CORRELATION BETWEEN LIPID PROFILE AND IMMUNOLOGICAL MARKERS IN POST-COVID PATIENTS WITH DELAYED FRACTURE HEALING

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Annotation The treatment of traumatic injuries with delayed bone consolidation remains a pressing issue due to its severe functional and social consequences. The COVID-19 pandemic has intensified systemic immune and metabolic disturbances that impair osteogenesis. Understanding how lipid disorders correlate with inflammatory and immune markers is crucial for developing targeted therapies. This article provides scientific research information about correlation between lipid profile and immunological markers in post-COVID patients with delayed fracture healing.

Keywords: bone healing, Post-COVID-19, patients, bone fractures.

Relevance: T helper 17 cells (Th17 cells), a subset of CD4+ T cells, are key in bone homeostasis and immune responses during SARS-CoV-2 infection. Th17 cells produce IL-17, a stimulator of osteoclastogenesis, and express RANKL, a key mediator of bone homeostasis. During SARS-CoV-2 infection, Th17 cells contribute to the pathogenesis of the cytokine storm, which is known to promote bone loss. Elevated IL-17 levels are observed in mild cases of COVID-19, further implicating the role of Th17 cells in the disruption of bone homeostasis. An imbalance between Th17 and regulatory T cells is known to contribute to bone-related diseases, providing another mechanism by which SARS-CoV-2 infection may impact bone.

Purpose of the study: To assess the correlation between lipid metabolism parameters and immunological indicators of inflammation in patients with delayed fracture healing after COVID-19.

Materials and Methods: The study included 126 patients with post-COVID long bone fractures. Lipid markers (TC, LDL, HDL) and immune-inflammatory parameters (IgA, IgG, IgE, CRP, IL-1β, IL-4, IL-6, lactoferrin, INF-γ) were evaluated by VektorBest. Correlation coefficients were calculated using Pearson or Spearman methods; significance was set at $P < 0.05$.

Research Results: Elevated total cholesterol and LDL showed moderate positive correlations with IgA, IgG, IgE, CRP, IL-1β, IL-4, and IL-6 levels. Negative correlations were observed between LDL/TC and lactoferrin and INF-γ, indicating suppression of osteogenic and antiviral responses. HDL demonstrated inverse correlations with pro-inflammatory markers, reflecting its protective role.

Dyslipidemia was associated with chronic inflammation and immune dysregulation. These changes likely impair bone regeneration and promote prolonged consolidation delays.

Conclusion: Dyslipidemia contributes to immune imbalance and inflammatory activation, leading to impaired fracture healing in post-COVID patients. Monitoring lipid profiles may aid in predicting and managing delayed bone regeneration.

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