

Clinical Anatomical Perspective of Skull Bone Marrow's Contribution to Neuroimmune Regulation

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ABSTRACT

Inflammation is a protective immune response to injury or infection, promoting tissue repair and pathogen elimination. However, when it becomes dysregulated, it can worsen tissue damage, especially in brain injuries. The blood-brain barrier (BBB) typically protects the brain from immune cell infiltration, but during injuries, it can be compromised, leading to neuroinflammation and further damage. Recent research has highlighted the importance of skull bone marrow (SBM) in the brain's immune response, a concept once overlooked. SBM offer a direct pathway for immune cells to move between the skull and the brain, enhancing the brain's immune defense. These cells, which are closer to the cerebrospinal fluid (CSF), show distinct molecular profiles that support tissue repair and immune modulation rather than promoting excessive inflammation. SBM play a key role in regulating immune responses during conditions like brain injuries, infections, and neurodegenerative diseases. Their ability to support both pro-inflammatory and anti-inflammatory responses makes them crucial for CNS immunity and recovery. The aim of this work is to investigate the role of SBM in central nervous system (CNS) immunity, particularly during brain injury, infection, and neurodegenerative disease. The current review seeks to highlight SBM's unique contribution to immune regulation and tissue repair in the brain, emphasizing its potential as a therapeutic target for modulating neuroinflammation and improving clinical outcomes in neurological disorders. Future research on skull bone marrow could lead to new therapeutic strategies for treating neurological disorders and improving clinical outcomes.

Vol No: 09, Issue: 01 Received Date: April 29, 2025 Published Date: May 27, 2025

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Citation: Ahmed AS, et al. (2025). Clinical Anatomical Perspective of Skull Bone Marrow's Contribution to Neuroimmune Regulation. Mathews J Neurol. 9(1):31.

Copyright: Ahmed AS, et al. © (2025). This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. **Keywords:** Inflammation, Blood-Brain Barrier, Skull Bone Marrow, Neuroinflammation.

INTRODUCTION

Inflammation is a vital response of the immune system to injury or infection, aimed at repairing tissues and eliminating pathogens. However, when this inflammatory response becomes excessive or prolonged, it can lead to tissue damage and hinder recovery. In the context of brain injuries, this phenomenon is particularly concerning. The blood-brain barrier (BBB) plays a central role in protecting the brain from harmful immune cells and pathogens under normal conditions. However, during brain injuries, the BBB becomes compromised, allowing immune cells to infiltrate the brain. This infiltration can trigger neuroinflammation, which, while initially protective, can lead to worsening neurological damage if unchecked [1].

The bone marrow, traditionally considered the primary source of immune cells, has a fundamental role in this process. It produces various immune cells that circulate throughout the body and are mobilized during injury or infection. While the bone marrow's role in immune defense has been well-established (Figure 1), its involvement in the brain's immune response has been less clear [1]. Recent studies have raised significant questions about how immune responses are coordinated at various injury sites within the brain, especially when considering how immune cells from the bone marrow interact with the brain tissue [2].



Figure 1. Original diagram illustrates bone marrow's role in generating immune cells like lymphocytes and macrophages, which circulate through the bloodstream and lymphatic system to reach the brain, where they contribute to immune responses at injury sites and interact with brain tissue.

Historically, the brain has been considered an immuneprivileged organ, meaning it was thought to be largely isolated from the peripheral immune system to prevent inflammation that could damage delicate neural structures [2]. However, emerging research has challenged this notion, revealing a much more complex relationship between the brain and the immune system. Structures like the meninges, choroid plexus, and perivascular spaces are now recognized as crucial components in both immune surveillance and repair mechanisms within the brain [1]. The discovery of newly identified microvascular channels, known as skull bone marrow (SBM) channels, has provided further

insight into this process. These channels serve as a direct communication pathway between the skull's bone marrow and the brain, allowing immune cells to migrate between these regions and thus strengthening the brain's immune response [3].

The skull bone marrow (SBM) has emerged as a vital contributor to central nervous system (CNS) immunity, particularly under pathological conditions such as infection, ischemia, and brain injury. In these states, SBM becomes activated, increasing immune cell production and directing these cells to the meninges—the protective membranes surrounding the brain—where they help regulate immune responses and promote tissue repair [3]. Anatomical studies reveal that SBM is densely distributed throughout the frontal, parietal, and occipital regions of the skull, with species-specific differences in structure and connectivity. Notably, in humans, skull bone marrow contains larger cavities and more extensive connections to the meninges compared to those in animal models, indicating a potentially greater role in immune modulation within the human brain [1]. This structural complexity may enable more robust or nuanced immune responses during CNS stress or injury. The discovery of the SBM's active role challenges the longstanding assumption that brain immune cells are exclusively derived from peripheral sources. Instead, it highlights the skull as a local, specialized site of immune support. Recognizing the importance of SBM not only deepens our understanding of neuroimmunology but also opens new avenues for diagnostic and therapeutic strategies targeting CNS disorders, emphasizing its role in brain resilience and recovery [4].

SKULL BONE MARROW AND PERIPHERAL BONE MARROW IN CNS IMMUNITY

Bone marrow, as a site of hematopoiesis, is the primary source of the body's immune cells, which play a pivotal role in defending against infections, repairing tissue, and maintaining homeostasis [3]. However, when considering the brain's immune system, the contribution of skull bone marrow is distinct from that of peripheral bone marrow, particularly during pathological events such as brain injury, neuroinflammation, or infection [5].

The primary difference between skull and peripheral bone marrow lies in their anatomical proximity and access to the brain [4]. Peripheral bone marrow, located in distant bones such as the femur, pelvis, and vertebrae, is generally isolated from the brain by the BBB (Table 1). The BBB is a selective barrier that protects the brain from potentially harmful substances, including immune cells, under normal conditions [5]. This separation means that peripheral bone marrow-derived immune cells can only reach the brain under specific conditions where the BBB is compromised, such as during infection, injury, or neuroinflammation [6].

Table 1. Comparison Between SBM and Peripheral Bone Marrow (PBM) in CNS Immunity

Characteristic	SBM	PBM
Anatomical Location	Located within the skull, in close proximity to the CNS	Located in distant bones (femur, pelvis, vertebrae), isolated by BBB
Access to CNS	Direct communication with cerebrospinal fluid (CSF) and CNS regions	Access to the brain is indirect, through the compromised BBB
Role in Immune Response	Rapid immune cell mobilization to the brain, supporting local immunity	Immune cells must travel through the bloodstream to reach the CNS
Immune Cell Characteristics	Immune cells exhibit regulatory and tissue repair properties	Immune cells often express pro-inflammatory markers
Specialization	Regulates CNS-specific immune responses, controls neuroinflammation	Primarily responds to systemic infections and injuries

In contrast, skull bone marrow is positioned in close proximity to the brain and has direct access to CSF, a vital medium in the regulation of the brain's immune system. The SBM enable this close interaction, creating a direct pathway for immune cells to migrate from the skull to the meninges and other parts of the CNS [5]. This unique location and access allow skull bone marrow to participate in immune responses in the CNS more quickly and efficiently than peripheral bone marrow. This spatial advantage positions skull bone marrow as a critical player in supporting the immune system within the brain, especially during pathological events [7].

MOLECULAR DIFFERENCES IN IMMUNE SIGNATURES

One of the most striking distinctions between skull and peripheral bone marrow is the molecular profile of the immune cells they generate. While both bone marrow sources produce a variety of immune cells, skull bone marrow-derived immune cells exhibit unique characteristics that are particularly suited to responding to CNS-specific challenges [8].

In peripheral bone marrow, immune cells like neutrophils, monocytes, and macrophages typically express high levels of pro-inflammatory molecules such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and C-C motif ligand 5 (CCL5) [9]. These molecules are typically associated with heightened inflammation and immune activation in response to infections or injuries [7]. However, immune cells derived from skull bone marrow show a different molecular profile [9]. They exhibit lower expression of these pro-inflammatory molecules and instead upregulate genes that are involved in tissue repair, metabolic balance, and the resolution of inflammation [4]. These observations suggest that skull bone marrow-derived immune cells are more likely to play a regulatory or immunomodulatory role (Figure 2), which is especially important during neuroinflammation, where excessive inflammation can result in additional damage to brain tissue [10].



Figure 2. Original diagram presenting peripheral bone marrow immune cells produce pro-inflammatory molecules like IL-6, TNF- α , and CCL5. In contrast, skull bone marrow-derived immune cells show lower inflammation and focus on tissue repair and inflammation resolution, suggesting a regulatory role in neuroinflammation.

This immunomodulatory role is particularly important in the context of chronic neurological diseases, where prolonged inflammation can contribute to disease progression and neurodegeneration. Studies of skull bone marrow-derived neutrophils, for example, have shown that these cells are involved in extracellular matrix organization and immune regulation, which can prevent excessive tissue damage [9]. This contrasts with peripheral neutrophils, which are often recruited to inflamed sites and exacerbate inflammation. These differences underscore the specialized functions of skull bone marrow in managing the immune response within the brain [11].

Interaction with CSF

Another critical distinction between skull bone marrow and peripheral bone marrow is their interaction with CSF, which serves as an essential medium for immune signaling in the CNS (Figure 3). CSF carries important biochemical signals that help regulate immune responses in the brain, particularly during injury or infection. Unlike peripheral bone marrow, which responds to systemic inflammatory signals, skull bone marrow is uniquely positioned to interact directly with CSF [10]. This direct access allows skull bone marrow to respond more quickly and specifically to local inflammatory cues [12].



Figure 3. Original diagram about immune signaling in the CNS involves interactions between neural and immune cells, notably microglia and astrocytes. This process regulates responses to injury and infection but, when dysregulated, contributes to neurodegenerative diseases.

During a brain injury or infection, the CSF carries signals that activate immune responses in the brain, prompting the skull bone marrow to produce immune cells such as neutrophils and macrophages. These cells then migrate to the meninges and other regions of the brain, where they help protect the CNS from further damage and promote healing. This direct communication pathway between the skull bone marrow and CSF is a key feature that distinguishes it from peripheral bone marrow. It enables the skull bone marrow to play a more integral role in the brain's immune defense, particularly under pathological conditions [13].

Future Research Directions

Immune cells originating from skull bone marrow exhibit expression of proteins implicated in synaptic signaling and immunoregulatory pathways, underscoring their role in sustaining immune homeostasis within the central nervous system. These cells demonstrate the capacity to engage with the brain's extracellular matrix, thereby facilitating their migration and integration into the CNS microenvironment, where they contribute to the orchestration of contextspecific immune responses [14]. In contrast, peripheral bone marrow-derived immune cells generally promote pro-inflammatory responses, which can exacerbate injury and prolong inflammation in the CNS. This difference in functionality suggests that skull bone marrow is more suited to meet the specialized immune needs of the brain (Figure 4), providing a more balanced and regulated immune response to injury, infection, and neurodegeneration [15].



Figure 4. Original diagram about skull bone marrow-derived immune cells express proteins involved in synaptic communication and immune regulation, supporting the brain's immune functions. Unlike peripheral cells, they avoid exacerbating inflammation, offering a more balanced response in the CNS. Future research should explore how these cells interact with CSF signals, potentially leading to new treatments for neuroinflammatory and neurodegenerative conditions, as well as brain injuries.

Given the specialized role of skull bone marrow in the immune response within the CNS, future research should focus on understanding the complex mechanisms by which skull marrow-derived immune cells interact with CSF signals [11]. Investigating how these cells influence neuroinflammation, tissue repair, and the resolution of brain injuries could lead to new therapeutic approaches that target these pathways [10]. By modulating skull bone marrow-derived immune cells or the CSF signaling pathways that regulate them, it may be possible to develop novel treatments for a wide range of neurological diseases, including neuroinflammatory disorders, neurodegenerative diseases, and traumatic brain injuries [16].

BONE MARROW-DERIVED IMMUNE CELLS AND NEUROLOGICAL DISORDERS

The specialized role of skull bone marrow in CNS immunity becomes even more significant when considering various neurological disorders. Skull bone marrow-derived immune cells exhibit unique characteristics that are particularly beneficial in managing immune responses to CNS-specific injuries or diseases [17].

Multiple Sclerosis (MS)

In MS, a chronic autoimmune disease characterized by demyelination in the CNS, skull marrow-derived immune cells

play an important role in modulating inflammation. Myeloid cells from SBM, such as monocytes and neutrophils, exhibit anti-inflammatory properties that help resolve inflammation and promote tissue repair during both the acute and chronic phases of MS. This contrasts with peripheral-derived immune cells, which are typically more pro-inflammatory and contribute to the progression of disease [16]. Targeting skull bone marrow-derived immune cells in MS therapy could therefore help modulate inflammation and improve disease outcomes [18].

Stroke

Following a stroke, skull bone marrow-derived neutrophils and monocytes rapidly infiltrate the infarcted area and contribute to both the inflammatory response and recovery process [16]. These SBM-derived cells exhibit unique gene expression profiles compared to peripheral-derived immune cells, underscoring the specific role that skull bone marrow plays in stroke recovery. Understanding the unique characteristics of SBM-derived immune cells in stroke could lead to novel therapeutic strategies to enhance recovery and minimize secondary injury [19].

Traumatic Brain Injury (TBI)

Traumatic brain injury triggers a complex immune response, with both beneficial and detrimental effects. Skull

marrow-derived immune cells, particularly neutrophils and monocytes, contribute to both the inflammatory response and the repair process following TBI [18]. These cells exhibit distinct phenotypes compared to peripheral-derived immune cells, highlighting the importance of skull bone marrow in managing immune responses and promoting recovery after brain injury [20].

Subarachnoid Hemorrhage (SAH)

In SAH, SBM-derived Ly6Chigh monocytes play a crucial role in mediating both inflammatory and anti-inflammatory responses, depending on the stage of the injury. Inhibiting CCL2 signaling, which is necessary for monocyte migration, impairs recovery, highlighting the dual role that SBM- derived immune cells play in both protecting the brain and promoting repair [21].

CNS Infections and Neurodegenerative Diseases

Skull bone marrow is also essential for immune responses to CNS infections, including bacterial meningitis, where it plays a key role in producing immune cells that protect the meninges and the brain. Moreover, in neurodegenerative diseases such as Alzheimer's and Parkinson's, SBM-derived immune cells influence neuroinflammation and disease progression (Table 2). Targeting these cells could provide a potential therapeutic approach to modulate immune responses in these diseases [22].

Table 2. Skull Bone Marrow-Derived Immune Cells in Neurological Disorders

Neurological Disorder	Role of SBM-Derived Immune Cells	Therapeutic Implications
Multiple Sclerosis (MS)	Modulate inflammation, promote tissue repair in both acute and chronic phases	Targeting SBM-derived cells could improve disease outcomes by modulating inflammation
Stroke	Neutrophils and monocytes infiltrate infarcted areas, contributing to inflammation and recovery	Understanding SBM-derived immune cells in stroke could enhance recovery and minimize secondary injury
Traumatic Brain Injury (TBI)	Neutrophils and monocytes involved in both inflammation and repair	Modulating SBM-derived immune cells could promote recovery after TBI
Subarachnoid Hemorrhage (SAH)	Ly6Chigh monocytes mediate inflammatory and anti- inflammatory responses	Targeting monocyte migration pathways could enhance recovery
CNS Infections and Neurodegenerative Diseases	Essential for immune responses, modulate neuroinflammation	Targeting SBM-derived immune cells for therapies in diseases like Alzheimer's and Parkinson's
CNS Tumors	SBM-derived neutrophils play a role in tumor microenvironment, differentiate into tumor- associated neutrophils (TANs)	SBM as a target for novel immunotherapies in glioblastoma and metastatic tumors

Aging and Immune Dysregulation

Aging impacts skull bone marrow function, leading to decreased immune cell production and increased peripheral infiltration. However, skull bone marrow shows resistance to age-related decline, maintaining its ability to produce immune cells that help mitigate neuroinflammation and protect against neurodegenerative diseases [23].

CNS Tumors and Immune Response

In CNS tumors such as glioblastoma (GBM), SBMderived neutrophils play a significant role in the tumor microenvironment. These cells differentiate into hybrid tumor-associated neutrophils (TANs) with antigenpresenting capabilities, offering potential for novel immunotherapies. Similarly, SBM is involved in the migration of tumor cells to the meninges in metastatic cancer, providing a novel target for therapeutic interventions [24].

CONCLUSION

Skull bone marrow plays a critical role in central nervous system (CNS) immunity by housing specialized immune cells that interact directly with cerebrospinal fluid (CSF), influencing neuroinflammation and neurodegeneration. Unlike peripheral marrow, skull marrow uniquely contributes to brain immune responses. Non-invasive imaging techniques such as TSPO-PET show strong potential for detecting skull marrow activity in conditions like Alzheimer's and multiple sclerosis, offering an alternative to invasive bone marrow biopsies (Table 3). Additionally, intracalvarial drug delivery, which bypasses the blood-brain barrier, significantly enhances therapeutic delivery to the brain. This emerging approach holds promise for improving treatment outcomes in CNS disorders and warrants further investigation. Table 3. Molecular Signatures of SBM-Derived Immune Cells vs. Peripheral Bone Marrow (PBM)-Derived Immune

Molecular Characteristic	SBM	Peripheral Bone
Pro-inflammatory Markers	Low expression of pro-inflammatory molecules (e.g., IL-6, TNF- α , CCL5)	High expression of pro-inflammatory molecules (e.g., IL-6, TNF- α , CCL5)
Immunomodulatory Genes	Upregulation of tissue repair and resolution of inflammation genes	Generally limited involvement in tissue repair and resolution of inflammation
Phenotype of Neutrophils	Involved in tissue repair, extracellular matrix organization, and immune regulation	Often exacerbate inflammation and contribute to prolonged injury
Interaction with CSF	Direct access to CSF, responds to local CNS signals	Responds to systemic inflammatory signals, limited interaction with CSF
Response to CNS Injury	Quickly mobilizes immune cells to support CNS recovery	Delayed and typically only activated during BBB disruption

Cells

AUTHOR CONTRIBUTION

- Ahmed S. Ahmed: Planned designed and final review of the manuscript.
- Jim Schank: Data collection, Data analysis, discussion of idea.
- Mark M. Rohn: Data collection, Data analysis, discussion of idea.
- Asim S. Khan: Data analysis, discussion of idea.
- Ehab M. Hantash: Data analysis.
- Liju S. Mathew: Data analysis.

FUNDING

The authors declare that no funds were obtained (The current study is self-funded).

DATA AVAILABILITY

On reasonable request, the data sets generated and analyzed during the current study are available if requested from the corresponding author.

ACKNOWLEDGEMENTS

None.

CONFLICT OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

REFERENCES

1. Harris MG, Hulseberg P, Ling C, Karman J, Clarkson BD,

Harding JS, et al. (2014). Immune privilege of the CNS is not the consequence of limited antigen sampling. Sci Rep. 4:4422.

- Shechter R, London A, Varol C, Raposo C, Cusimano M, Yovel G, et al. (2009). Infiltrating blood-derived macrophages are vital cells playing an anti-inflammatory role in recovery from spinal cord injury in mice. PLoS Med. 6(7):e1000113.
- Shi SX, Shi K, Liu Q. (2021). Brain injury instructs bone marrow cellular lineage destination to reduce neuroinflammation. Sci Transl Med. 13(589):eabc7029.
- 4. Sutherland TE, Dyer DP, Allen JE. (2023). The extracellular matrix and the immune system: A mutually dependent relationship. Science. 379(6633):eabp8964.
- Breuss M, Fritz T, Gstrein T, Chan K, Ushakova L, Yu N, et al. (2016). Mutations in the murine homologue of TUBB5 cause microcephaly by perturbing cell cycle progression and inducing p53-associated apoptosis. Development. 143(7):1126-1133.
- Soliman E, Gudenschwager Basso EK, Ju J, Willison A, Theus MH. (2025). Skull bone marrow-derived immune cells infiltrate the injured cerebral cortex and exhibit anti-inflammatory properties. Brain Behav Immun. 123:244-253.
- Ajabi Z, Keinath AT, Wei XX, Brandon MP. (2023). Population dynamics of head-direction neurons during drift and reorientation. Nature. 615(7954):892-899.
- 8. Nowotschin S, Hadjantonakis AK. (2009). Use of KikGR a photoconvertible green-to-red fluorescent protein for

cell labeling and lineage analysis in ES cells and mouse embryos. BMC Dev Biol. 9:49.

- Russo MV, Latour LL, McGavern DB. (2018). Distinct myeloid cell subsets promote meningeal remodeling and vascular repair after mild traumatic brain injury. Nat Immunol. 19(5):442-452.
- Salvador AFM, Kipnis J. (2022). Immune response after central nervous system injury. Semin Immunol. 59:101629.
- Mastorakos P, Mihelson N, Luby M, Burks SR, Johnson K, Hsia AW, et al. (2021). Temporally distinct myeloid cell responses mediate damage and repair after cerebrovascular injury. Nat Neurosci. 24(2):245-258.
- 12. Engelhardt B, Coisne C. (2011). Fluids and barriers of the CNS establish immune privilege by confining immune surveillance to a two-walled castle moat surrounding the CNS castle. Fluids Barriers CNS. 8(1):4.
- Mastorakos P, McGavern DB. (2019). The anatomy and immunology of vasculature in the central nervous system. Science Immunology. 4(37):eaav0492.
- Rua R, McGavern DB. (2018). Advances in Meningeal Immunity. Trends Mol Med. 24(6):542-559.
- 15. Nick JA, Dedrick RM, Gray AL, Vladar EK, Smith BE, Freeman KG, et al. (2022). Host and pathogen response to bacteriophage engineered against Mycobacterium abscessus lung infection. Cell. 185(11):1860-1874.e12.
- Bar-Or A, Li R. (2021). Cellular immunology of relapsing multiple sclerosis: interactions, checks, and balances. Lancet Neurol. 20(6):470-483.
- 17. Dong Y, Yong VW. (2019). When encephalitogenic T cells collaborate with microglia in multiple sclerosis. Nat Rev Neurol. 15(12):704-717.

- Guerrero BL, Sicotte NL. (2020). Microglia in Multiple Sclerosis: Friend or Foe? Front Immunol. 11:374.
- Hao J, Liu R, Piao W, Zhou Q, Vollmer TL, Campagnolo DI, et al. (2010). Central nervous system (CNS)-resident natural killer cells suppress Th17 responses and CNS autoimmune pathology. J Exp Med. 207(9):1907-1921.
- Lee GA, Lin TN, Chen CY, Mau SY, Huang WZ, Kao YC, et al. (2018). Interleukin 15 blockade protects the brain from cerebral ischemia-reperfusion injury. Brain Behav Immun. 73:562-570.
- 21. Li M, Li Z, Yao Y, Jin WN, Wood K, Liu Q, et al. (2017). Astrocyte-derived interleukin-15 exacerbates ischemic brain injury via propagation of cellular immunity. Proc Natl Acad Sci U S A. 114(3):E396-E405.
- 22. Fan L, Zhang CJ, Zhu L, Chen J, Zhang Z, Liu P, et al. (2020). FasL-PDPK1 Pathway Promotes the Cytotoxicity of CD8+ T Cells During Ischemic Stroke. Transl Stroke Res. 11(4):747-761.
- Hansson LS, Axelsson J, Petrovic P, Paues Göranson S, Olsson MJ, Lekander M, et al. (2021). Regulation of emotions during experimental endotoxemia: A pilot study. Brain Behav Immun. 93:420-424.
- 24. Fiala M, Lin J, Ringman J, Kermani-Arab V, Tsao G, Patel A, et al. (2005). Ineffective phagocytosis of amyloidbeta by macrophages of Alzheimer's disease patients. J Alzheimers Dis. 7(3):221-232.