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Dynamic Changes in Circulating Mitokines Following Bariatric Surgery in Patients with Obesity

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Objectives : Obesity is linked to mitochondrial dysfunction. Mitokines are released in response to mitochondrial stress and the mitochondrial unfolded protein response. They play a crucial role in inter-organ communication. Bariatric surgery has been shown to improve mitochondrial dysfunction. In this study, we aimed to investigate the effects of obesity and bariatric surgery on mitokine levels.
Methods : We prospectively recruited patients with morbid obesity (n = 45) 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 obesity, mitokine levels were reassessed at 3 and 6 months following bariatric surgery.
Results : In obese patients, FGF21 and humanin levels were increased, while GDF15 levels were decreased compared to healthy controls (Figure 1). MOTS-c levels did not differ significantly between the two groups. When patients with obesity were stratified into two groups based on their respective baseline mitokine levels, GDF15 and humanin decreased at 3 months after bariatric surgery and remained unchanged at 6 months in the high baseline group. In the low baseline group, GDF15 decreased at 3 months after bariatric surgery but increased at 6 months.
Conclusions : Obesity is associated with dysregulated circulating mitokine levels. Mitokine levels decrease early after bariatric surgery. This suggests that bariatric surgery could mitigate mitochondrial damage in patients with obesity.

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