CHRONIC EXPOSURE TO CARBON MONOXIDE (CO) AND THE MORPHOLOGICAL & MORPHOMETRIC ALTERATIONS OF PULMONARY TISSUE: A COMPARATIVE STUDY

Author: ***Namozov Elyorbek Ilhom og'li***
Osiyo Xalqaro Universiteti

1. Introduction

Carbon monoxide (CO) is a ubiquitous gas produced by incomplete combustion of carbon-containing fuels. In Europe and globally, occupational and ambient exposures to CO remain a public health concern. CO binds to haemoglobin, forming carboxyhaemoglobin (COHb) and reducing the oxygen-carrying capacity of blood, thereby inducing tissue hypoxia. While the acute toxic effects of CO are well documented, less is known about the chronic impact of sustained low- to moderate-level CO exposure on lung morphology and morphometry. The present study seeks to fill this gap by comparing morphological and morphometric indices of lung tissue in controlled animal models and in the light of human epidemiological evidence.

2. Research Objectives

The primary aims of this study are to characterise the morphological changes (histopathological alterations) in pulmonary tissue following chronic CO exposure; to perform morphometric assessment of key pulmonary parameters (alveolar diameter, inter-alveolar septal thickness, capillary density); to compare findings from experimental models with European epidemiological data; and to interpret the pathogenic significance of these alterations with respect to chronic hypoxia, oxidative stress and structural remodelling.

3. Materials and Methods

Experimental design: Laboratory Wistar rats were divided into three groups: control (clean air), short-term CO exposure (1-3 months), and long-term CO exposure (6-9 months). Pulmonary tissues were harvested, fixed, and processed histologically using H&E, Van Gieson and PAS stains. Morphometric analysis included alveolar diameter, inter-alveolar septal thickness and capillary density measurements. Epidemiological correlation: Data from European studies on occupational exposure to gases and fumes were reviewed to support experimental findings. Statistical analysis was conducted using ANOVA, and morphometric parameters were correlated with COHb levels ($p<0.05$).

4. Results

Histopathological analysis revealed alveolar septal thickening, mild fibrosis and reduced alveolar diameters (by 20–30%) in the chronic CO exposure group. Capillary congestion and mild dilation were observed, along with increased pneumocyte type II proliferation. These morphometric changes correspond to chronic hypoxia and oxidative stress-induced structural remodelling. Comparable findings are reported in European occupational studies linking gas and fume exposure to impaired lung function and architectural alteration.

5. Discussion

The observed morphological and morphometric alterations suggest that chronic CO exposure can induce structural remodelling of pulmonary tissue characterised by septal thickening, alveolar air-space reduction, and capillary changes. These changes are mediated by persistent hypoxia, oxidative stress and vascular remodelling. Epidemiological data from Germany, the Netherlands and the UK confirm that long-term exposure to gases and fumes contributes significantly to structural and functional deterioration of the lungs, independent of smoking. This highlights the importance of continued preventive measures and further research into CO-specific chronic toxicity.

6. Conclusion

Chronic exposure to carbon monoxide is associated with measurable morphological and morphometric alterations in lung tissue, including alveolar diameter reduction and septal thickening. These findings, integrated with European epidemiological evidence, underscore CO’s role in chronic pulmonary remodelling. Preventive strategies and detailed dose-response investigations are essential for understanding CO’s long-term pathophysiological effects.

7. References

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