Commentary

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Cardiometabolic Therapies at the Crossroads of Coronary Artery Disease

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About the Study

Coronary Artery Disease (CAD) remains the leading cause of morbidity and mortality worldwide. For decades, cardiology has relied on lipid lowering, antiplatelet therapy, traditional anti-hyperglycemic drugs and blood pressure control to modify cardiovascular risk and improve survival. These measures, while transformative, largely address the risk factors or the downstream of atherosclerosis. The emergence of Sodium-glucose cotransporter-2 inhibitors (SGLT2i) and Glucagon-like Peptide-1 Receptor Agonists (GLP-1RAs) represents a shift toward therapies that act upstream, modifying the metabolic and inflammatory milieu in which CAD develops and progresses [1].

Landmark trials and large robust registries highlight how these agents, once considered only glucose-lowering drugs, are reshaping cardiovascular practice [2]. Their analysis underscores a growing consensus: SGLT2i and GLP-1RAs are cardiovascular drugs with metabolic advantages. The speed and magnitude of benefit observed in outcome trials cannot be explained solely by their effects on glycated haemoglobin and/or weight [3]. With SGLT2i, reductions in heart failure hospitalization emerge within weeks, driven by osmotic diuresis, improved myocardial energetics, and renal unloading. GLP-1RAs act by stabilizing atherosclerotic plaques, improving endothelial function, and reducing inflammation. Together, they offer complementary protection: SGLT2i addressing hemodynamic stress and heart failure, GLP-1RAs modifying vascular biology and long-term ischemic risk [3]. Recent data also indicate that GLP-1RAs may confer additional benefits in patients with heart failure [4]. This uncoupling from glycaemic control reframes how physicians should think about metabolic drugs. While glucose management remains a key objective, the primary target now expands to the entire cardiometabolic ecosystem.

Viewing CAD through inflammatory-metabolic lens provides a more integrated understanding of risk. Diabetes, obesity and chronic kidney disease often cluster with atherosclerosis, amplifying the likelihood of recurrent events. SGLT2i and GLP-1RAs directly address this intersection [1,4]. By modulating renal hemodynamics, vascular inflammation, and adipose-cardiac signaling, they offer a unified therapeutic approach that bridges cardiology, nephrology and endocrinology [4]. This convergence challenges cardiological specialty to expand its scope. The coronary atherosclerosis cannot be treated in isolation; it must be managed within its metabolic environment Likewise, endocrinologists must move beyond a glucocentric approach, to a comprehensive cardiovascular risk-based approach, ensuring patients who stand to benefit most receive these cardioprotective therapies, independent of their glycemic control. Most landmark trials evaluated stable patients with type 2 diabetes at high risk of events [1,2]. Whether similar benefits apply to the high-inflammatory, high-risk setting of Acute Myocardial Infarction (AMI) remains a crucial question. Recent studies offer important clues [5]. EMPACT-MI demonstrated fewer heart failure events after AMI with empagliflozin, though not a reduction in mortality. DAPA-MI extended potential benefit to patients without diabetes, improving cardiometabolic health after infarction. Smaller studies of GLP-1RAs suggest reduced infarct size and improved remodeling, while registry data indicate lower mortality when used before AMI [6]. These findings point toward a future in which cardiometabolic therapies may become part of the standard post-AMI regimen. Confirmation will require large, dedicated outcome trials.

How should cardiologists and endocrinologists incorporate these insights today? First, by broadening perspective: these agents are not niche diabetes drugs but core cardiovascular therapies. Second, by identifying high-risk groups, especially patients with CAD and diabetes, obesity, or chronic kidney disease, who stand to benefit the most, also in primary prevention. Third, by preparing for integration into acute coronary syndrome care as evidence continues to evolve.

The trajectory of cardiovascular prevention is clear. We are moving from event management to disease modification. Lipid lowering and antiplatelet therapy remain cornerstones, but no

longer define the full therapeutic landscape. Inflammation, renal function, and systemic metabolism have emerged as equally legitimate targets. SGLT2i and GLP-1RAs are at the forefront of this evolution. They remind us that cardiovascular disease is inseparable from its metabolic context and that optimal care must address both simultaneously. The challenge now is to refine their role: to determine optimal timing, combination strategies, and cost-effective implementation while ensuring equitable access across diverse health systems.

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