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Anemia in Neurodegenerative Disorders: Implications for Cognitive Decline and Disease Progression

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ABSTRACT

Anemia, a hematological disorder characterized by reduced hemoglobin levels or erythrocyte count, has gained increasing attention as a potential risk factor for neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), and multiple sclerosis (MS). Emerging evidence suggests that the systemic consequences of anemia, such as impaired oxygen delivery, increased oxidative stress, and dysregulated iron metabolism, may play a crucial role in the pathophysiology of neurodegeneration. Hypoxia-induced neuronal stress, mitochondrial dysfunction, and neuroinflammation are among the key pathways through which anemia may contribute to synaptic dysfunction, neuronal loss, and progressive cognitive impairment. This review comprehensively examines the mechanistic links between anemia and neurodegeneration, highlighting molecular and cellular pathways involved in anemia-associated neuronal injury. We also evaluate clinical and epidemiological studies that establish correlations between anemia and the onset or progression of neurodegenerative diseases, addressing potential confounding factors and variations across populations. Furthermore, we discuss current and emerging therapeutic strategies aimed at mitigating anemia-induced neurodegenerative changes, including iron supplementation, erythropoiesis-stimulating agents, and novel pharmacological approaches targeting oxidative stress and inflammation. Understanding the complex interplay between anemia and neurodegeneration may open new avenues for early diagnosis, risk stratification, and therapeutic interventions in vulnerable populations. By bridging the gap between hematological and neurological research, this review aims to provide a comprehensive perspective on the potential role of anemia as a modifiable factor in neurodegenerative disease management.

Keywords: Anemia, Neurodegenerative Disorders, Cognitive Decline, Alzheimer's Disease, Parkinson's Disease, Oxidative Stress, Neuroinflammation, Iron Deficiency

INTRODUCTION

Neurodegenerative disorders are characterized by progressive neuronal dysfunction and loss, leading to cognitive decline, motor impairments, and a significant reduction in quality of life[1]. These disorders, which include Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), and Huntington's disease (HD), are driven by complex mechanisms such as oxidative stress, neuroinflammation, mitochondrial dysfunction, and protein aggregation[2]. While genetic and environmental factors play critical roles in their onset and progression, emerging evidence suggests that systemic conditions, including metabolic and hematological abnormalities, may also influence neurodegeneration[3]. Among these systemic factors, anemia has garnered increasing attention as a potential contributor to neurodegenerative disease pathophysiology. Anemia, defined as a reduction in red blood cell count or hemoglobin levels, leads to impaired oxygen delivery to tissues, including the brain[4, 5]. Chronic cerebral hypoxia resulting from anemia may exacerbate neuronal damage, promote neuroinflammation, and accelerate cognitive and motor decline[6]. The elderly population, which is at the highest risk for neurodegenerative disorders, also has a high prevalence of anemia, further highlighting the need to explore the potential link between these conditions [7]. Understanding the interplay between anemia and neurodegenerative disorders is crucial for developing effective preventive and therapeutic strategies. Investigating whether anemia serves as a modifiable risk factor for neurodegeneration could open new avenues for early intervention, potentially delaying disease onset or progression. This review aims to explore the underlying mechanisms connecting anemia to neurodegeneration, evaluate epidemiological evidence, and discuss potential clinical implications for patient management.

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Pathophysiological Mechanisms Linking Anemia to Neurodegeneration

Reduced Oxygen Delivery

Hemoglobin plays a crucial role in oxygen transport, ensuring that tissues, including the brain, receive the oxygen required for optimal cellular function[8]. In cases of anemia, particularly those involving a significant reduction in hemoglobin levels, oxygen delivery to the brain is compromised. This results in cerebral hypoxia, which impairs mitochondrial function and increases susceptibility to neurodegeneration. Mitochondria are essential for cellular respiration, and their dysfunction leads to decreased adenosine triphosphate (ATP) production, increased production of reactive oxygen species (ROS), and eventual neuronal death[9, 10].

Chronic hypoxia has been linked to various neurodegenerative diseases. For example, in Alzheimer's disease (AD), prolonged oxygen deprivation has been implicated in the accumulation of amyloid-beta (Aβ) plaques[11]. Hypoxia-induced upregulation of beta-site amyloid precursor protein cleaving enzyme 1 (BACE1) promotes the pathological processing of amyloid precursor protein (APP), resulting in increased Aβ production and deposition. Similarly, in Parkinson's disease (PD), reduced oxygen availability contributes to the degeneration of dopaminergic neurons in the substantia nigra[12]. These neurons are highly metabolically active and rely on adequate oxygen supply to maintain dopamine synthesis and proper neurotransmission[12]. Additionally, hypoxia can alter synaptic plasticity, reducing cognitive function and memory retention. The hippocampus, a brain region critical for learning and memory, is particularly vulnerable to oxygen deprivation. Studies have shown that chronic hypoxia induces synaptic dysfunction, reduces dendritic spine density, and impairs long-term potentiation (LTP), a cellular mechanism underlying memory formation[13]. These findings suggest that anemia-associated cerebral hypoxia can contribute to the cognitive decline observed in neurodegenerative disorders.

Iron Dysregulation and Oxidative Stress

Iron homeostasis is essential for normal brain function, playing a key role in neurotransmitter synthesis, myelin formation, and mitochondrial function[14]. However, disturbances in iron metabolism, such as those seen in iron deficiency anemia (IDA), can have profound effects on neural health. IDA results in inadequate iron availability for critical biochemical processes, leading to impaired dopamine synthesis, reduced myelination, and alterations in neuronal energy metabolism[14]. These deficits can contribute to cognitive dysfunction, mood disorders, and increased risk of neurodegenerative diseases. Conversely, excessive iron accumulation in the brain is also detrimental. Iron is a potent catalyst for the Fenton reaction, which generates highly reactive hydroxyl radicals[15, 16]. These radicals contribute to oxidative stress, lipid peroxidation, and DNA damage, all of which are implicated in neurodegeneration. Studies have shown that iron overload is a common feature in AD, PD, and amyotrophic lateral sclerosis (ALS), with increased iron deposits observed in affected brain regions. For example, in PD, elevated iron levels in the substantia nigra contribute to dopaminergic neuronal loss through oxidative stress and ferroptosis, a form of iron-dependent cell death. In AD, iron accumulation is associated with the aggregation of Aβ and hyperphosphorylated tau protein, exacerbating neuroinflammation and neuronal toxicity[15]. Iron-induced oxidative stress also disrupts mitochondrial function, further promoting neuronal degeneration. Given these findings, maintaining iron homeostasis is crucial for preventing neurodegenerative processes. Strategies such as dietary modifications, iron supplementation in IDA, and iron chelation therapy in cases of iron overload have been explored as potential therapeutic approaches.

Neuroinflammation and Immune Activation

Chronic inflammation is a key driver of neurodegenerative diseases, and anemia has been shown to exacerbate neuroinflammatory processes[17]. Anemia, particularly in its chronic form, induces the production of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β). These cytokines can cross the blood-brain barrier (BBB) and activate microglia, the resident immune cells of the brain. Persistent microglial activation leads to excessive production of ROS and nitric oxide (NO), contributing to neuronal damage and synaptic dysfunction[18]. In AD, neuroinflammation plays a central role in disease progression. Microglia become chronically activated in response to Aβ plaques, releasing inflammatory mediators that further exacerbate neuronal toxicity[19]. Anemia-induced cytokine elevation may accelerate this process, increasing the burden of neuroinflammation in AD patients. Similarly, in PD, microglial activation in the substantia nigra leads to the release of neurotoxic factors that contribute to dopaminergic neuronal loss[19]. Multiple sclerosis (MS) is another neurodegenerative disorder strongly influenced by immune dysregulation. Anemia-related inflammation may exacerbate MS pathology by promoting T-cell activation and increasing BBB permeability, allowing autoreactive immune cells to infiltrate the central nervous system (CNS). This immune-mediated attack on myelin sheaths results in demyelination, axonal loss, and progressive neurological impairment. Furthermore, systemic inflammation associated with anemia can impair neurovascular function, reducing cerebral blood flow and increasing the risk of vascular cognitive impairment[20]. Chronic inflammatory states have also been linked to depression and cognitive decline, highlighting the broader impact of anemia-induced inflammation on mental health.

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Impaired Erythropoiesis and Neurotrophic Support

Erythropoiesis, the process of red blood cell production, is regulated by erythropoietin (EPO), a glycoprotein hormone primarily produced by the kidneys in response to hypoxia[21]. Beyond its hematopoietic role, EPO exerts neuroprotective effects, promoting neuronal survival, neurogenesis, and synaptic plasticity. EPO receptors are widely expressed in the brain, and EPO signaling has been shown to enhance neuronal resilience against oxidative stress, excitotoxicity, and apoptosis[21]. In anemia, impaired erythropoiesis and dysregulated EPO signaling may contribute to neurodegenerative processes. Reduced EPO levels lead to diminished neuroprotection, increasing neuronal vulnerability to stressors. Experimental studies have demonstrated that EPO administration can mitigate neurodegeneration in animal models of AD, PD, and stroke by reducing inflammation, enhancing synaptic plasticity, and protecting against oxidative damage[22]. Moreover, EPO plays a crucial role in cognitive function and mood regulation. Clinical studies have suggested that EPO therapy may improve cognitive performance in patients with neurodegenerative disorders and anemia-associated cognitive decline. The neurotrophic effects of EPO also extend to psychiatric conditions, with evidence supporting its potential use in treating depression and schizophrenia[22]. However, the therapeutic use of EPO in neurodegeneration is complex. While exogenous EPO administration has shown promise in preclinical studies, its erythropoietic effects can lead to increased blood viscosity and thrombotic risks[23]. Therefore, novel strategies such as non-erythropoietic EPO derivatives and targeted delivery systems are being explored to harness EPO's neuroprotective benefits without hematological complications[23]. Anemia exerts profound effects on brain function and contributes to the pathogenesis of neurodegenerative diseases through multiple mechanisms, including reduced oxygen delivery, iron dysregulation, neuroinflammation, and impaired neurotrophic support. Given the growing evidence linking anemia to cognitive impairment and neurodegeneration, early diagnosis and effective management of anemia may represent a valuable strategy for mitigating neurodegenerative risk[24]. Future research should explore targeted interventions that address both the hematological and neurological consequences of anemia, potentially improving outcomes for individuals at risk of neurodegenerative diseases.

Clinical and Epidemiological Evidence

Alzheimer's Disease: In Alzheimer's Disease (AD), a growing body of research indicates that anemia is a common comorbidity that may exacerbate the disease's progression. Studies have shown that anemia in AD patients is linked to an acceleration in cognitive decline, as evidenced by the work of Fehsel[25], who demonstrated a significant correlation between low hemoglobin levels and faster cognitive deterioration. This relationship highlights how anemia might worsen the pathophysiological processes of AD. Additionally, the reduction in hemoglobin levels in AD patients has been associated with an increase in amyloid plaque burden, which is a key hallmark of AD, and greater hippocampal atrophy, a characteristic feature of the disease's neurodegenerative process[25, 26]. The decreased oxygen-carrying capacity due to anemia could worsen the oxygen-deprived environment in the brain, potentially contributing to further neuronal damage and cognitive impairment. Therefore, anemia in AD patients may not only reflect the severity of the disease but also act as a contributor to its progression.

Parkinson's Disease: Parkinson's Disease (PD) is another neurodegenerative disorder frequently associated with anemia, especially iron deficiency anemia (IDA). PD patients often exhibit a significant reduction in hemoglobin levels, which may worsen the neurological manifestations of the disease[27]. Iron dysregulation is a critical feature in PD pathophysiology, with excessive iron accumulation occurring in key regions of the brain, particularly in the substantia nigra[27]. This iron buildup contributes to oxidative stress, neuronal damage, and the subsequent loss of dopaminergic neurons that characterize PD. The bidirectional relationship between anemia and neurodegeneration in PD has been suggested by Levi et al. [28], who argue that iron deficiency anemia might exacerbate the oxidative damage from iron overload, thereby accelerating neurodegeneration. Moreover, anemia could be a direct consequence of the neurodegenerative process in PD, with the brain's reduced capacity to regulate iron potentially influencing hematologic function[28]. Addressing anemia in PD patients could thus serve as a potential therapeutic approach to mitigate some of the oxidative stress and neuronal damage, although further research is needed to fully understand this complex interplay.

Multiple Sclerosis: Multiple Sclerosis (MS) is another chronic neurological condition where anemia is prevalent, contributing significantly to the disease's symptoms, including fatigue and cognitive dysfunction[29]. Anemia in MS patients, especially iron deficiency anemia, has been observed to exacerbate these symptoms, severely impacting the quality of life. The mechanisms behind anemia in MS remain complex, but it is believed to involve a combination of factors, including chronic inflammation, reduced erythropoiesis, and impaired iron metabolism[30]. Notably, anemia in MS may lead to a worsened clinical outcome, as lower hemoglobin levels decrease oxygen delivery to tissues, including the brain, potentially affecting the function of neurons. One promising therapeutic approach is the use of erythropoietin (EPO), a hormone involved in red blood cell production, which has been explored for its potential neuroprotective effects in MS models. Cappellini et al. [30] investigated the neuroprotective benefits of erythropoietin therapy in MS, suggesting that its ability to stimulate red blood cell production and modulate inflammation could provide a dual benefit in alleviating anemia while also offering direct protection to the central nervous system. However, while initial results are

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promising, more clinical studies are needed to assess the safety and efficacy of such therapies in MS patients with anemia.

Therapeutic Implications

Iron Supplementation and Management

Iron supplementation in patients with anemia, especially those with neurodegenerative disorders, is critical for addressing iron deficiency anemia (IDA) while preventing iron overload, which can have detrimental effects on the brain[31]. While iron is an essential element for cognitive function, excessive iron accumulation in the brain is associated with neurodegeneration, particularly in areas such as the basal ganglia. Therefore, careful management of iron levels is paramount. Supplementation should be tailored to individual needs, considering factors like baseline serum ferritin levels and the patient's ability to metabolize and store iron[31]. Iron supplementation, when appropriately administered, can help improve cognitive function by addressing IDA-related cognitive impairments such as memory loss, attention deficits, and fatigue. According to Georgieff (2020), the goal of supplementation is not only to correct anemia but also to optimize brain iron homeostasis to prevent exacerbating neurodegenerative processes.

Erythropoietin Therapy

Erythropoietin (EPO), a glycoprotein hormone primarily involved in red blood cell production, has gained attention for its potential neuroprotective properties beyond its role in erythropoiesis[32]. Recombinant EPO has shown promise in several neurodegenerative disorders due to its ability to reduce neuroinflammation, enhance neuronal survival, and improve cognitive function. Research by Brines and Cantarelli et al [32] highlights the neuroprotective effects of EPO, including its ability to attenuate oxidative stress and apoptosis in neurons. Furthermore, EPO administration has been associated with enhanced synaptic plasticity and neurogenesis, which are crucial for cognitive improvement. In conditions like Alzheimer's disease and Parkinson's disease, where neuroinflammation is a key pathological feature, EPO may help mitigate the inflammatory response and slow disease progression. Thus, recombinant EPO represents a promising therapeutic strategy for patients with both anemia and neurodegenerative disorders.

Anti-inflammatory and Antioxidant Strategies

Anemia-induced inflammation and oxidative stress contribute significantly to the progression of neurodegenerative diseases. These conditions can exacerbate neuronal damage, impair cognitive function, and accelerate disease progression[33]. Nutritional interventions, such as vitamin B12 and folate supplementation, are particularly beneficial in this context [34]. Vitamin B12 plays a crucial role in myelin synthesis and nerve function, while folate is vital for DNA synthesis and repair[35]. Deficiencies in these nutrients are linked to cognitive decline and may aggravate anemia-related cognitive impairment. As noted by Halczuk et al., these vitamins help reduce homocysteine levels, which are known to be neurotoxic and contribute to both anemia and neurodegeneration.[35] Additionally, antioxidants, such as vitamin E and C, may help mitigate the oxidative stress that damages neurons. By reducing the oxidative load and promoting a balanced inflammatory response, these strategies may reduce neurodegeneration and improve cognitive outcomes in patients with neurodegenerative disorders. Thus, combining anti-inflammatory and antioxidant therapies with iron supplementation and EPO therapy could provide a multifaceted approach to managing anemia and its cognitive implications in neurodegenerative diseases.

CONCLUSION

Anemia and neurodegenerative disorders share common pathophysiological pathways, including oxidative stress, neuroinflammation, and impaired oxygen metabolism. Addressing anemia in neurodegenerative patients could provide a novel therapeutic avenue to slow disease progression and cognitive decline. Future research should focus on clinical trials to evaluate anemia-targeted interventions in neurodegenerative disorders.

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