

**PROGNOSTIC SIGNIFICANCE OF THE CLINICAL FEATURES OF
MYOCARDIAL INFARCTION FOR SIX-MONTH CARDIOVASCULAR
OUTCOMES DURING SGLT2 INHIBITOR THERAPY**

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Despite advances in reperfusion and secondary prevention, patients who survive an acute myocardial infarction (MI) remain at substantial residual risk of recurrent cardiovascular events and heart failure. Sodium–glucose cotransporter-2 inhibitors (SGLT2i) have shown cardioprotective and heart-failure benefits in landmark trials (EMPA-REG OUTCOME, EMMY, DAPA-MI, EMPACT-MI), and their early initiation in the acute phase of MI is an area of active investigation. At the same time, the electrocardiographic type of infarction — with or without pathological Q-wave formation — reflects the extent of myocardial injury and has traditionally carried prognostic weight. Whether MI type retains its prognostic value in patients treated with early SGLT2i therapy remains insufficiently studied.

To assess the prognostic significance of the type of myocardial infarction (MI) — Q-wave versus non-Q-wave — for major adverse cardiovascular events (MACE) over 6 months in patients receiving early initiation of the sodium–glucose cotransporter-2 inhibitor (SGLT2i) empagliflozin.

A total of 251 patients with acute MI were included and divided by infarction type into two groups: Q-wave MI (n=131; 52.2%) and non-Q-wave MI (n=120; 47.8%). All patients (100%) received empagliflozin during the acute phase (median dose 10 mg), against a background of high adherence to guideline-recommended therapy: statins — 96.0%, acetylsalicylic acid — 90.8%, P2Y₁₂ inhibitors — 94.0%, beta-blockers — 90.8%, and mineralocorticoid receptor antagonists (MRAs) — 77.7%. The composite endpoint was MACE (death, non-fatal recurrent MI, hospitalization, and emergency PCI). Between-group differences were analyzed using Fisher's exact test, with calculation of the odds ratio (OR) and 95% confidence interval (CI).

Given identical empagliflozin therapy, the groups were comparable with respect to antiplatelet prescribing; however, patients with Q-wave MI significantly more often received beta-blockers (96.2% vs. 85.0%; $p=0.004$), MRAs (85.5% vs. 69.2%; $p=0.002$), and loop diuretics (30.5% vs. 18.3%; $p=0.028$), reflecting a greater extent of myocardial injury. Over the 6-month follow-up, MACE were recorded in 41 (31.3%) patients in the Q-wave group versus 21 (17.5%) in the non-Q-wave group (OR 2.15; 95% CI 1.17–3.84; $p=0.013$). The increased risk was driven predominantly by repeat hospitalizations — 20.6% vs. 10.0% (OR 2.34; 95% CI 1.11–4.70; $p=0.023$), the only component to reach significance on individual analysis. Mortality (6.1% vs. 3.3%; $p=0.382$) and the rate of emergency PCI (4.6% vs. 0.8%; $p=0.122$) were numerically higher in Q-wave MI but did not reach statistical significance.

Against a background of uniform empagliflozin therapy and comparable guideline-recommended treatment, the type of myocardial infarction retains independent prognostic significance: Q-wave MI is associated with a more than twofold increase in the risk of MACE over 6 months, driven predominantly by repeat hospitalizations.