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Letter To Editor Open Access

Role of TGF-β1 variation in type 1 diabetes and cardiovascular complications

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Dear Editor

I read the article by Tjahjono et al., which examined the levels of TGF-β1 in patients with type 1 diabetes (T1D) and healthy controls (HC)[1]. They found a significant decrease in plasma TGF-*β*1 levels in T1D patients relative to controls. These results are consistent with the findings of another study, which indicates decreased serum levels of TGF-*β* in T1D patients ($P < 0.001$)[2]. Their results also indicated a strong correlation between two atherosclerosis markers common carotid artery intima-media thickness (IMT) and flow-mediated dilatation and TGF-*β*1 concentrations in T1D patients.

These findings are important because multiple observations suggest that T1D is a risk factor for atherosclerosis, although the exact mechanism remains unknown. Studies show that a variety of factors can increase the risk of cardiovascular disease in patients with T1D. Among them, dysfunctional immune and inflammatory responses hold a distinctive position. Inflammation is a natural defense mechanism against harmful stimuli and is highly regulated by a multitude of cytokines. These mediators are crucial components of inflammation, and their altered profiles are believed to play a role in the pathogenesis of various diseases, including T1D and atherosclerosis.

TGF-*β*1 is a promising candidate for inflammation research because it plays a crucial role in the immune system and human diseases[3]. Nonetheless, determining its precise characteristics is challenging, as it can exert dual and opposing effects on inflammatory responses. TGF-*β*1 is a multifunctional molecule with pleiotropic activity, involved in various cellular processes, including tissue and immune homeostasis, cell proliferation, and differentiation. It is conventionally regarded as an anti-inflammatory agent, attenuating the expression of cytokines,

chemokines, adhesion molecules, and reactive oxidative species, while suppressing cells of both the innate and adaptive immune systems. However, not all effects of TGF-β1 are suppressive. In conjunction with IL-6 or IL-21, it can induce the differentiation of Th17 cells, which are considered a subset of pro-inflammatory T helper cells[4]. It also possesses potent chemoattractive properties, even at very low concentrations, and can induce the rapid accumulation of white blood cells at the site of inflammation.

These complex properties of TGF-β1 contribute to ongoing controversies regarding its role in the development of atherosclerosis in T1D patients. Despite its enigmatic nature in T1D, multiple studies suggest that abnormal TGF-β levels may increase the risk of atherosclerosis[5]. It has been revealed that aberrant TGF-β1 expression is involved not only in changes in vascular architecture but also plays a role in shifting vasculature toward a pro-atherogenic state.

Moreover, the deletion of a single allele of the TGF-β1 gene leads to an approximately fifty percent reduction in the amount of TGF-β1 protein in the vessel media. This decrease is accompanied by increased susceptibility to endothelial cell activation and the formation of vascular lipid lesions in response to pro-atherogenic stimuli, including a lipid-rich diet. Additionally, the inhibition of TGF-β signaling can increase vascular inflammation, accelerate lipid lesion formation, and alter plaque morphology toward an unstable lesion[6].

Based on the above findings, TGF-β1 can exhibit either pro- or anti-atherogenic properties, depending on its concentration and context. Therefore, further research is needed to elucidate the intricacies of the relationship between TGF-β1, T1D, and atherosclerosis.

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