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Acute Effects of Dapagliflozin on Myocardial Work in Type 2 Diabetics With Heart Failure With Reduced Ejection Fraction: A Crossover Trial

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Recent studies have demonstrated benefits of sodium-glucose co-transporter 2 inhibitors (SGLT2i) in the management of heart failure with reduced ejection fraction (HFrEF). Although current literature postulates several salient effects, the primary mechanism is unclear. Intriguingly, benefit is seen rapidly. Improved myocardial energetics and substrate efficiency through Na⁺/H⁺ exchanger inhibition and increased β-hydroxybutyrate metabolism may rapidly reduce myocardial work.

Our study aimed to evaluate the early effects of dapagliflozin on myocardial work. In a post hoc analysis of the SGLT2i Acute Effects Crossover Trial, we studied the effects of 2-week treatment with dapagliflozin on echocardiographic parameters of myocardial work in type 2 diabetic (T2DM) HFrEF subjects in a double-blinded, placebo-controlled, randomised crossover trial.

Nineteen patients completed the study with 3 excluded due to missing data. Dapagliflozin reduced systolic [114 (105, 131) vs 106 (98, 113) mmHg, p<0.01] and diastolic BP [71 (61, 78) vs 62 (55, 70) mmHg, p<0.01] and ventricular ectopy [1.4 (0.1, 2.9) vs 0.2 (0.1, 1.4) %, p<0.05]. Left ventricular ejection fraction was unchanged [42 (35, 52) vs 44(36, 51) %]. There was no change in parameters of myocardial work (global longitudinal strain, peak strain dispersion, global work index [865 (724, 1,143) vs 898 (714, 1,216) mmHg%], global constructive work, global wasted work and global work efficiency [85 (79, 89) vs 83 (79, 90) %] after dapagliflozin treatment. β-hydroxybutyrate was also unchanged.

While we were able to demonstrate salient haemodynamic and antiarrhythmic effects with dapagliflozin, improvement in myocardial work was not shown at 2 weeks in this T2DM HFrEF cohort.

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Acute Reversible Cardiomyopathy With Multi-System Inflammatory Syndrome in Adults (MIS-A) Secondary to SARS-CoV-2 Infection: A Case Report

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Background: Multi-system inflammatory syndrome is a rare yet serious complication of COVID-19 infection and can affect multiple organ systems including cardiovascular manifestations.

Case Presentation: A previously healthy 49-year-old gentleman presented with a 1-week history of high-grade fever, conjunctivitis, diarrhoea and a generalised blanching rash following recent SARS-CoV-2 infection. He demonstrated signs of cardiogenic shock requiring inotropic support. Investigations revealed elevated inflammatory markers, liver derangement and an acute kidney injury. There was radiological evidence of mediastinal and axillary lymphadenopathy along with pulmonary venous congestion. CT abdomen revealed acute proctocolitis.

Troponin level was elevated at 189 ng/L, as was NT-proBNP levels at >35,000 ng/L. Echocardiogram showed severe biventricular systolic dysfunction. Percutaneous coronary angiogram confirmed normal coronary arteries. He was treated with 1 g/kg intravenous immunoglobulin (IVIG), and 1 g IV methylprednisolone daily for 72 hours, followed by high-dose oral prednisone. His inotropic requirements diminished and repeat echocardiogram 10 days later demonstrated normalisation of biventricular function. Troponin levels also normalised. He was discharged home on a steroid tapering regimen. A cardiac MRI was performed 17 days following initial presentation which showed normal ventricular volumes, systolic function, and no evidence of myocardial inflammation.

Discussion: MIS-A post-acute COVID-19 infection is a rare yet life-threatening syndrome with an increasing association with delayed-onset myocarditis. Here we present a case of severe acute cardiac dysfunction related to MIS-A myocarditis with normalisation of cardiac function and biomarkers after prompt initiation of heavy immunosuppression. Early evaluation with echocardiogram and initiation of IVIG and corticosteroid immunosuppression may prevent long-term morbidity and/or death.

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