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Perspective

Various Effects of Smoking in Patients with Systemic Lupus Erythematosus

Carlos Linn*

Department of Clinical Medicine, University of Oxford, Oxford, United Kingdom

DESCRIPTION

Systemic Lupus Erythematosus (SLE) is a type of autoimmune disease that is distinguished by persistent, multisystem inflammation. Patients' illness progression is unpredictable, resulting in end-stage organ destruction and early death. Smoking, according to epidemiological evidence, helps to the growth of ANA and dsDNA autoantibody development, both of which are important in the pathogenesis of SLE, especially Lupus Nephritis (LN), and cigarette smoking has been related to an elevated likelihood of developing a variety of autoimmune diseases, such as SLE. Smoking has been connected to cutaneous symptoms, damage accrual, including a rapid development of kidney failure in those with LN, and premature death in SLE patients. However, the link between smoking and SLE disease activity has received little attention.

Cigarette smoke exposures the epithelial cells of the larynx, bronchi, and lung to over 60 chemical carcinogens, each of which has the ability to harm DNA. Furthermore, smoking has been demonstrated to raise and reduce various proinflammatory and anti-inflammatory cytokines in the general population as well as in patients with and without SARS-CoV-2 (COVID-19), and in a Sjogren's disease cohort. However, there is (very) little data on the effect of smoking on serum cytokines in SLE patients, particularly in the context of drug use and organ damage. Cytokines such B-Cell Activating Factor (BAFF), Transforming Growth Factor Beta 1 (TGF-1), and interferons (IFNs) are linked to disease activity and severity in SLE.

As a result, if smoking increased to aberrant levels of these cytokines, it could have ramifications for SLE therapy and management. Thus, the goal of this study was to describe the impact of smoking on a variety of clinical, serological, and immunological features in a cohort of SLE patients, as well as to determine the connection between smoking as well as cytokine levels while controlling for age, gender, medication use, disease activity, and organ damage. Some studies found that current smoking is associated with higher BAFF levels and lower IFN-levels in SLE patients. This occurred against a backdrop of current smokers experiencing increased joint symptoms demanding higher NSAID usage, migraine, Raynaud's phenomenon, and higher GDA scores. Current smokers had a higher incidence of malar rash and mucosal ulcers, yet did not experience skin damage. Having smoked in the past was linked to an elevated risk of damage to organs and cancer development.

Current smokers and nonsmokers, on the other hand, were identical in terms of the remaining ACR97 categorization criteria items and scores, cross-sectional SLEDAI-2K scores, damage accrual, comorbidity, and other medication necessity.

The negative effects of cigarette smoking are widely known in the general population, and the relatively recent habit of consuming e-cigarettes is increasingly being demonstrated to be just as detrimental. The smoking rates of 35.4% of SLE patients and 25 (39.1%) ex-smokers were consistent with Norwegian national survey statistics on the prevalence of smoking and quitting at the time of the study. When compared to other studies of SLE patients, smoking prevalence was comparable to studies from the United States (32% current smokers) and Denmark (51% ever-smoked), but higher than studies from Canada (14% current smokers) and Brazil (8% current smokers).

Despite Norwegian efforts to promote the cessation of smoking, smoking prevalence is currently over 20% and is not expected to decline below 10% until 2029, indicating that doctors still have a chance to promote the cessation of smoking in their patients.

Correspondence to: Carlos Linn, Department of Clinical Medicine, University of Oxford, Oxford, United Kingdom, E-mail: linncarlos@gmail.uk

Received: 13-Mar-2023, Manuscript No. LOA-23-24264; **Editor assigned:** 15-Mar-2023, PreQC No. LOA-23-24264 (PQ); **Reviewed:** 06-Apr-2023, QC No. LOA-23-24264; **Revised:** 14-Apr-2023, Manuscript No. LOA-23-24264 (R); **Published:** 21-Apr-2023, DOI: 10.35248/2684-1630.23.08.237

Citation: Linn C (2023) Various Effects of Smoking in Patients with Systemic Lupus Erythematosus. Lupus: Open Access. 08:237.