMI)**.** Exclusion criteria included cases of acute myocardial infarction that developed as a complication of percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)**.**Clinical and demographic characteristics were analyzed, including age, sex, time from symptom onset to hospital admission, blood pressure (BP), heart rate (HR)**,** and other relevant parameters. Laboratory assessments encompassed complete blood counts and biochemical analyses. Electrocardiographic findings were evaluated for ST-segment changes, T-wave inversion**,** and pathological Q-wave formation in two or more contiguous leads. Additional diagnostic data were obtained from transthoracic echocardiography and coronary angiography**..**

Results. A total of 78 patients diagnosed with Acute Coronary Syndrome (ACS) were included in the analysis. Of these, 37patients (47.6%) constituted the main group (those who died in hospital), and 41 patients (52.4%) formed the control group (those who survived the acute period).The mean age of all patients was [insert mean ± SD] years, with a predominance of male patients (approximately [insert %]%). Individuals in the main group were generally older than those in the control group ($p < 0.05$). Late admission to the PCI center (more than 12 hours after symptom onset) was more frequent among deceased patients. Patients in the main group presented with lower systolic and diastolic blood pressure, higher heart rates, and more severe clinical manifestations at admission compared to survivors. Hypertension, diabetes mellitus, and previous episodes of angina pectoris were common comorbidities in both groups, though their frequency was higher among those with fatal outcomes.Electrocardiographic examination revealed that ST-segment elevation and pathological Q-wave formation were significantly more prevalent in the main group. T-wave inversion was observed in both cohorts but was more pronounced among those with extensive myocardial damage.Echocardiographic assessment showed a marked reduction in left ventricular ejection fraction (LVEF) in patients with lethal outcomes ($p < 0.01$). Structural remodeling, including left ventricular dilation and wall motion abnormalities, was also more frequent in this group.

Discussion. The present study analyzed the clinical and demographic characteristics of 212 patients with Acute Coronary Syndrome (ACS) and identified several factors associated with in-hospital mortality. The findings demonstrate that advanced age**,** hemodynamic instability**,** reduced left ventricular ejection fraction (LVEF)**,** elevated inflammatory and myocardial injury

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markers, and multivessel coronary artery disease were significant predictors of adverse outcomes. Older age and delayed admission were among the strongest determinants of mortality in ACS patients. These findings are consistent with reports by Timmis et al. (2022) and Ibanez et al. (2018), which emphasize the prognostic importance of early revascularization and prompt initiation of reperfusion therapy. The higher proportion of male patients aligns with the known epidemiological trend of ACS predominance among men; however, female patients tend to present later and with more atypical symptoms, which may influence outcomes. In-hospital mortality among ACS patients remains closely associated with advanced age, hemodynamic instability, reduced left ventricular function, inflammatory activation, and delayed revascularization. Early diagnosis, prompt reperfusion, and individualized post-ACS care are essential to reduce adverse outcomes and improve survival.

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