

EFFECT OF THE TGFB1 Arg25Pro POLYMORPHISM ON TREATMENT EFFICACY AND CLINICAL-FUNCTIONAL PARAMETERS IN CHRONIC HEART FAILURE

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Relevance of the Study

Chronic heart failure (CHF) is one of the common and severe complications of ischemic heart disease (IHD), in the development of which myocardial remodeling and fibrotic processes play an important role [1–2]. The TGFβ1 gene is involved in the molecular regulation of these processes. Therefore, investigation of the TGFβ1 Arg25Pro (rs1800471) polymorphism is relevant to the genetic assessment and prediction of the risk of CHF in patients with IHD [5].

Identifying the association between this polymorphism and the development and clinical course of CHF may help stratify patients into risk groups and improve early preventive measures and individualized therapeutic approaches [3–4].

Aim of the Study

To determine the role of the TGFB1 gene polymorphism in the development of chronic heart failure and to assess its prognostic significance.

Materials and Methods

The analysis included 42 patients receiving empagliflozin and 61 patients who did not receive the drug. The study was conducted in accordance with standard diagnostic criteria for CHF, using instrumental, biochemical, molecular genetic, and statistical research methods.

Results

Among patients not treated with empagliflozin, the Arg/Arg genotype of the TGFβ1 gene was the most common, identified in 46 individuals. In this subgroup, LVEF increased from $48.67 \pm 2.00\%$ to $49.80 \pm 2.00\%$, an absolute increase of 1.13 percentage points, which was statistically significant ($p=0.008$). By comparison, patients receiving empagliflozin showed a more pronounced improvement in LVEF, with an increase of 3.22% versus 1.13% in those who did not receive the drug. Therefore, among Arg/Arg genotype carriers, the increase in LVEF observed during empagliflozin treatment was approximately 2.9-fold greater than that recorded in

patients who did not receive empagliflozin. A difference was also observed between the empagliflozin-treated and untreated groups among Arg/Pro genotype carriers. In 13 patients with the Arg/Pro genotype who received empagliflozin, LVEF increased from $39.14 \pm 2.90\%$ to $40.92 \pm 3.05\%$. Although the increase was 1.78 percentage points, it did not reach statistical significance ($p=0.140$) (Table 4.7). In 15 patients with the Arg/Pro genotype who did not receive empagliflozin, the mean LVEF was $45.13 \pm 4.47\%$ before treatment and $45.13 \pm 4.18\%$ at the end of follow-up, indicating virtually no change ($p=1.0$).

The Pro/Pro genotype was detected in 2 patients in the empagliflozin-treated group, in whom LVEF increased from $28.5 \pm 4.5\%$ to $30.0 \pm 6.0\%$. The magnitude of the change was 1.5 percentage points; however, the change was not statistically significant ($p=0.8$). The Pro/Pro genotype was not detected in the group of patients who did not receive empagliflozin ($n=0$).

Conclusion

The results for the TGF β 1 Arg25Pro polymorphism showed differences in LVEF dynamics by genotype. In patients with the Arg/Arg genotype who did not receive empagliflozin, LVEF increased by 1.13 percentage points, whereas no change in LVEF was observed in patients with the Arg/Pro genotype. Notably, the greater improvement in LVEF among empagliflozin-treated patients with the Arg/Arg genotype may indicate a potential role of this

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