Associations of interleukin-17 and monocyte chemoattractant protein-1 with vascular lesions in patients with rheumatoid arthritis.

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Abstract

This study aimed to investigate the associations of Interleukin-17 (IL-17) and monocyte chemoattractant protein-1 (MCP-1) with vascular lesions in patients with rheumatoid arthritis (RA). Thirty RA patients (RA group) and thirty healthy subjects (control group) were enrolled in this study. In all subjects, the serum levels of rheumatoid and inflammation related indexes including rheumatoid factor (RF), erythrocyte sedimentation rate (ESR), tumor necrosis factor-α (TNF-α), highsensitivity C-reactive protein (hs-CRP), IL-17 and MCP-1 were determined. In addition, the brachial-ankle pulse wave velocity (baPWV) and ankle-brachial index (ABI) were measured. Results showed that, the levels of ES, RF, serum TNF-α, hs-CRP, IL-17 and MCP-1 in RA group were significantly higher those in control group, respectively (P<0.05). The levels of serum TNF-α, hs-CRP, IL-17 and MCP-1 in RA patients with abnormal baPWV (>1400 cm/s) were significantly higher those in RA patients with normal baPWV (≤1400 cm/s), respectively (P<0.05). The levels of serum hs-CRP, IL-17 and MCP-1 in RA patients with abnormal ABI (≤0.9) were significantly higher those in RA patients with normal ABI (>0.9), respectively (P<0.05). In 30 RA patients, hs-CRP, IL-17 and MCP-1 were the main independent risk factors of RA patients with abnormal baPWV, and IL-17 and MCP-1 were the main independent risk factors of RA patients with abnormal ABI. In conclusion, IL-17 and MCP-1 are involved in the occurrence and development of RA, and they are the reliable indicators for judging the vascular lesions in RA.

Keywords: Rheumatoid arthritis, Vascular lesions, Interleukin-17, Monocyte chemoattractant protein-1.

Introduction

Rheumatoid arthritis (RA) is a systemic autoimmune disease which is typically characterized by chronic symmetric non-suppurative arthritis. It seriously affects the health and life quality of patients. The main clinical manifestations of RA include joint involvement, weight loss and fatigue and other symptoms. For severe cases, the function of joint is seriously affected [1,2]. Vascular lesion is one of the manifestations of extra-articular lesions of RA, which is mainly characterized by vessel wall invasion of lymphocytes, neutrophils and plasma cells, intimal hyperplasia, fibrin necrosis or thrombosis [3,4]. The cardiovascular events account for 42% of the cause of death in RA patients [5]. The atherosclerosis is the main lesion in the cardiovascular events of RA [6]. Previous studies have shown that, the increase of vascular stiffness is closely related to the mortality rate of cardiovascular events [7,8]. It is found that, the arterial stiffness exists in patients with RA [9]. The early assessment of the vascular lesions in RA patients and the related early intervention are important for improving the prognosis of RA. At present it is agreed that the inflammatory response is a common pathway of atherosclerosis occurrence and progression and rupture of plaques for thrombosis formation, and a large number of inflammatory cells and inflammatory mediators are involved in this pathway [10]. Interleukin-17 (IL-17) is a potent cytokine that induces the inflammatory response. It plays an important role in a variety of autoimmune diseases. The function of IL-17 in atherosclerosis has also been extensively studied, but the conclusions are still controversial [11,12]. Monocyte chemoattractant protein-1 (MCP-1) is a specific monocyte chemoattractant factor. It can promote the formation of atherosclerosis and plays an important role in the early stage of atherosclerosis [13]. This study investigated the association of interleukin-17 and MCP-1 with vascular lesions in patients with RA. The objective was to further elucidate the mechanisms of IL-17 and MCP-1 in artery atherosclerosis in RA patients, which was conducive to early detect and change the inflammatory state of RA, and provide new ideas for the treatment of vascular lesions of RA.