POST-COVID-19 OSTEOPOROSIS: EMERGING CHALLENGES AND THERAPEUTIC PERSPECTIVES

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Abstract. The COVID-19 pandemic, beyond its acute respiratory effects, has led to a wave of long-term complications, including a notable increase in post-infection osteoporosis. Recent studies suggest that SARS-CoV-2 disrupts bone remodelling processes via immune dysregulation, cytokine storms, and prolonged immobilization. Furthermore, corticosteroid therapy and reduced physical activity during recovery exacerbate bone demineralization. This article explores the pathophysiological mechanisms, clinical consequences, and therapeutic strategies for managing post-COVID-19 osteoporosis. Understanding these interlinked processes is vital for early intervention and preventing fractures in vulnerable populations, especially the elderly and chronically ill. Timely diagnosis, rehabilitation programs, and targeted pharmacological treatments could help mitigate the burden of this emerging skeletal health crisis.

Key words. COVID-19, osteoporosis, post-COVID syndrome, bone loss, cytokine storm, corticosteroids, vitamin D deficiency, bone mineral density, fracture risk, bone metabolism

Introduction. The global health crisis caused by the novel coronavirus disease (COVID-19) has left a lasting impact on multiple organ systems. While the respiratory tract was primarily affected, it is now evident that the virus also exerts long-term effects on the musculoskeletal system. Among these, osteoporosis characterized by decreased bone density and increased fracture risk has emerged as a growing concern in post-COVID-19 patients.

SARS-CoV-2 not only triggers acute systemic inflammation but also induces a cascade of immune responses, hormonal imbalances, and prolonged inactivity,

all of which are known contributors to bone loss. In addition, many COVID-19 patients were treated with glucocorticoids, a well-documented risk factor for secondary osteoporosis. This compounding of biological and treatment-related factors has raised alarm within the medical community.

As countries transition into the post-pandemic phase, physicians are beginning to observe an increase in osteoporotic changes and fragility fractures in patients with a history of COVID-19. This highlights the need for a comprehensive understanding of the underlying mechanisms, early diagnostic markers, and prevention strategies to reduce long-term skeletal complications. This paper aims to synthesize the latest scientific findings regarding post-COVID-19 osteoporosis and propose evidence-based approaches to management.

Pathophysiological Mechanisms. The pathogenesis of post-COVID-19 osteoporosis is multifactorial, involving direct and indirect effects of SARS-CoV-2 on bone metabolism. One major contributor is the cytokine stormóan exaggerated inflammatory responseófrequently observed in severe COVID-19 cases. Elevated levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-?), and other pro-inflammatory cytokines activate osteoclastogenesis, promoting bone resorption while inhibiting osteoblast function.

Additionally, hypoxia caused by respiratory distress may impair osteocyte viability, further disrupting bone homeostasis. Another important factor is vitamin D deficiency, common among COVID-19 patients due to reduced sunlight exposure, malnutrition, and altered metabolism during illness. Vitamin D plays a critical role in calcium absorption and bone mineralization; its deficiency exacerbates osteoporosis risk.

The widespread use of systemic corticosteroids to control inflammation in moderate-to-severe COVID-19 cases significantly impacts bone density. Corticosteroids accelerate bone turnover, reduce calcium absorption, and increase

urinary calcium loss. Immobilization during hospitalization or recovery also contributes to disuse osteopenia, especially in elderly or critically ill patients.

Moreover, the ACE2 receptor, used by SARS-CoV-2 for cell entry, is expressed in bone tissue. Viral interaction with ACE2 may disrupt the renin-angiotensin-aldosterone system (RAAS), influencing bone remodeling processes. These complex interactions underscore the need for targeted monitoring of skeletal health in COVID-19 survivors

Epidemiology and Risk Factors. While comprehensive global data is still emerging, multiple observational studies have reported a rising trend in osteoporotic fractures among individuals recovering from COVID-19, particularly in older adults. According to recent European and Asian cohort studies, the incidence of vertebral compression fractures and hip fractures has significantly increased within 6ñ12 months following recovery from moderate-to-severe COVID-19.

- Key risk factors for post-COVID-19 osteoporosis include:
- Advanced age (>65 years)
- Female sex, especially postmenopausal women
- Prolonged hospitalization and ICU stay
- High-dose corticosteroid therapy
- Vitamin D deficiency
- Sedentary lifestyle during recovery
- Pre-existing chronic diseases (e.g., diabetes, hypertension, CKD)

Moreover, patients with multiple comorbidities are at a higher risk of accelerated bone loss due to compounded inflammatory and metabolic stress. These findings highlight the necessity for early screening and preventive measures in high-risk populations.

Clinical Manifestations. Post-COVID-19 osteoporosis may remain asymptomatic in its early stages, often going undetected until the occurrence of low-impact fractures. Common clinical features include:

- Chronic lower back pain or bone pain
- Loss of height over time
- Kyphosis due to vertebral collapse
- Fragility fractures, especially in the spine, hip, and wrist

In elderly or immunocompromised individuals, even minor falls may lead to severe fractures, significantly impacting mobility and quality of life. Some patients may experience delayed fracture healing, attributed to impaired bone regeneration following systemic inflammation.

Diagnosis and Assessment. Timely diagnosis of osteoporosis in post-COVID-19 patients requires a combination of clinical evaluation and diagnostic imaging. The standard method is Dual-Energy X-ray Absorptiometry (DEXA), which measures bone mineral density (BMD). A T-score of ? ñ2.5 confirms osteoporosis.

Other useful assessments include:

- Serum vitamin D levels
- Parathyroid hormone (PTH)
- Calcium and phosphate levels
- Bone turnover markers (e.g., CTX, P1NP)

In patients with recent COVID-19, these parameters should be monitored during follow-up visits, especially if corticosteroid use or immobilization occurred. Spinal X-rays are also recommended in cases of unexplained back pain.

Conclusion. The long-term skeletal consequences of COVID-19, particularly the emergence of secondary osteoporosis, demand urgent attention in clinical practice. As evidence continues to accumulate, it becomes increasingly clear that bone health must be prioritized in the post-pandemic healthcare landscape. Early

diagnosis, patient education, and individualized treatment can significantly reduce morbidity associated with osteoporotic fractures.

Healthcare providers must incorporate bone density screening and preventive care into COVID-19 recovery protocols, especially for vulnerable groups. A multidisciplinary approach involving internists, endocrinologists, physiotherapists, and nutritionists is essential for optimal outcomes.

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