CHRONIC HEART FAILURE AND CELLS IMMUNE ACTIVATION

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Abstract: Chronic heart failure (CHF) is a major public health concern in developed countries, and the introduction of new treatments has improved its prognosis in recent years. However, the immunological aspects of the disease are still not directly targeted. Chronic immune activation with increased levels of pro-inflammatory substances in the blood remains an important characteristic of the disease, regardless of its underlying cause. Autoimmune mechanisms are involved in a subset of patients with dilated cardiomyopathy, but the interaction between these two systems is not yet fully understood. This review summarizes the immune and autoimmune aspects of CHF. I-10, originally referred to as cytokine synthesis inhibitory factor (CSIF), was discovered as a product of Th2 cells that suppresses the production of IFN-3 by Thl cells. It joins TGF-fl as one of the few lymphocyte suppressor factors that have been purified and cloned.

Key words:chronic heart failure, coronary heart disease, proinflammatory cytokine, CD4,CD8.

The mechanisms by which these suppressor factors influence lymphocyte function are not fully understood. I-10 inhibits Th1 cells only in the presence of accessory cells, particularly macrophages (mOb). This suggests that it might primarily act on mOb to alter the balance between their stimulating and inhibiting secretory products, thereby affecting lymphocytes indirectly. Only a few purified cytokines are known to inhibit the function of macrophages. In fact, two cytokines have been identified that block or reverse the activation of mouse macrophages: TGF-β (including TGF-α, -β, and -γ) and macrophage deactivating factor (MDF). Monocytes and macrophages have a reparative effect in ischemic heart tissue after MI. As their activation mainly occurs in acute phase, other components also regulate repair mechanism from acute to chronic phase. The adaptive immune response, especially T cells, participate in those cascades including myofibroblasts transition. CD4+ T helper (Th) cells provide proper immune cell homeostasis and host defense but CD4+ T cells have also been shown to promote autoimmune and inflammatory diseases. CD8+ T-cells also have been involved in both beneficial and detrimental cardiac remodeling. CD8+ T cells, which have angiotensin II receptors, infiltrate the peri-infarct myocardium 7 days after MI. Thus, the development and progression of congestive heart failure (CHF) in ischemic etiology is associated with a variety of complex immune processes. It is important to note that the reactions of the immune system are typically compensatory, and therefore, the pro-inflammatory effects of immune cells during the initial stage of cardiomyocyte injury are necessary, as well as their anti-inflammatory effects. However, the development of maladaptive myocardial remodeling leading to CHF is due to an imbalance between pro-inflammatory and anti-inflammatory immune responses.