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Visfatin Levels in Asthma, Chronic Obstructive Pulmonary Disease, and Pneumonia: A systematic Review and Meta-Analysis

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ABSTRACT

Introduction: Adipokines, particularly visfatin, are crucial for inflammation and metabolism regulation. Despite its metabolic role, visfatin involvement in the inflammation of respiratory disorders like chronic obstructive pulmonary disease (COPD), asthma, pneumonia, interstitial lung disease (ILD), and bronchiectasis is increasingly recognized. This systematic review and meta-analysis aimed to assess serum visfatin levels in these conditions.

Search Strategy: PubMed, Scopus and Web of Science were searched through June 29, 2023, using these search terms and their equivalents: "visfatin" AND ("asthma" OR "COPD" OR "bronchiectasis" OR "pneumonia" OR "ILD"). Studies had to be human observational or trial-based and meet the following criteria: (1) patients diagnosed with COPD, pneumonia, asthma, ILD, or bronchiectasis, and (2) serum visfatin levels were assessed. Two independent reviewers extracted data and stored it in Microsoft Excel. Data were presented as OR with 95% CI. Heterogeneity was assessed using I^2 and Cochran's Q. Random- or fixed-effects models were applied based on heterogeneity. Publication bias was examined using funnel plots and Begg's and Egger's tests. Sensitivity analyses used the leave-one-out method. Subgroup analyses were performed for pneumonia, COPD, and asthma. Statistical analyses were conducted using CMA V.3.

Results: The search identified 397 studies. After screening, 16 observational studies were included; however, none focused on ILD or bronchiectasis. Thirteen studies entered meta-analysis with 803 patients and 458 healthy controls. Serum visfatin levels were not significantly different in patients and controls (OR = 0.941 [0.183, 4.834]; p = 0.942). COPD analysis with 397 cases and 260 controls showed no significant difference between patients and controls using random effects (OR = 0.947 [0.326, 2.751]; p = 0.921) or sensitivity analysis. The fixed-effect model showed a significantly lower visfatin in COPD (OR = 0.725 [0.539, 0.976]; p = 0.034). Asthma analysis with 160 cases and 73 controls indicated no significant difference between patients and controls using random- and fixed-effects (random: OR = 0.062 [0.0001, 158.669]; p = 0.488; fixed: OR = 0.769 [0.380, 1.554]; p = 0.464). Sensitivity analysis revealed higher serum visfatin levels in patients with asthma (OR = 8.473 [3.927, 18.280]; p = 0.0001). Pneumonia analysis included 246 cases and 125 controls. Fixed and random-effect models demonstrated significantly higher visfatin in patients (random: OR = 33.733 [5.326, 213.643], p = 0.0001; fixed: OR = 45.997 [28.369, 74.579], p = 0.0001). COPD severity correlated with higher IL-6 and visfatin, and visfatin exhibited a negative association with SpO_2 and FEV1.

Conclusion and Discussion: Hypoxia and inflammatory cytokines likely stimulate the production of visfatin. This hormone inhibits neutrophil apoptosis, prolongs their lifespan, and contributes to inflammation in COPD. Underweight patients with COPD exhibit higher levels of visfatin, potentially due to severe systemic inflammation, which may account for the observed heterogeneity in results. Additionally, visfatin levels are elevated in asthma, consistent with its inflammatory characteristics. Asthma involves various immune cells, with Th2 cells and non-classical lymphocytes playing significant roles. Similar to COPD, differences in BMI account for variations in visfatin levels across asthma studies. Elevated visfatin levels are associated with the severity of pneumonia and hypoxia, serving as a prognostic biomarker.

Keywords: Asthma, Chronic obstructive pulmonary disease, Pneumonia