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Down regulation of allograft inflammatory factor-1 prevents B cell infiltration to cardiac tissue during the development of type 1 diabetes

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Diabetes mellitus (DM) often causes chronic inflammation, hypertrophy, apoptosis and fibrosis in the heart and subsequently leads to myocardial remodeling, deteriorated cardiac function and heart failure. However, the etiology of the cardiac disease is unknown. Therefore, we assessed the gene expression in diabetic and nondiabetic groups of mouse left ventricular hearts using Affymetrix microarray analysis. Of the 517 inflammatory genes altered in the diabetic hearts, 97 and 61 at 4 weeks and 8 weeks respectively were up- or down-regulated at fold change >1.3 with $P<0.05$ vs. control, 23 genes from each time point were identified as B cell inflammatory genes responsible for humoral immunity, one of the top of these genes is allograft inflammatory factor-1 (AIF-1) which is associated with B cell function during inflammatory responses. Real–time reverse transcriptase-polymerase chain reaction confirmed the Affymetrix data. CD19 B cell marker and AIF-1 were both downregulated in diabetic hearts at 4W and 8W as compared to control. Interestingly, we showed for the first time that AIF-1 is responsible for B cell migration to the myocytes. This study suggests that diabetes attenuates AIF-1 expression, and this, in turn, prevents B cell migration that may lead to an increase of inflammation in the heart.

Biography

Khadija Rafiq has her expertise in immunology and cellular biology. Over the past several years she has been investigating how the immune system affects cardiac myocyte growth and cardiac function with a focus on signaling molecules that are activated by inflammatory proteases. Her research interest focuses on elucidating the role of inflammatory serine proteases in the development of diabetic cardiomyopathy. The goals of her research are to identify novel signaling mechanisms that control cardiac cell growth and apoptosis.

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