The impact of sodium-glucose cotransporter-2 inhibitors on ventricular arrhythmia burden in patients with implantable cardioverter defibrillators

Nikša Bušić*,
Ana Jordan,
Ante Lisičić,
Sanda Sokol Tomić,
Ivan Zeljković,
Šime Manola,
Nikola Pavlović

Dubrava University Hospital, Zagreb, Croatia

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***ADDRESS FOR CORRESPONDENCE**: Nikša Bušić, Klinička bolnica Dubrava, Avenija Gojka Šuška 6, HR-10000 Zagreb, Croatia. / Phone: +385-99-3532-343 / E-mail: niksabusic@gmail.com

ORCID: Nikša Bušić, https://orcid.org/0000-0002-7082-4932 • Ana Jordan, https://orcid.org/0000-0003-4996-2203
Ante Lisičić, https://orcid.org/0000-0002-4365-9652 • Sanda Sokol Tomić, https://orcid.org/0000-0002-4551-9231
Ivan Zeljković, https://orcid.org/0000-0002-4550-4056 • Šime Manola, https://orcid.org/0000-0001-6444-2674
Nikola Pavlović, https://orcid.org/0000-0001-9187-7681

Introduction: Sodium-glucose cotransporter-2 inhibitors (SGLT2i), initially developed to treat type 2 diabetes mellitus (T2DM), have shown cardiovascular benefits beyond glucose control, particularly in heart failure patients.[1] However, limited data exist on the effects of SGLT2i on the ventricular arrhythmia (VA) burden in patients with implantable cardioverter defibrillators (ICDs). This study aims to investigate the impact of SGLT2i on VA burden in ICD patients.

Patients and Methods: This was a prospective, single-center, observational study conducted on patients with ICDs. All patients are enrolled in an institutional registry (CaRD registry-HF). The patients were divided into three groups based on the use of SGLT2 inhibitors: no use (SGLT2 = 0, N=27), and consistent use (SGLT2 = 1, N=50). Variables such as age, sex, body mass index, and primary versus secondary ICD indication were considered. The primary outcome was the recurrence of ventricular tachycardia (VT) events, recorded as the number of VA episodes detected by ICDs.

Results: The mean age of patients was similar across groups, with a slight variation: 64.5 years (SGLT2 = 0), 61.7 years (SGLT2 = 1). The majority of patients in the SGLT2 = 1 group had primary ICD indications (87.9%), compared to 54.8% in the no-SGLT2 group. Ischemic heart disease was the leading cause in the SGLT2 = 0 group (77.4%) compared to 65.7% in the SGLT2 = 1 group. In patients with no SGLT2i use, 22, 2% patients experienced the recurrence of VT. Patients with SGLT2i use had a lower recurrence rate of VT, with 13,4% of patients experiencing VT (n=21).

Conclusion: The results of this study indicate that the use of SGLT2 inhibitors is associated with a lower VA burden in patients with ICDs. These findings suggest that SGLT2i may have antiarrhythmic effects, possibly due to their ability to reduce cardiac fibrosis, inflammation, and improve myocardial energy efficiency. Additionally, the reduction in VA could be explained by the favorable effects of SGLT2i on heart failure outcomes, such as improved diastolic function, reduced left ventricular wall stress, and a decrease in overall cardiac workload. Further research is warranted to confirm these findings and establish the role of SGLT2 inhibitors in arrhythmia management.

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LITERATURE

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