The Role of Neuroinflammation in Post-Traumatic Stress Disorder (PTSD)

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Abstract

Post-traumatic stress disorder (PTSD) is an anxiety disorder that can typically occur after traumatic events in which the individual felt severe helplessness or horror. PTSD has a lifetime prevalence of 5–8% and women show higher prevalence than in men. Neuroinflammation is associated with anxiety and related disorders such as PTSD. It is an early, specialized immune reaction following tissue damage and/or pathogen invasion in the central nervous system (CNS). Regarding the importance of anxiety disorders especially PTSD and their effect on patients’ personal and social life, considering the neuroinflammation in PTSD can be used for new treatment. Several studies have been reported that there is a relationship between neuroinflammation and PTSD. Current epidemiological evidence shows that increased expression of pro-inflammatory cytokines such as interleukin-1β, interleukin-6, and tumor necrosis factor-α, and decreased anti-inflammatory factors have a key role in creating PTSD. Also, neuroinflammation induced by inhibition of the NADPH oxidase (NOX2) can lead to trigger the PTSD symptoms. Various inflammatory markers such as Cortisol, C-reactive protein (CRP), Th1 cytokines, and Th2 cytokines are associated with PTSD. Furthermore, microglial activation may also initiate inflammation and can be involved in PTSD. Thus, scrutinized research about PTSD and understanding of creating mechanisms of this disorder is essential and can provide grounds for its treatment.

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