DNA INTEGRITY IN ACUTE AND CHRONIC LONG COVID PATIENTS

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INTRODUCTION: Long COVID, also known as post-COVID-19 condition, comprises heterogeneous, multisystemic symptoms that persist or arise weeks to months after SARS-CoV-2 infection. The underlying pathophysiology remains unclear. One proposed mechanism involves genomic instability driven by DNA damage—particularly oxidative lesions—that could sustain inflammatory and clinical sequelae.

OBJECTIVE: To assess DNA integrity in individuals with acute and chronic long COVID versus uninfected controls using the alkaline and enzyme-modified comet assays.

METHODS: We conducted a cross-sectional study with 231 participants from Campo Bom (RS, Brazil), allocated into three groups: control (n=74), acute long COVID (symptoms 3–12 weeks; n=79), and chronic long COVID (symptoms >12 weeks; n=78). Peripheral blood was collected in heparin tubes. DNA strand breaks were quantified by the standard alkaline comet assay; oxidative purine lesions (8-oxoGua) were detected with formamidopyrimidine-DNA glycosylase (FPG). Slides were analyzed using Comet Assay IV. Group comparisons used nonparametric statistics (global comparison across three groups followed by pairwise tests when applicable); significance was set at $p \leq 0.05$.

RESULTS AND CONCLUSION: In the standard comet assay, mean %DNA in tail was 5.19±0.26 (control), 5.05±0.40 (acute), and 5.03±0.39 (chronic), with no significant difference among groups ($p=0.632$). A sex-specific effect was observed in the acute group, with males showing higher damage than females. All groups exhibited a significant increase after FPG treatment ($p<0.001$), confirming the presence of oxidative DNA lesions; however, no inter-group differences were detected post-FPG. Altogether, these findings indicate that oxidative DNA damage is present across participants irrespective of long-COVID status, suggesting that persistent symptoms are not explained by increased genomic instability. Further studies should investigate alternative mechanisms underlying long-COVID pathophysiology.

Keywords: Long COVID; DNA damage; oxidative stress; comet assay; FPG; genomic instability.

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