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An Overview of Neuroinflammation

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Introduction

Inflammation of the neurological system is known as neuroinflammation. It can be triggered by a multitude of factors, including infection, traumatic brain injury, toxic metabolites, or autoimmune, to name a few. Microglias are resident innate immune cells in the Central Nervous System (CNS), which includes the brain and spinal cord that are activated in response to these stimuli. Because peripheral immune cells are normally restricted by the Blood–brain Barrier (BBB), a specialised structure made up of astrocytes and endothelial cells, the CNS is typically an immunologically privileged region. However, circulating peripheral immune cells may pass through a weakened BBB and come into contact with neurons and glial cells that express major histocompatibility complex molecules, causing the immune response to continue. Although the response is launched to protect the central nervous system from the infectious pathogen, it has the potential to cause toxic inflammation and additional leukocyte migration via the blood–brain barrier [1].

Description

Neuroinflammation is a term used to describe persistent inflammation of the central nervous system as opposed to acute inflammation. Acute inflammation is characterised by inflammatory markers, endothelial cell activation, platelet deposition, and tissue edoema, and occurs shortly after central nervous system damage. Chronic inflammation is the activation of glial cells and the migration of other immune cells into the brain on a long-term basis. Chronic inflammation is most commonly linked to neurological disorders. Chronic neuroinflammation can be caused by a variety of factors, including:

- Toxic metabolites are a type of toxic metabolite
- Autoimmunity
- Aging/Microbes/Viruses
- Traumatic Brain Injury (TBI) is a type of brain injury
- Injury to the spinal cord
- Pollution of the air
- Smoke that is passive

Viruses, bacteria, and other infectious organisms stimulate the body's defensive systems, causing immune cells to defend the targeted location. Some of these invasive infections can cause a significant inflammatory response, compromising the blood-brain barrier's integrity and altering the flow of inflammation in adjacent tissue. The location of the infection, as well as the type of infection, can influence the sort of inflammatory response that is triggered and whether or not specific cytokines or immune cells are activated [2,3].

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Neuroimmune response

Glial cells: The innate immune cells of the central nervous system are known as microglia. In response to neurological damage, microglia actively surveys their environment and modifies their cell shape dramatically. Rapid activation of microglia is a hallmark of acute inflammation in the brain. There is no peripheral immunological response at this time. Chronic inflammation, on the other hand, causes tissue and the blood–brain barrier to deteriorate over time. Microglia produces reactive oxygen species and send out signals to attract peripheral immune cells for an inflammatory response during this time.

Glial cells, or astrocytes, are the most abundant cells in the brain. They play an important role in the preservation and support of neurons and make up a large part of the blood–brain barrier. Astrocytes may become activated in response to signals released by injured neurons or activated microglia after a brain insult, such as traumatic brain injury. Astrocytes may release a variety of growth factors and undergo morphological changes if triggered. After an injury, astrocytes develop a glial scar, which is made up of a proteoglycan matrix that prevents axonal regrowth. Recent research has showed that glia scarring is not harmful to axonal regeneration, but rather advantageous [4].

Cytokines: Cytokines are a type of protein that controls inflammation, cell signalling, and a variety of cell functions like growth and survival. Chemokines are a type of cytokine that regulates cell migration, for example, drawing immune cells to an infection or injury location. Various cell types in the brain, such as microglia, astrocytes, endothelial cells, and other glial cells, can produce cytokines and chemokines. Chemokines and cytokines are neuromodulators that govern inflammation and development in the body. In a healthy brain, cells emit cytokines to create a local inflammatory milieu that attracts microglia and allows infection or injury to be cleared. In neuroinflammation, however, cells may produce a steady stream of cytokines and chemokines, compromising the blood–brain barrier. These cytokines attract peripheral immune cells to the site of injury, allowing them to penetrate the blood-brain barrier and enter the brain. Interleukin-6 (IL-6) is produced during astrogliosis, while interleukin-1 beta (IL-1) and tumour necrosis factor alpha (TNF-) are neuronal cytotoxic cytokines that are released in response to brain injury. Despite the fact that pro-inflammatory cytokines can cause cell death and consequent tissue damage, they are required for tissue repair. TNF-, for example, induces neurotoxicity in the early phases of neuroinflammation but promotes tissue development in the later stages [5].

Peripheral immune response

The blood–brain barrier is a barrier formed by endothelial cells and astrocytes that separates the brain from circulating blood. In terms of physiology, this protects the brain from potentially hazardous substances and cells in the blood. Astrocytes produce tight junctions, which mean they can control what passes through the blood–brain barrier and into the interstitial space. The blood–brain barrier may become permeable to circulating blood components and peripheral immune cells after damage and persistent production of inflammatory agents such as chemokine. Macrophages, T cells, and B cells, which are implicated in both innate and adaptive immune responses, may then infiltrate the brain. This contributes to chronic neuroinflammation and neurodegeneration by exacerbating the brain's inflammatory environment [6].

Aging: Cognitive impairment and an increased risk of acquiring neurodegenerative disorders like Alzheimer's disease are common side effects of ageing. Increased inflammatory markers appeared to hasten the ageing of the brain. There are continuously increasing levels of pro-inflammatory cytokines and decreased levels of anti-inflammatory cytokines in the aged brain without any obvious pathology. One factor that raises the

risk of neurodegenerative illness is a homeostatic imbalance between anti-inflammatory and pro-inflammatory cytokines that occurs as people age. In addition, elderly brains have a higher number of activated microglia, which express higher levels of MHC II, ionised calcium binding adaptor-1 (IBA1), CD86, ED1 macrophage antigen, CD4, and leukocyte common antigen. The ability of neurons in the hippocampus to undergo long-term potentiation (LTP) is reduced by this activated microglia [7].

Role in neurodegenerative disease

Alzheimer's disease: Two key hallmarks of Alzheimer's disease (AD) have been identified in the past: neurofibrillary tangles and amyloid-beta plaques. Neurofibrillary tangles are insoluble tau protein clumps, whereas amyloid-beta plaques are extracellular amyloid-beta protein deposits. Current thinking in Alzheimer's pathology goes beyond these two conventional characteristics, suggesting that neuroinflammation is responsible for a considerable amount of Alzheimer's neurodegeneration. In post-mortem AD brains, activated microglia are abundant. Microglia stimulated by inflammatory cytokines is thought to be incapable of phagocytosing amyloid-beta, which may contribute to plaque buildup rather than removal. In addition, the inflammatory cytokine IL-1 is elevated in Alzheimer's disease and is linked to synaptophysin deficiency and synapse loss. Individuals who frequently take non-steroidal anti-inflammatory medicines (NSAIDs) have been related with a 67 percent reduction in the beginning of AD (compared to the placebo group) in a four-year follow-up study, adding to the evidence that inflammation is linked to disease progression in AD. Increased inflammatory markers were linked to faster brain ageing, which could explain the link to neurodegeneration in AD-related brain areas [8].

Multiple Sclerosis: Multiple sclerosis (MS) is the most frequent neurological illness that disables young individuals. It is characterised by demyelination and neurodegeneration, which contribute to cognitive impairments, limb weakness, and weariness, among other symptoms. Inflammatory cytokines break down the blood–brain barrier, allowing peripheral immune cells to enter the central nervous system and cause multiple sclerosis. B cells and plasma cells create antibodies against the myelin sheath that insulates neurons as they move into the central nervous system, damaging the myelin and delaying conduction in the neurons [9].

Role as a therapeutic target

Drug therapy: Because neuroinflammation has been linked to a number of neurodegenerative illnesses, researchers are increasingly interested in seeing if lowering inflammation can reverse neurodegeneration. Neuronal loss in neurodegenerative illnesses is reduced by inhibiting inflammatory cytokines like IL-1. Interferon-B, Glatiramer acetate, and Mitoxantrone are three current therapies for multiple sclerosis that work by lowering or blocking T cell activation, but have the adverse effect of systemic immunosuppression. The usage of nonsteroidal anti-inflammatory medicines (NSAIDs) reduces the chance of getting Alzheimer's disease.

Exercise is a viable preventative and treatment method for a variety of disorders characterised by neuroinflammation. Aerobic exercise is commonly used to lower peripheral inflammation by activating defensive systems in the body that help to balance the internal environment. Exercise has been demonstrated to lower the proliferation of microglia in the brain, the expression of immune-related genes in the hippocampus nucleus, and the expression of inflammatory cytokines like TNF [10].

Conclusion

The concept of neuroinflammation is broad and encompasses two large areas of biological science in the nervous and immune systems. We are just now beginning to parse out the positive and negative aspects of immune system-nervous system interaction. It is difficult to make generalized conclusions about the positivity or negativity of neuroinflammation when considering systems as nuanced as those discussed in this review. Additionally, due to the complexity of the subject, we are unable to probe each topic discussed here as deeply as is necessary.

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