

Review Article: The Role of Microglia as Prime Component of CNS Immune System in Acute and Chronic Neuroinflammation

Abstrak :

The understanding of microglia function in central nervous system (CNS) immune system has changed the neuroscientists' view on CNS response to activating stimuli. Once microglia were considered a supporting cell, in the last 2 decades however they have been recognized as the prime component of CNS immune system due to their ability to actively respond to both acute and chronic/persistent stimuli by proliferation and production of pro-inflammatory cytokines and other toxic substances. Many studies have shown that pro-inflammatory cytokines produced by microglia play role in secondary CNS injury. Many other studies however have established that potentially toxic mediators (e.g. glutamate, Ca²⁺ and reactive oxygen species/ROS) released in traumatic CNS injury play prominent role in secondary CNS injury. We think therefore that the role of pro-inflammatory cytokines in secondary CNS injury is uncertain. This uncertainty is supported by the finding that pro-inflammatory cytokines affect only damaged neurons, which are caused by those potentially toxic mediators. The dual inflammatory response of cytokines also supports this uncertainty. On the basis of the above findings, we therefore suggest studies investigating the role of microglia in contributing secondary CNS injury and the effectiveness of suppressing microglia response to produce toxic substances to improve the outcome of CNS injured patients should be carried out. It has been proposed that neuroinflammation precedes neuronal loss in many neurological disorders such in Alzheimer's disease (AD). Non-steroid anti-inflammation drugs (NSAIDs) therefore have been widely used in the management of AD. Many studies however have shown that NSAIDs are not effective to suppress the progressive neuronal loss in AD. Looking at the responsible of microglia (neuroinflammation) in neuronal loss and the failure of NSAIDs in improving outcomes of AD patients, we therefore hypothesize that suppressing microglia response to produce toxic substances may account for reducing neuronal loss in AD.

Keyword :

neuroinflammation, microglia, cytokines