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The Potential Role of MicroRNA in Diabetic Cardiomyopathy

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Diabetic cardiomyopathy (DCM) is a chronic and irreversible diabetic complication, which is manifested as abnormal cardiac structure and function, that is independent of coronary artery disease, valvular disease, and cardiovascular risk factors such as hypertension and dyslipidemia [1,2]. It is characterized by ventricular dilation and hypertrophy, diastolic dysfunction, later by systolic dysfunction, and consequently lead to heart failure [3]. Minimal criteria for diagnosis of DCM include left ventricular diastolic dysfunction and/or reduced left ventricular ejection fraction, left ventricular hypertrophy and interstitial fibrosis [4]. Increased incidence of diabetes can result in an increased prevalence of DCM, which is a major cause of morbidity and mortality in people with diabetes [5,6].

Understanding and identifying the pathogenesis and the underlying mechanism may allow for earlier precision interventions that would lead to improved survival rates and prevention of DCM. Furthermore, identifying the underlying molecular mechanisms of DCM would be valuable for targeted therapy and provide an opportunity to reduce DCM and improve overall public health. Previous studies suggested that cardiac metabolic disturbances such as insulin resistance and glucotoxicity, mitochondrial dysfunction, subcellular signaling disturbance, impaired autophagy, autonomic dysfunction, activation of renin-angiotensin-aldosterone system, inflammation, dysregulation of exosomes, oxidative stress, and immune maladaptation are potential pathophysiological factors [1,4,6,7]. However, the pathogenesis of DCM is complex, which may involve various factors such as cardiac hypertrophy, myocardial fibrosis, cardiomyocyte apoptosis, and myocardial metabolic disorders, and the precise molecular and metabolic pathways underlying cardiac dysfunction remains to be understood.

This article entitled "Role of microRNA-34a in anti-apoptotic effects of granulocyte-colony stimulating factor in diabetic cardiomyopathy," along with that published by Park et al. [8], evaluated the mechanism underlying the anti-apoptotic effects of granulocyte-colony stimulating factor (G-CSF) via regulation of microRNA (miRNA)-34a in a DCM rat model. In this study, G-CSF reduced apoptosis of myocardium and induced down-regulation of miR-34a expression. Moreover, transfection with miRNA-34a mimic significantly induced apoptosis in H9c2 cells. The authors suggested that the anti-apoptotic effects of G-CSF in a rat model of DCM are mediated by reduced expression of miRNA-34a. This approach is quite valuable in establishing a potential therapeutic biomarker for cardiac remodeling in DCM. The authors have clearly shown their results in this manuscript.

Recently, miRNA, which are a class of endogenous, noncoding RNAs with regulatory functions, have been identified as a key element involved in cardiac gene remodeling through post-transcriptional regulation of target gene proteins and suggested a potential diagnostic, prognostic and therapeutic role in diabetes [9,10]. A previous study showed that, in cardiac remodeling and the development of heart failure, the synthesis of miRNA and levels of specific miRNA were altered [11]. miRNAs (miR-34b, miR-34c, miR-199b, miR-210, miR-223, and miR-650) might be involved in the pathogenesis of failing

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myocardium in patients with diabetes [12]. miRNAs may be involved in cardiac hypertrophy, remodeling and heart failure progression through various process such as mitochondrial function, apoptosis, fibrosis, pyroptosis, and neurohormone secretion [11].

The current study provides a potential new therapeutic approach based on miRNAs and closer precision medicine in diabetic cardiac pathology. Further investigation to determine the potential contribution of various miRNAs to cardiomyopathy in diabetes are needed.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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