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The Effect of Smoking on Rheumatoid Joint Pain Results

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Editorial

Rheumatoid arthritis is a chronic inflammatory illness that can be triggered by both genetic and environmental causes. One of the most prominent extrinsic risk factors for its development and severity has been identified as smoking. Recent research has given information on the pathophysiology of RA in smokers, such as oxidative stress, inflammation, autoantibody production, and epigenetic alterations. Epidemiologic research, as well as in vivo and animal models of RA, has shown a link between smoking and the development of RA. The rising use of biological agents in addition to traditional disease-modifying antirheumatic medications [1].

Researchers are looking into how smoking influences drug responsiveness in RA treatment. According to new findings, persons receiving anti-tumour necrosis factor therapy have a lower response and medication survival. Rheumatoid arthritis is a systemic inflammatory illness marked by persistent synovitis and the generation of autoantibodies against a variety of factors, including rheumatoid factor and cyclic citrullinated peptide. RA typically appears as chronic inflammation of the synovium, which leads to joint degeneration [2]. Uncontrolled RA can result in irreversible joint degeneration, diminished mobility and quality of life, as well as cardiovascular and other extra-articular problems. It is generally known that hereditary variables such as human leukocyte antigen, as well as environmental factors such as infection, UV light, radiation, and smoking, can influence the development of many autoimmune illnesses.

Cigarette smoking, among these risk factors, considerably raises the chance of not only numerous types of cancer, cardiovascular disorders, and infections, but also autoimmune diseases like systemic lupus erythematosus and RA. Although the precise pathogenic effect of smoking on RA is unknown, several mechanisms have been proposed to better understand how cigarette smoking plays a role in various autoimmune diseases, and citrullination has been reported to be an important factor in the development of RA in the anti-citrullinated protein antibody [3]. Smoking has previously been recognised as a major risk factor for RA in epidemiological studies. The most important studies are summarised. Some research show that smoking raises the chance of developing RA in males more than it does in women, whereas other studies show that smoking increases the risk of developing RA in women. Sugiyama recently conducted the first meta-analysis on the relevance of smoking as a risk factor for developing RA, which found that smoking is really a risk factor for RA in RF-positive men and heavy smokers. Smokers have roughly twice the chance of acquiring RA as non-smokers. Female smokers were at a slightly higher risk than nonsmokers [4].

Despite the fact that numerous prior research were unable to show a substantial relationship between smoking and the development RA in women Sugiyama provided quantitative evidence that smoking is an important risk factor for women in developing RA Several previous epidemiological studies showed an increasing risk of developing RA with a heavier lifetime burden of smoking while a recent report suggested that even light smoking had a connection with the development of RA. Di Giuseppe conducted a meta-analysis to quantitatively summarize accumulated evidence regarding the association of lifelong exposure to smoking and concluded that lifelong smoking was positively associated with the risk of RA, even among smokers with overall low lifelong exposure. The risk did not further increase with an exposure higher than in the same study. Smoking can increase the oxidative stress in the body. Pryor and Stone reported that there are two phases of cigarette smoke: as a particulate tar phase and a gaseous vapour phase, both of which contain very high concentrations of free radicals. Cigarette smoke is also known to activate endogenous sources of free radicals. It has been reported that oxidative stress increases in rheumatoid inflammation due to impaired antioxidant systems caused by free radicals [5].

Conflict of Interest

None.

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