Adiponectin and Diabetic Cardiomyopathy

Yajing Wang, PhD

Abstract

Diabetic cardiomyopathy, characterized by structural and functional alterations in the myocardium independent of traditional cardiovascular risk factors, poses a significant clinical challenge. Adiponectin, an adipose-derived hormone with known insulin-sensitizing and anti-inflammatory properties. Clinical observations consistently reveal reduced adiponectin levels in individuals with cardiomyopathy, suggesting a potential link between lower adiponectin concentrations and the development or progression of this cardiac complication. Furthermore, evidence from our lab suggests that diminished levels of adiponectin may be a critical contributor to the initiation and acceleration of diabetic cardiomyopathy through intricate signaling pathways. We explore the intricate relationship between the pathogenesis of diabetic cardiomyopathy and adiponectin, highlighting its dual role in this context. Adiponectin activates the AMP-activated protein kinase (AMPK)-dependent pathway to protect diabetic cardiomyocytes, exhibiting antiapoptotic properties associated with the hormone's protective effects. This involves modulating cellular energy homeostasis and mitigating myocardial stress. Notably, adiponectin also regulates an anti-cell death pathway through ceramide-S1P transition and promotes autophagy in an AMPK-independent signaling pathway. Adiponectin inhibits diabetes-induced release of miRNA 449b, an indicator of diabetic cardiomyopathy severity, through an AMPK-independent manner, underscoring the complexity of adiponectin's role and investigating mechanisms beyond traditional insulin sensitization. A comprehensive understanding of

adiponectin's intricate signaling in diabetic cardiomyopathy could pave the way for novel therapeutic interventions targeting this adipokine, offering new avenues to mitigate the impact of diabetic cardiomyopathy on cardiovascular health.

Key Words: Adiponectin; Diabetic Cardiomyopathy; AMPK; Autophagy; S1P; miRNA