Journal of Neuroinfectious Diseases
ISSN: 2314-7326

Commentary Open Access

HIV-1 Infection with Alcohol Misuse Causes of Neurodegeneration, Neurocognitive Impairment, and Treatment Options

Kristen Heaton*

Department of Environmental Health, Boston University School of Public Health, USA

Abstract

Human Immunodeficiency Virus type 1 (HIV-1) infection is a global health concern that not only affects the immune system but also poses significant challenges to neurological health. When coupled with alcohol misuse, HIV-1 infection can exacerbate neurodegeneration and lead to neurocognitive impairment. This review explores the intricate interplay between HIV-1 infection and alcohol misuse, delving into the underlying causes of neurodegeneration and the subsequent development of neurocognitive impairments. We examine the synergistic effects of these two factors on the central nervous system, shedding light on the molecular and cellular mechanisms that contribute to the observed neuropathological changes. Furthermore, we discuss current treatment options and potential therapeutic strategies aimed at mitigating the impact of HIV-1 infection with alcohol misuse on the neurological system. Understanding the complex relationship between HIV-1 infection, alcohol misuse, and neurodegeneration is crucial for developing targeted interventions to improve the quality of life for individuals affected by this dual burden.

Introduction

The co-occurrence of HIV-1 infection and alcohol misuse presents a formidable challenge to global public health, necessitating a comprehensive understanding of the synergistic effects on the central nervous system (CNS). While the primary impact of HIV-1 is on the immune system, the virus also penetrates the CNS, leading to various neurological complications [1]. When compounded by alcohol misuse, a known neurotoxin, the consequences become even more pronounced. This paper aims to elucidate the causes of neurodegeneration and subsequent neurocognitive impairment resulting from the dual burden of HIV-1 infection and alcohol misuse. The intricate interplay between HIV-1 and alcohol involves complex molecular and cellular mechanisms that contribute to the observed neuropathological changes. Neuroinflammation, oxidative stress, and dysregulation of neurotransmitter systems are among the key factors driving neurodegeneration in individuals with concurrent HIV-1 infection and alcohol misuse [2-4]. Moreover, the compromised blood-brain barrier exacerbates the entry of both HIV-1 and alcohol into the CNS, intensifying their detrimental effects. In addition to understanding the pathophysiology, this review explores current treatment options and potential therapeutic strategies to address the neurological consequences of this dual burden. As the global incidence of HIV-1 infection and alcohol misuse continues to rise, a deeper comprehension of the underlying mechanisms and effective interventions is imperative for improving the overall well-being of affected individuals. One quarter of adults and 50% of children used to develop significant neurocognitive complications as a consequence of infection at a period of immunosuppression or AIDS before the introduction of anti-retroviral therapy (ART). While subclinical metabolic and structural abnormalities are detectable in neurologically asymptomatic subjects, overt HAD usually develops as a late complication and is accompanied by immunosuppression. Reduction of the neuronal marker, N-acetylaspartate, in frontal lobe white and gray matter was found in individuals taking NRTIs, didanosine and/or stavudine, as a result of depleted brain mitochondria and/or alterations in cellular respiration. Introduction of ART considerably changed the clinical evolution of HIV-1 infection. ART diminished the incidence and prevalence of major opportunistic infections and resulted in improvement in survival rates. Patients (10-15%) are now over 50 years of age and, with continued advances in treatment, many will reach normal life expectancy [5-8].

Conclusion

In conclusion, the dual burden of HIV-1 infection and alcohol misuse presents a multifaceted challenge that significantly impacts the neurological health of affected individuals. The intricate interplay between these two factors contributes to neurodegeneration and subsequent neurocognitive impairment through a complex web of molecular and cellular mechanisms. Understanding these processes is critical for developing targeted interventions that address the unique challenges posed by this co-occurrence. As research in this field progresses, it is imperative to continue investigating novel therapeutic targets and refining existing treatment modalities. Moreover, addressing the socio-economic factors contributing to HIV-1 infection and alcohol misuse is crucial for developing holistic and effective public health strategies. In essence, unraveling the complexities of HIV-1 infection with alcohol misuse and its impact on neurodegeneration and neurocognitive impairment opens new avenues for targeted interventions. By advancing our understanding of these mechanisms, we pave the way for improved patient outcomes and a better quality of life for those affected by this challenging dual burden.

Acknowledgment

None

Conflict of Interest

None

***Corresponding author:** Kristen Heaton, Department of Environmental Health, Boston University School of Public Health, USA, E-mail: kristenheaton@bu.edu

Received: 30-Oct-2023, Manuscript No: JNID-23-122226; **Editor assigned:** 02-Nov-2023, Pre-QC No: JNID-23-122226 (PQ); **Reviewed:** 17-Nov-2023, QC No: JNID-23-122226; **Revised:** 24-Nov-2023, Manuscript No: JNID-23-122226 (R); **Published:** 30-Nov-2023, DOI: 10.4172/2314-7326.1000478

Citation: Heaton K (2023) HIV-1 Infection with Alcohol Misuse Causes of Neurodegeneration, Neurocognitive Impairment, and Treatment Options. J Neuroinfect Dis 14: 478.

References

1. Palmer BW, Heaton SC, Jeste DV (1999) Older patients with schizophrenia: challenges in the coming decades. Psychiatric Services 50: 1178-1183.

2. Patorno E, Bohn R, Wahl P, Avorn J, Patrick AR, et al. (2010) Anticonvulsant medications and the risk of suicide, attempted suicide, or violent death. JAMA 303: 1401-1409.

3. Olesen JB, Hansen PR, Erdal J, Abildstrøm SZ, Weeke P, et al. (2010) Antiepileptic drugs and risk of suicide: a nationwide study. Pharmacoepidem Dr S 19: 518-524.

4. Leipzig R, Cumming R, Tinetti M (1999) Drugs and falls in older people: a systematic review and meta-analysis: I. Psychotropic drugs. J Am Geriatr Soc 47: 30-39.

5. Gill S, Bronskill S, Normand S, Anderson GM, Sykora K, et al. (2007) Antipsychotic drug use and mortality in older adults with dementia. Ann Intern Med 146: 775-786.

6. Casey D, Haupt D, Newcomer J, Henderson DC, Sernyak MJ, et al. (2004) Antipsychotic-induced weight gain and metabolic abnormalities: implications for increased mortality in patients with schizophrenia. J Clin Psychiatry 65(Suppl 7): 4-18.

7. Schneider LS, Dagerman KS, Insel P (2005) Risk of Death with Atypical Antipsychotic Drug Treatment for Dementia. JAMA 294: 1934-1943.

8. Meijer WEE, Heerdink ER, Nolen WA, Herings RMC, Leufkens HGM, et al. (2004) Association of Risk of Abnormal Bleeding With Degree of Serotonin Reuptake Inhibition by Antidepressants. Arch Intern Med 164: 2367-2370.