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SGLT2 Inhibitor Alters Circulating Mitokine Levels in Patients with Chronic Kidney Disease

Seunghwan Jeong, Jin Seok Jeon, Hyunjin Noh, Soon Hyo Kwon, Haekyung Lee
Department of Internal Medicine-Nephrology, Soonchunhyang University Seoul Hospital, Korea, Republic of

Objectives : Mitokines are released in response to mitochondrial stress and the mitochondrial unfolded protein response. They play a crucial role in inter-organ communication. Sodium–glucose co-transporter 2 (SGLT2) inhibitors mitigate mitochondrial dysfunction. In this study, we aimed to investigate changes in mitokine levels in patients with chronic kidney disease (CKD) and the effects of SGLT2 inhibitor treatment.
Methods : We prospectively recruited SGLT2 inhibitor-naïve patients with CKD (n = 68; 22.1 % with diabetes) and healthy controls (n= 35). Circulating mitokine levels—fibroblast growth factor 21 (FGF21), growth differentiation factor 15 (GDF15), mitochondrial open reading frame of the 12S rRNA-c (MOTS-c), and humanin—were measured using ELISA. In patients with CKD, mitokine levels were reassessed at 3 and 6 months following SGLT2 inhibitor treatment.
Results : Compared to healthy controls, MOTS-c, FGF21, and humanin levels were increased, while GDF15 levels were decreased in CKD patients (Figure 1). Mitokine levels did not differ based on diabetes status in CKD patients. When patients with CKD were stratified into two groups based on their respective baseline mitokine levels, MOTS-c decreased 3 months after SGLT2 inhibitor treatment in the high baseline group but increased at 6 months in both the low and high baseline groups. In patients with low baseline mitokine levels, GDF15, FGF21, and humanin increased 3 months after SGLT2 inhibitor treatment but decreased at 6 months. Additionally, FGF21 decreased 6 months after SGLT2 inhibitor treatment in the high baseline group.
Conclusions : CKD is associated with dysregulated circulating mitokine levels. SGLT2 inhibitor treatment resulted in dynamic changes in mitokine levels, suggesting that SGLT2 inhibitors could attenuate mitochondrial damage in patients with CKD.

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