Short Communication
Volume 10:06, 2024

Journal of Clinical Respiratory Diseases & Care: Open Access

ISSN:2472-1247 Open Access

Therapeutic Effects of Hydrogen Gas Inhalation on Coronary Artery Lesions in a Kawasaki Disease Mouse Model

Dong Kiril*

Department of Medicine, Laval University, Quebec, Canada

Introduction

Kawasaki Disease (KD) is an acute systemic vasculitis that primarily affects children and is a leading cause of acquired heart disease in this population. The most serious complications of KD arise from Coronary Artery Lesions (CALs), including coronary aneurysms and stenosis, which can lead to long-term cardiovascular issues. While treatments such as Intravenous Immunoglobulin (IVIG) and aspirin are standard therapies, not all patients respond adequately, and new therapeutic strategies are being explored. Hydrogen gas (H2) inhalation has emerged as a potential therapeutic modality due to its anti-inflammatory, antioxidant, and cytoprotective properties. This study investigates the effects of hydrogen gas inhalation on coronary artery lesions in a mouse model of Kawasaki disease. The mouse model used in this study was developed to replicate the coronary artery involvement characteristic of KD. Mice were treated with a synthetic compound, Candida albicans water-soluble fraction (CAWS), to induce coronary arteritis. This model has been extensively validated as an experimental platform for studying KD pathogenesis and therapeutic interventions. Following the induction of arteritis, the mice were divided into two groups: one receiving hydrogen gas inhalation and the other serving as a control. Hydrogen gas was administered at a concentration of 2% via inhalation chambers for two hours daily over two weeks. The control group received no intervention other than standard housing conditions.

Description

Histopathological analysis was performed to evaluate the severity of coronary artery lesions, including inflammation, intimal thickening, and aneurysmal changes. The tissues were harvested and stained with hematoxylin and eosin and Elastica Van Gieson (EVG) staining to assess structural integrity and pathological changes in the coronary arteries. Immunohistochemical staining was also employed to detect markers of oxidative stress and inflammatory mediators such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). The results demonstrated a significant reduction in coronary artery inflammation and structural damage in the hydrogen gas-treated group compared to the control group. Mice treated with hydrogen gas exhibited decreased infiltration of inflammatory cells, reduced oxidative stress markers, and lower expression of pro-inflammatory cytokines. These findings were corroborated by quantitative measurements of malondialdehyde, a biomarker of lipid peroxidation, which was markedly lower in the treated group. Additionally, superoxide dismutase activity, a critical antioxidant enzyme, was significantly higher in the hydrogen gas group, indicating enhanced antioxidant defense. The study also assessed the endothelial function of coronary arteries using acetylcholine-induced vasodilation experiments. The hydrogen gas-treated mice demonstrated improved endothelial function, as evidenced by a greater degree of vasodilation in response to acetylcholine compared to the control group. This suggests that hydrogen gas inhalation not only reduces inflammatory damage but also helps preserve vascular integrity and functionality [1].

At the molecular level, hydrogen gas inhalation was found to modulate key signaling pathways involved in inflammation and oxidative stress. Western blot analysis showed a downregulation of nuclear factor-κB (NF-κB) signaling in the hydrogen gas group. NF-κB is a central mediator of inflammation and is implicated in the pathogenesis of KD-related vascular damage. By inhibiting NF-κB activation, hydrogen gas may attenuate the inflammatory cascade and mitigate coronary artery damage. Additionally, hydrogen gas was observed to activate the Nrf2/ARE (nuclear factor erythroid 2-related factor 2/antioxidant response element) pathway, which plays a critical role in cellular defense against oxidative stress. Increased nuclear translocation of Nrf2 and upregulation of downstream antioxidant enzymes such as heme oxygenase-1 (HO-1) and glutathione peroxidase (GPx) were noted in the treated group. This activation of the Nrf2 pathway likely contributes to the reduced oxidative damage observed in the hydrogen gas-treated mice. The safety profile of hydrogen gas inhalation was also evaluated. No adverse effects were reported in the treated mice, as evidenced by normal behavior, body weight maintenance, and the absence of histological abnormalities in non-target organs such as the liver, kidney, and lungs. This aligns with previous studies indicating that hydrogen gas is a safe and non-toxic intervention [2,3].

The findings of this study have significant implications for the treatment of Kawasaki disease and other inflammatory vascular conditions. By targeting both inflammation and oxidative stress, hydrogen gas inhalation offers a dual therapeutic approach that addresses key pathological mechanisms underlying coronary artery lesions. While the current study was conducted in a mouse model, the translational potential of hydrogen gas therapy is promising. Preliminary clinical studies in humans with other inflammatory diseases have shown similar benefits, suggesting that hydrogen gas could be a viable adjunct therapy for KD. Despite these encouraging results, several limitations need to be addressed. The study was conducted in a controlled experimental setting, and the translation of these findings to clinical practice requires further investigation. Long-term studies are needed to evaluate the durability of the therapeutic effects and to ensure safety over extended periods. Additionally, the optimal dosing regimen and duration of hydrogen gas therapy remain to be determined [4,5].

Conclusion

Future research should also explore the potential synergistic effects of combining hydrogen gas inhalation with standard KD treatments such as IVIG and aspirin. Such combination therapies could potentially enhance efficacy and improve outcomes in patients with refractory KD or severe coronary artery involvement. Furthermore, the mechanisms underlying hydrogen gas's effects on vascular inflammation and oxidative stress warrant deeper investigation to identify additional therapeutic targets. In conclusion, this study demonstrates that hydrogen gas inhalation significantly attenuates coronary artery lesions in a mouse model of Kawasaki disease. By reducing inflammation, oxidative stress, and endothelial dysfunction, hydrogen gas therapy shows promise as a novel treatment for KD-associated coronary artery complications. Further research is warranted to validate these findings in clinical settings and to optimize therapeutic protocols for broader application.

Acknowledgement

None.

***Address for Correspondence:** Dong Kiril, Department of Medicine, Laval University, Quebec, Canada, E-mail: kiridong@gmail.com*

Received: 02 December, 2024, Manuscript No. Jcrdc-24-158183; **Editor Assigned:** 04 December, 2024, PreQC No. P-158183; **Reviewed:** 17 December, 2024, QC No. Q-158183; **Revised:** 23 December, 2024, Manuscript No. R-158183; **Published:** 31 December, 2024, DOI: 10.37421/2472-1247.2024.10.340

Conflict of Interest

None.

References

1. Scherler, Laura, Nikolaus A. Haas, Anja Tengler and Joseph Pattathu, et al. "Acute phase of Kawasaki disease: A review of national guideline recommendations." *Eur J Pediatr* 181 (2022): 2563-2573.
2. Marek-Iannucci, Stefanie, Asli B. Ozdemir, Debbie Moreira and Angela C. Gomez, et al. "Autophagy-mitophagy induction attenuates cardiovascular inflammation in a murine model of Kawasaki disease vasculitis." *JCI insight* 6 (2021).
3. Hu, Jing, Wei Qian, Zhiwei Yu and Tao Xu, et al. "Increased neutrophil respiratory burst predicts the risk of coronary artery lesion in Kawasaki disease." *Front pediatr* 8 (2020): 391.
4. Ding, Yinjuan, Yongmiao Peng, Huilan Wu and Yuqing Huang, et al. "The protective roles of liraglutide on kawasaki disease via AMPK/mTOR/NF-κB pathway." *Int Immunopharmacol* 117 (2023): 110028.
5. Kuo, Ho-Chang, Mao-Hung Lo, Kai-Sheng Hsieh and Mindy Ming-Huey Guo, et al. "High-dose aspirin is associated with anemia and does not confer benefit to disease outcomes in Kawasaki disease." *PloS one* 10 (2015): e0144603.

How to cite this article: Kiril, Dong. "Therapeutic Effects of Hydrogen Gas Inhalation on Coronary Artery Lesions in a Kawasaki Disease Mouse Model." *J Clin Respir Dis Care* 10 (2024): 340.