EVALUATION OF OXIDATIVE STRESS MARKERS AND TOTAL ANTIOXIDANT DEFENSE IN LONG-COVID PATIENTS

Aline Fagundes Cerbaro¹; Luciana Bavaresco Andrade Touguinha¹; Daniel Maurer Ferreira¹; Valéria Pretti Schumann¹; Ana Letícia Garcia²; Juliana da Silva²; Cátia dos Santos Branco¹

¹ Oxidative Stress & Antioxidants Laboratory, Institute of Biotechnology, University of Caxias do Sul, Caxias do Sul, Rio Grande do Sul, Brazil.

² Laboratory of Genetic Toxicology, La Salle University (UniLaSalle), Canoas, Rio Grande do Sul, Brazil

INTRODUCTION: Long COVID (LC) is a complex, multisystem condition that impacts over 60 million people worldwide. It is characterized by fatigue, post-exertional malaise, joint pain, sleep changes, and cognitive impairment, among other symptoms. In the post-acute phase of long COVID (LC), patients typically present symptoms from 3 to 12 weeks after SARS-CoV-2 infection, while in the chronic phase of LC, symptoms emerge more than 12 weeks after infection. Oxidative stress is a central mechanism in clinical toxicology, playing a significant role in the pathophysiology of LC. Due to the absence of effective treatments and reliable biomarkers, it is crucial to investigate the mechanisms underlying this condition. OBJECTIVE: To evaluate oxidative damage to lipids and total antioxidant capacity in the serum of patients with post-acute and chronic LC. MATERIALS AND METHODS: The sample consisted of 231 individuals divided into three groups: control (n=74), post-acute (n=79), and chronic (n=78). Serum was collected to assess oxidative damage to lipids (TBARS assay) and antioxidant capacity by Trolox Equivalent Antioxidant Capacity - TEAC assay. RESULTS: In the control group, there were 36 males (mean age 38.72 ± 2.35 years) and 38 females (42.08 ± 1.99). In the post-acute group. 39 males (46.08 ± 1.80) and 40 females (49.00 ± 1.39) were included. Forty males (45.23 ± 1.72) and 38 females (44.97 ± 1.63) years comprised the chronic group. Oxidative damage to lipids was shown to be higher in the LC groups when compared to the control, both for individuals with post-acute and chronic conditions. Lipid peroxidation plays a key role in the onset and persistence of LC symptoms by worsening inflammation and tissue damage. Regarding total antioxidant capacity, no significant difference was found between groups. The increment in TBARS levels without TEAC modulation suggests that the body's antioxidant defenses are insufficient against increased ROS production. CONCLUSION: The imbalance between oxidative damage and antioxidant capacity is a key feature of conditions like LC, and further studies are needed to better understand the disease's pathophysiology, aiming to develop antioxidant-based strategies.

Keywords: COVID-19; post-acute COVID syndrome; redox imbalance

Acknowledgments: CNPq, FAPERGS