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Opinion Article

Effects of Cigarette Smoking on Bone Metabolism

Mari Dallas*

Department of Bone Marrow Transplantation and Cellular Therapy, University of Pennsylvania, Philadelphia, USA

DESCRIPTION

Despite the fact that bone fracture is a common musculoskeletal problem, 5%–10% of fractures have poor healing, which results in delayed union or non-union fracture healing. Longer hospital stays and higher medical expenses are directly linked to these illnesses. In USA, 17% of all treatment costs go towards caring for people with common musculoskeletal disorders. The majority of these costs are attributable to the need for multiple surgeries following delayed or unsuccessful healing of fractures. In addition to the kind, location, and severity of the injury, a number of other factors, such as the patient's age, way of life, and coexisting disorders, play a role in the pathological process of poor fracture healing. Cigarette smoking can worsen the detrimental effects of other factors on bone homeostasis and has a negative impact on how fractures heal.

The healing of bone fractures is negatively impacted by Cigarette Smoking (CS). According to a clinical trial, heavy smokers had a much higher chance of poor fracture healing than non-smokers do after total joint arthroplasty. In another investigation, functional fracture repair following femoral osteotomy in mice exposed to CS for three months was found to be considerably impaired. Moreover, treatment with CS extract induced osteoporotic alterations in a cell model by enhancing osteoclast activity and inhibiting osteoblast activity. Alendronate and zoledronate are two examples of the often prescribed Bisphosphonates (BP) used to treat osteoporosis. The increased activity of bone-resorbing cells is thought to be the cause of the effect that these drugs have on preventing bone loss and fractures in musculoskeletal illnesses. *In vitro* studies have shown that BPs significantly counteract the osteoporotic alterations brought on by CS, especially in relation to CS.

A complex mixture of more than 6,000 molecular species makes up CS, with nicotine being the main pharmacologically active component. A thorough investigation into how nicotine affects bone fracture healing has not been conducted at all. Mesenchymal Stem Cells (MSC) osteogenic differentiation is negatively impacted by nicotine. On the other hand, nicotine positively influences MSC proliferation and differentiation in a dose-dependent manner at dosages lower than its blood concentration in smokers. After exposure to nicotine at concentrations similar to its serum levels in smokers, MSCs osteogenic differentiation remained unaffected. Nicotine has no impact on osteoclastogenesis either. There is therefore some proof that nicotine does not affect bone strength or homeostasis on its own. It is possible that other substances in CS have adverse effects on bone formation during fracture healing, as nicotine is unlikely to be the cause of the impaired fracture healing seen in smokers.

The best method to minimize the negative effects of CS on tobacco-related delayed fracture healing is to stop smoking cigarettes. Due to withdrawal symptoms and the abrupt end of their smoking routine when they stop, this may not be a choice for many smokers. The development of Reduced-Risk Products (RRP), such as heated tobacco products and electronic vapor products, has recently received more attention due to the limited success of Nicotine Replacement Therapies (NRT) like nicotine chewing gum, transdermal patches, and nasal sprays in supporting long-term smoking cessation. As they give nicotine while enabling the same ritual of use that is a part of the smoking experience, these RRPs have an advantage over NRTs. It has been demonstrated that while these RRP aerosols have similar nicotine concentrations to CS, they have fewer or less hazardous components overall. THS (a heated tobacco product) avoids tobacco combustion by only heating tobacco rolls up to 350 °C, in contrast to cigarettes that burn tobacco up to 600 °C. Chemical analysis, for instance, revealed that THS aerosol contains more than 75% water and 20% tar. In comparison to study cigarettes, which contain 35% water and 64% tar, this translates to a 3 fold decrease in tar levels. Also, when THS aerosol was compared to study cigarettes, 53 molecular species designated as hazardous and possibly harmful constituents by the U.S. Food and Drug Administration were reduced by more than 85%. Aqueous extract from THS is less harmful to primary human osteoblasts and MSCs than aqueous extract from a standard cigarette. THS's impact on osteoclast viability and functionality, as well as its capacity to control bone homeostasis, are unknown. Osteoclasts have drawn attention for their direct influence on osteoblast activity in addition to their involvement in bone resorption. Hence, it is crucial to investigate how THS affects the interaction between bone-forming and bone-resorbing cells, which is important for maintaining bone homeostasis.

Correspondence to: Mari Dallas, Department of Bone Marrow Transplantation and Cellular Therapy, University of Pennsylvania, Philadelphia, USA, E-mail: dallasmari.dr@gmail.com

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