The chronic inflammation-enhanced atherosclerosis syndrome

Recent decades have brought a vast body of evidence that atherosclerosis is an inflammatory disease [1, 2]. It seems clear that the development of atherosclerosis is stimulated by a number of factors known as risk factors. On the other hand, more and more reports indicate an association of chronic autoimmune disorders with accelerated development of atherosclerosis and its sequel, especially cardiovascular diseases. This phenomenon has been shown in patients with rheumatoid arthritis, systemic lupus erythematosus [3, 4], seronegative spondyloarthropathy [5], psoriatic arthritis [6], and some other diseases. The incidence of cardiovascular events or premature death rate due to these events is significantly higher in these patients [7].

The aim of this letter is to propose a new syndrome, the chronic inflammation-enhanced atherosclerosis syndrome (CIEAS). The syndrome is characterized by extensive progressive atherosclerosis and results from chronic inflammatory disease, especially of autoimmune origin. The clinical features of CIEAS are similar regardless of the causative disease, and this is the main reason for identification of the syndrome. It is possible that both prevention and treatment of CIEAS will be the same or similar despite various diseases considered as its primary cause.

The mechanism of CIEAS is suggested to be complex. The primary autoimmune inflammatory process is probably the most potent mechanism. Several phenomena related to inflammation (e.g. cytokines) have been proposed to enhance atherosclerosis [8], and there are suggestions that some pathways of atherosclerosis development are of autoimmune nature [9]. Additionally, management of systemic disorders of the connective tissue is associated with potential atherosclerosis-accelerating mechanisms (e.g. long-lasting medication with glucocorticoids), and plasma lipid disturbances are found to be common in these patients. Other risk factors (e.g. impaired physical activity in patients with musculoskeletal disorders) may also contribute to development of the syndrome.

It is an open question whether CIEAS may be a sequel of chronic inflammation of other than autoimmune nature. This question cannot be answered definitely because a significant proportion of infections are cured successfully or not cured, ending with loss of the patient; thus fewer patients have long-lasting infection-induced inflammation. Chronic immunosuppression, which is a commonly used therapeutic method in patients with autoimmune diseases, is not applied in those with identifiable infections. There is however some evidence that infection enhances development of atherosclerosis and/or is involved in autoimmunity. These findings suggest that CIEAS is not limited to only autoimmune inflammatory diseases.

In summary, CIEAS is proposed as a common late sequel occurring in patients with long-lasting inflammation, and is revealed currently when survival of these patients is becoming longer due to progress in disease treatment. Recognition of CIEAS may facilitate management of patients, and further studies on the role of various therapeutic methods or other factors controlling the development of CIEAS are needed.

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