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By Modulating Microglia via the TLR4/Myd88/NLRP3/Casp1 Pathway, Endometrial Stem Cells Isolated by Menstrual Blood Suppress Neuroinflammation

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Abstract

Many neurological illnesses frequently result in neuroinflammation as a reaction. A possible therapeutic for conditions linked to neuroinflammation is mesenchymal stem cell therapy. Mesenchymal stem cell effects are debatable, and the underlying process is not well known. In the current study, a mouse model of neuroinflammation created by peripheral lipopolysaccharide injection received an intravenous transplant of endometrial stem cells generated from menstrual blood. Endometrial stem cell-derived conditioned media was used to cultivate microglial cells that had been exposed to lipopolysaccharide.

Keywords: Menstrual blood; Neuroinflammation; Endometrial stem cells

Introduction

Neuroinflammation is a common response that accompanies various disorders including infectious and noninfectious neurological and psychiatric diseases such as sepsis-associated encephalopathy, intracerebral haemorrhage, Alzheimer's disease (AD), Parkinson's disease (PD), depression and schizophrenia. Early inflammatory reactions contribute to neuronal repair and maintain homeostasis in the nervous system. However, chronic and aberrant inflammation leads to neuronal death and brain atrophy, which results in mental and behavioural abnormalities [1, 2].

MenSC transplantation reduced the neuroinflammatory response brought on by peripheral LPS treatment in NIM

According to microscopy studies, menSCs obtained from healthy female donors exhibited a spindle fibroblast-like shape and spiral development. According to the results of flow cytometry, MenSCs were not haematopoietic cells but rather MSCs since they were primarily positive for CD73, CD90, and CD105, only marginally positive for CD44, and negative for CD11b, CD19, CD34, CD45, and HLA-DR.

The initial line of immunological defence in the brain system is provided by resident immune cells called microglia. Microglia's that are at rest keep an eye on brain homeostasis. Microglia release cytokines and mediators to aid in the removal of pathogens and nerve tissue regeneration after becoming triggered by infections, inflammatory agents, or brain injury. The pioneering cells known as microglia start the inflammatory response in the brain, influencing astrocytes, neurons, and other cells to start their own inflammatory responses. Dysfunctional microglia continuously releases proinflammatory substances under pathophysiological circumstances, causing neuronal death and nerve damage. Recent transcriptome research and single [3, 4].

Microglia exhibit geographical and pathological variability, which may reflect different functional groups of microglia, according to recent investigations of the transcriptome and single cell sequencing. These results imply that microglia treatments might be an effective way to treat disorders connected to neuroinflammation [5].

Several substances are secreted by mesenchymal stem cells (MSCs), which also have immunomodulatory properties. MSCs have a promising future in the therapy of organ dysfunction brought on by inflammation, according to recent studies. By paracrine signalling, organelle transmission, exosome transfer, implantation, and differentiation, MSCs reduce disease symptoms and shield the organs from damage. Immunomodulation, antioxidant activity, anti-apoptotic activity, metabolic regulation, and autophagy may all play a role in how these effects work. However, the effects of MSCs on the neuroinflammatory response in the brain have only been documented in a small number of researches, and the findings are debatable [6, 7].

Conclusion

The current study provided the first proof that MenSCs and conditioned media were efficient at reducing the inflammatory response in microglia and a mouse model of neuroinflammation. In an inflammatory setting, MenSCs restored the functions of microglia and prevented their hyper activation. Moreover, both in vivo and in vitro, MenSCs altered the TLR4/MyD88/NLRP3/Casp1 signalling pathway [8, 9, and 10].

Acknowledgement

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Conflict of Interest

None.

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