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Iron Overload in HIV: Implications for Antiretroviral Therapy

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Abstract

Iron overload is increasingly recognized as a significant comorbidity in individuals living with HIV, with potential implications for disease progression and management. This review explores the implications of iron overload for antiretroviral therapy (ART) in individuals with HIV, focusing on its effects on drug metabolism, treatment efficacy, and toxicity. Mechanisms underlying iron overload in HIV, including chronic inflammation, altered iron metabolism, and comorbidities, are discussed, highlighting the role of hepcidin in mediating the interplay between iron metabolism and HIV infection. Understanding these complex interactions is essential for optimizing treatment outcomes and improving the quality of life for individuals living with HIV and iron overload. Further research is needed to elucidate the mechanisms underlying iron dysregulation in HIV and develop targeted therapeutic interventions to address this important clinical issue.

Keywords*: Iron overload, HIV, Antiretroviral therapy, Hepcidin, Immune activation, Oxidative stress*

Introduction

Iron overload has emerged as a significant concern in the management of individuals living with HIV, presenting a complex interplay of factors that influence disease progression and treatment outcomes. While HIV infection itself is associated with chronic inflammation, immune activation, and dysregulation of iron metabolism, the coexistence of iron overload exacerbates this complexity. The introduction of antiretroviral therapy (ART) revolutionized the management of HIV, yet the implications of iron overload on ART efficacy and toxicity remain a topic of ongoing research and clinical interest. The epidemiology of iron overload in HIV reflects its multifactorial etiology, with contributions from chronic inflammation, altered iron metabolism, and

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comorbidities such as viral hepatitis and non-alcoholic fatty liver disease. Individuals living with HIV are particularly vulnerable to iron overload due to the interplay between HIV-induced inflammation and dysregulation of hepcidin, a key regulator of iron homeostasis. Consequently, understanding the mechanisms underlying iron dysregulation in HIV is crucial for optimizing treatment strategies and improving patient outcomes.[1-10]

Mechanistically, chronic inflammation and immune activation in HIV contribute to dysregulated iron metabolism through various pathways, including cytokine-mediated suppression of hepcidin and increased iron uptake by macrophages. This dysregulation leads to excessive iron accumulation in tissues and organs, exacerbating oxidative stress and tissue damage. Furthermore, comorbidities such as viral hepatitis and liver fibrosis further contribute to iron overload by impairing hepatic iron clearance and promoting iron deposition within hepatocytes. The implications of iron overload for ART in individuals with HIV are multifaceted, affecting drug metabolism, treatment efficacy, and toxicity. Iron is known to interact with drug absorption, distribution, metabolism, and excretion processes, potentially altering the pharmacokinetics of ART agents. Consequently, optimizing ART regimens in the context of iron overload requires careful consideration of drug interactions and potential toxicities, particularly in individuals with underlying liver disease or other comorbidities. Moreover, the role of hepcidin, a central regulator of iron homeostasis, in mediating the interplay between iron metabolism and HIV infection is of particular interest. Dysregulation of hepcidin expression in response to HIV-induced inflammation may contribute to iron sequestration within macrophages and alter iron distribution to tissues and organs. Targeted interventions aimed at modulating hepcidin activity represent a potential therapeutic strategy for mitigating the adverse effects of iron overload on HIV treatment outcomes.[11-20]

Mechanisms of Iron Overload in HIV

Iron overload in individuals living with HIV is the result of a multifaceted interplay between chronic inflammation, dysregulated iron metabolism, and comorbidities associated with HIV infection. Chronic inflammation and immune activation, hallmark features of HIV infection, play a central role in disrupting the delicate balance of iron homeostasis. Pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) stimulate the production of hepcidin, a key regulator of iron metabolism, by hepatocytes. Hepcidin acts by binding to ferroportin, the sole known iron exporter in vertebrates, inducing its internalization and degradation, thereby inhibiting iron release from macrophages and enterocytes into the circulation. However, HIV-induced inflammation may also lead to dysregulated hepcidin expression, resulting in hepcidin deficiency and uncontrolled iron absorption from the gastrointestinal tract. The dysregulation of hepcidin in HIV is complex and multifactorial, involving interactions with viral proteins, immune cells, and pro-inflammatory cytokines. For example, HIV proteins such as Tat and gp120 have been shown to directly stimulate hepcidin expression, while immune activation and pro-inflammatory cytokines promote hepcidin suppression. Consequently, this dysregulation leads to increased intestinal iron absorption and sequestration of iron within macrophages, contributing to systemic iron overload.[21-40]

Comorbidities associated with HIV infection, such as viral hepatitis, liver fibrosis, and non-alcoholic fatty liver disease (NAFLD), further exacerbate iron overload by impairing hepatic iron clearance and promoting iron deposition within hepatocytes. Hepatocytes play a crucial role in regulating systemic iron homeostasis by storing excess iron in the form of ferritin and releasing it into the circulation when needed. However, in the context of liver damage and fibrosis, this regulatory function is compromised, leading to impaired iron storage and clearance and further exacerbating systemic iron overload. Moreover, alterations in iron metabolism observed in individuals with HIV may also be attributed to disruptions in the hepcidin-ferroportin axis, resulting in impaired iron recycling and utilization. Macrophages, which play a central role in iron recycling through phagocytosis of senescent erythrocytes and release of iron into the circulation via ferroportin, may exhibit dysregulated iron handling in the setting of HIV-induced inflammation and immune activation. Dysfunctional macrophages may fail to appropriately regulate iron release, leading to increased iron retention within macrophages and further exacerbating systemic iron overload.[41-60]

Implications for Antiretroviral Therapy

Iron overload in individuals living with HIV presents several implications for antiretroviral therapy (ART), including effects on drug metabolism, treatment efficacy, and toxicity. The complex interplay between iron metabolism and ART pharmacokinetics and pharmacodynamics necessitates careful consideration in the management of individuals with HIV and iron overload. Iron is known to interact with drug absorption, distribution, metabolism, and excretion processes, potentially altering the pharmacokinetics of ART agents. Several antiretroviral drugs, including protease inhibitors and non-nucleoside reverse transcriptase inhibitors, are substrates for cytochrome P450 enzymes and drug transporters that may be influenced by iron status. Iron overload may therefore affect the metabolism of ART drugs, leading to altered plasma concentrations and potential changes in treatment efficacy. The impact of iron overload on ART efficacy remains a topic of ongoing research and debate. Iron has been shown to modulate immune function and viral replication in vitro, raising concerns that iron overload may exacerbate HIV pathogenesis and compromise treatment outcomes. Additionally, iron-mediated oxidative stress and mitochondrial dysfunction may contribute to immune dysregulation and viral persistence, potentially undermining the effectiveness of ART in suppressing viral replication and restoring immune function.[61-90]

Iron overload may also influence the toxicity profile of ART drugs, particularly in individuals with underlying liver disease or other comorbidities. Some antiretroviral agents, such as nucleoside reverse transcriptase inhibitors (NRTIs), are associated with mitochondrial toxicity and hepatotoxicity, which may be exacerbated by iron-mediated oxidative stress and hepatic injury. Furthermore, iron overload may increase the risk of drug-induced hepatotoxicity and other adverse effects, necessitating close monitoring and dose adjustments in individuals receiving ART. Iron chelators, used to treat iron overload, may interact with ART drugs, potentially affecting their absorption, distribution, metabolism, and excretion. Similarly, ART drugs may influence the pharmacokinetics of iron chelators, leading to altered drug concentrations and potential adverse effects. Careful monitoring and dose adjustments may be necessary when co-administering iron

chelators and ART drugs to minimize the risk of drug interactions and optimize treatment outcomes. Targeting iron metabolism pathways represents a potential therapeutic strategy for improving ART outcomes in individuals with HIV and iron overload. Modulating hepcidin activity, for example, may help mitigate the adverse effects of iron overload on HIV pathogenesis and treatment efficacy. Additionally, further research is needed to identify novel therapeutic targets for managing iron overload in individuals living with HIV, with the potential to enhance treatment outcomes and improve patient quality of life.[91-107]

Conclusion

Iron overload in individuals living with HIV represents a multifaceted challenge with implications for disease management and treatment outcomes. The complex interplay between chronic inflammation, dysregulated iron metabolism, and comorbidities associated with HIV infection underscores the importance of understanding the implications of iron overload for antiretroviral therapy (ART). The mechanisms underlying iron overload in HIV, including chronic inflammation, dysregulated hepcidin expression, and comorbidities such as viral hepatitis and liver fibrosis, contribute to systemic iron accumulation and oxidative stress, which may impact ART efficacy and toxicity. Iron-mediated alterations in drug metabolism, treatment efficacy, and toxicity underscore the need for careful consideration in the management of individuals with HIV and iron overload.

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