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# SGLT2 Inhibitors in Heart Failure: Beyond Glycemic Control

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**Dear Editor,**

Sodium–glucose cotransporter 2 (SGLT2) inhibitors have rapidly moved from being viewed solely as glucose-lowering agents to becoming central in the management of heart failure (HF). Evidence now consistently shows that their benefits extend well beyond glycemic control, reshaping clinical practice.

Several studies have demonstrated improvements in cardiac biomarkers, including natriuretic peptides and markers of inflammation, among patients on SGLT2 inhibitors (1). More importantly, randomized controlled trials confirm significant reductions in cardiovascular deaths and HF hospitalizations, with benefits observed even in patients without diabetes (2). This broader applicability has expanded their clinical value beyond endocrinology into mainstream cardiology.

A growing body of evidence also suggests that SGLT2 inhibitors may reduce myocardial fibrosis, particularly in heart failure with preserved ejection fraction (HFpEF), a subgroup with limited effective therapeutic options (3). Early findings from prospective studies even suggest potential cognitive benefits in older patients with HF, possibly through improved cerebral perfusion or reduced systemic inflammation (4). Furthermore, systematic reviews now increasingly support routine use of SGLT2 inhibitors in HFpEF, strengthening their role in contemporary management strategies (5).

Taken together, these findings highlight how SGLT2 inhibitors influence not only glucose metabolism but also cardiac remodelling, inflammation, and potentially neurocognitive outcomes. For clinicians, this represents a paradigm shift in heart failure management, emphasizing the importance of incorporating these agents into treatment guidelines. As research continues to expand, their role will likely extend even further, positioning them as a cornerstone therapy in both reduced and preserved ejection fraction HF.

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