Coagulation Factors in Multiple Sclerosis may Represent Diagnostic and Therapeutic Strategies

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Abstract

Multiple sclerosis (MS) is a neuroinflammatory autoimmune disease which mediated by various molecular and cellular immune components. However, recent reports have shown that coagulation factors that traditionally separate from the immune system might also be involved in MS development and progression. Studies on experimental autoimmune encephalomyelitis (EAE) and human MS patients report alterations of some factors of the coagulation cascade such as fibrin, thrombin, prothrombin, factor X, and FXII to confirm that coagulation factors have an important role in pathogenesis of autoimmune inflammatory disorders. Recent studies report that genetic deficiency or pharmacologic blockade of FXII significantly protected from EAE and also fibrin depletion, either genetically or using anticoagulants, significantly reduces neurolinflammation and axonal damage in EAE. Another important study shows that increase thrombin activity is an early event and increases with progression of neuroinflammatory disease, with noted microglial activation and axonal damage. In this review, we aim to evaluate elevated coagulation factors of tissue or blood as a new therapeutic strategy for the treatment of MS or other neuroinflammatory disorders. As we described, some coagulation factors such as fibrin and thrombin are significantly increased in MS and blockade of these factors in EAE improve neurolinflammation and axonal damage. These findings might lead to the development of anticoagulants as potential therapeutic strategies for MS.

Keywords: Coagulation factor, Multiple sclerosis, MS, Neuroinflammation

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