The Effect of Platelet Activating Factor on Inflammatory Response in Multiple Sclerosis

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Abstract

Multiple sclerosis is an autoimmune disease of the central nervous system which its main characteristic is an inflammation and demyelination and subsequent, neural degeneration. Many studies have shown that inflammation causing neuronal demyelination. MS is the most common cause of chronic neurological disability in during youth which the prognosis is that can be death. Platelet activating factor is a phospholipid mediator in central nerve system which acts as a messenger and plays role in platelet aggregation and inflammatory responses. Furthermore, these inflammatory mediators involve in many pathophysiological processes such as brain edema reperfusion injury through interactions with its receptor. PAFR (platelet activating factor receptor) is a seven transmembrane proteins that belongs to G protein receptor and express in many brain cells such as neurons and microglia. Expression of this receptor causes the release of many inflammatory cytokines like IL-6, IL-1β and TNF-α and also apoptosis marker including caspase-3 and bax/bcl-2. PAF and its receptor provides a strong inflammatory response and increased inflammation and recurrence of the disease. Thus, blocking the path connecting and interacting PAF and PAFR can significantly reduce inflammation and protect nerve cells. We hypothesized that Ablation of PAFA gene for example through knock-out can prevent the binding PAF to PAFA and realizing inflammatory cytokines so this can be a convenient way to reduce inflammation and recurrence of the disease. We suggest that it can be great target treatment in patients with relapsing-remitting MS.

Keywords: Inflammation, Multiple sclerosis, Platelet activating factor

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