CHAPTER **38**

RELATIONSHIP BETWEEN SMOKING AND PLANTAR CALLUS FORMATION OF THE FOOT

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Despite the decline in cigarette smoking in the last few years as reported by the Centers for Disease Control and Prevention, and the well known health risks in cardiovascular and pulmonary diseases, millions of Americans continue to smoke cigarettes. It has been proven by both experimental and clinical observation that cigarettes impair bone and wound healing. The purpose of this article is to review the chemical components of cigarette smoke and its relationship with plantar callus formation.

Increased plantar callus formation with patients who smoke cigarettes seems to be a common problem. There are approximately 46.6 million smokers in the US. There was a decline during 1997-2003 in the youth population but during the last years the rates are stable (1). Approximately 3,450 young people between 12 and 17 years of age smoke for the first time (2). Approximately eight billion cigarettes are smoked annually in the US alone. Per capita, cigarette consumption stands at 3,900 cigarettes per adult smoker per year (3, 4).

Smoking a single cigarette can produce cutaneous vasoconstriction for up to 90 minutes (5). Depending on the amount of inhalation, 85-98% of the nicotine in the smoke can remain in the body (6). The tobacco smoke components absorbed from the lungs reach the heart immediately. Smoking increases the heart rate, arterial blood pressure, and cardiac output. There is a 42% reduction in the digital blood flow after a single cigarette (7, 8). Nicotine has a direct cutaneous vasoconstrictive effect and is the principle vasoactive component in the gas phase of cigarette smoke. It is an odorless, colorless, and poisonous alkaloid that when inhaled or injected, can activate the adrenal catecholamines from the adrenergic nerve endings and from the adrenal medulla, which cause vasoconstriction of vessels especially in the extremities. Nicotine also induces the sympathetic nervous system, which results in the release of epinephrine and therefore peripheral vasoconstriction and decreases blood flow (9, 10).

The authors believe that vasoconstriction of the peripheral vessels can lead to atrophy of subcutaneous tissue, which in turn can lead to increased bone to skin contact and callus formation. These same catecholamines act as cofactors in the formation of chalones, which are wound hormones that inhibit epithelization (Figures 1-5).

Whole fresh cigarette smoke has more than 4,000

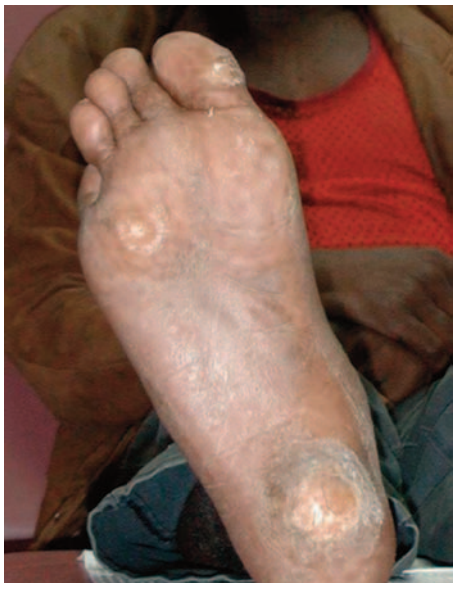

Figure 1. Multiple tobacco induced plantar lesions.

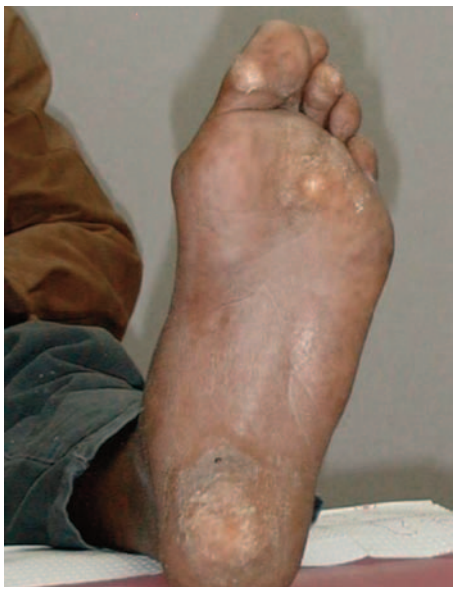

Figure 2. More than 2 lesions indicates tobacco induced plantar lesions.

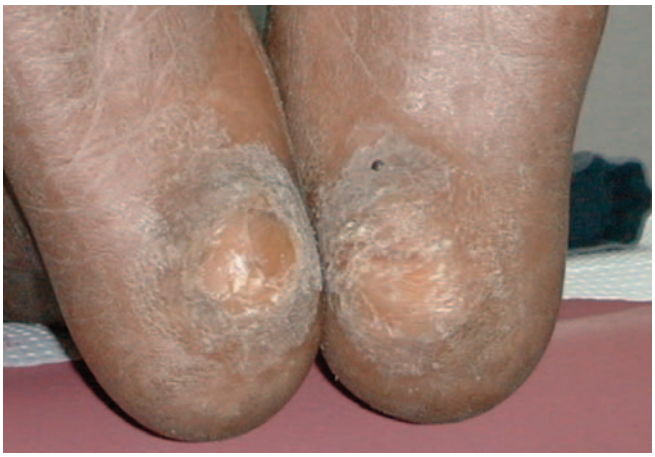

Figure 3. Plantar heel callous indicates tobacco hyperkeratosis.

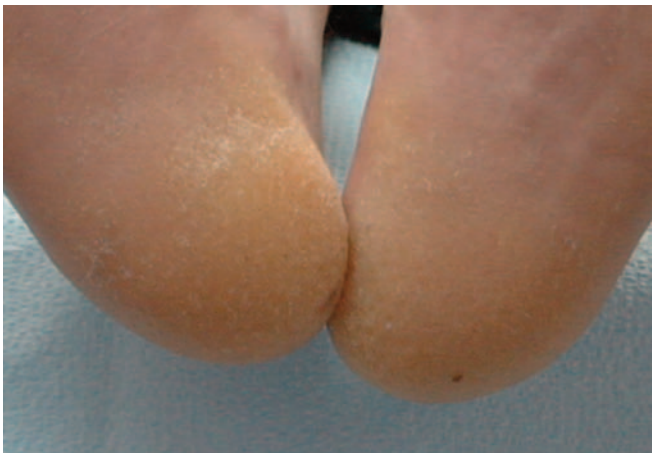

Figure 5. Four lesions including plantar heel.

different compounds either in the gaseous or particulate phase with the two most common gases in smoke being carbon monoxide and hydrogen cyanide. Two to three milligrams of nicotine and twenty to thirty milliliters of carbon monoxide are present in each cigarette (3, 4). It takes a minimum of twelve hours in order to clear the carbon monoxide from the blood and return the carboxyhemoglobin level to normal (4). The affinity of carbon monoxide hemoglobin binding is 200 times that of oxygen so the carbon monoxide inhibits the binding of oxygen and this in return leads to decreased oxygen carrying capacity of hemoglobin. This further reduced amount of oxygen to tissue leads to cellular hypoxia and cutaneous vasoconstriction (4). Hydrogen cyanide blocks cellular oxygen metabolism therefore decreasing tissue oxygen consumption (9, 10). Smoking a cigarette for ten minutes decreases the tissue oxygen tension for almost an hour.

Also, the odorless, colorless, poisonous alkaloid nicotine has been shown to increase platelet adhesiveness and platelet turnover, which in turn leads to increased blood viscosity.

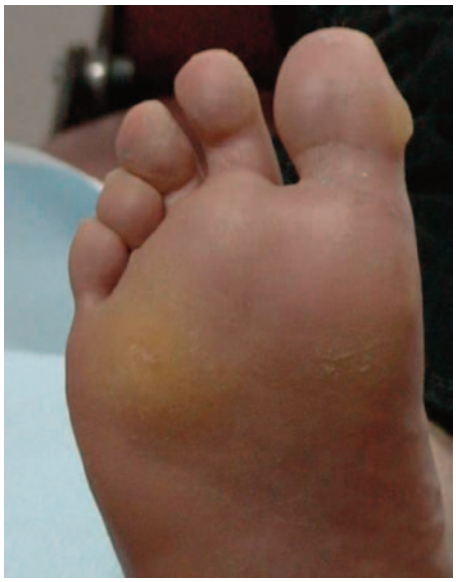

Figure 4. Four lesions including plantar heel.

This increased blood viscosity may cause microclots and decrease microperfusion (9-14). The rates of collagen deposition, epithelization, and angiogenesis are related to arterial and tissue oxygen content. Jensen et al showed that after smoking for ten minutes, the tissue oxygen content decreased to 60 +/- 6mmHg and continued to decrease to 44 +/- 3mmHg after thirty minutes (15). Compare this to tissue oxygen tension, which decreases to 0mmHg only after 30 minutes of tourniquet induced ischemia. Hunt TK et al, showed that a constant reduction of tissue oxygen tension in animals by 30% reduced collagen deposition by about 30-50% (16).

Smoking increases the risk for facial wrinkling two- to three-fold due to the decreased capillary and arterial blood flow in the skin. This may lead to ischemia of the dermis and leads to collagen and elastin damage. These are the connective tissue elements that are important to maintain the integrity of the skin. It has also been hypothesized that smoking decreases vitamin A levels, which provide protection against oxygen radicals that damage DNA and connective tissue (17).

In 1983, Ludwing and Hoidal (18) showed that neutrophils of smokers release excessive amounts of oxidants, which damage tissue and antiproteases. Fibroblasts can survive but cannot synthesize collagen without oxygen (16).

Strength is provided by members of the collagen family. The ability of skin to recoil after transient stretching is provided by elastic fibers. Elastin, a 70Kd glycoprotein forms an interlacing network of fibers in the papillary dermis and long thick fibers in the reticular layer of the dermis. Elastin is rich in glycine and prolene, but unlike collagen, it contains almost no hydroxylated amino acids. Elastin molecules are

cross-linked and form an extensive network. This network determines the capacity of stretching and recoil (12). It has been found that smokers have a higher blood concentration of neutrophils and lymphocytes (14). Neutrophils contain elastase, the enzyme that breaks up elastin. The authors believe that the increase in neutrophil levels will result in an increase in elastase levels and this in return will cause damage to the tissue elastin. The damaged elastin will cause the plantar skin to become less pliable and this will result in increased callus formation.

Cigarette smoke has been shown to increase plasma neutrophils and elastase activity, which may cause abnormal elastin formation (5). In vitro, cigarette smoke blocks the cross-linking that normally occurs with elastin (5). Elastin in smokers has shown to thicken and become more fragmented than in nonsmokers (5). Damage to elastin causes wrinkling of the skin and the wrinkles become coarse and the skin is thickened (11). This thickening of the skin can lead to increased plantar callus formation due to the loss of elasticity and pliability of the skin and increased sensitivity to friction (Figures 6-8).

Chronic ischemia of the dermis due to cigarette smoke also plays a role in damaged elastic fibers as well as the decrease in collagen synthesis (5). The typical pack-a-day smoker will remain hypoxic for most of the day (9, 11-13). The combination of the above effects will increase plantar callus formation.

There are other factors that are involved in the development of callus formation that were not discussed; however, our focus was to establish and determine the correlation between cigarette smoking and callus formation. The increase in elastase levels will result in the damage of elastin in the tissue and therefore lead to an increased callus formation. The vasoconstriction effect of nicotine contributes to increased callus formation by causing a decrease in blood supply and perfusion to the tissue, which ends in connective tissue degradation, atrophy, and more bone to skin contact.

Despite the known health hazard seen with cigarette smoking, millions of Americans will continue to smoke. Our duty as podiatrists is to provide the best care, attention, and education to our patients. Prevention should be our number one priority. Patients should be educated and aware of the relationship between smoking and plantar callus formation not only to benefit the health of their feet, but also to realize the extent of this habit. Sometimes we are only aware of the damage within when we can see it in the outside.

The clinical guidelines for tobacco induced hyperkeratosis are 1. multiple (more than three) plantar lesions; 2. plantar heel lesions identifies tobacco use; 3. multiple porokeratosis with plantar hyperkeratosis.

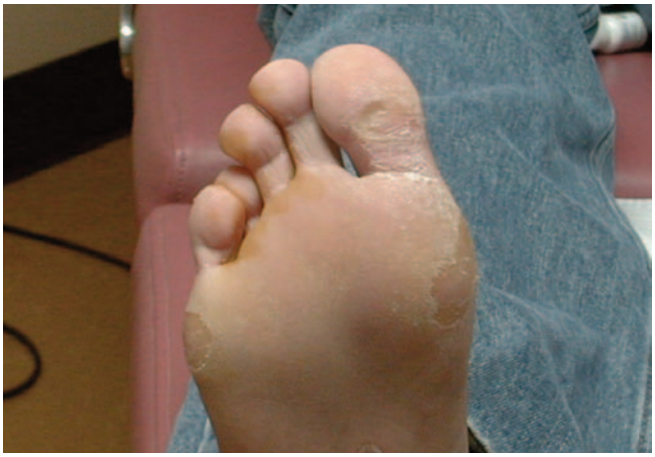

Figure 6. Five lesions including plantar heel.

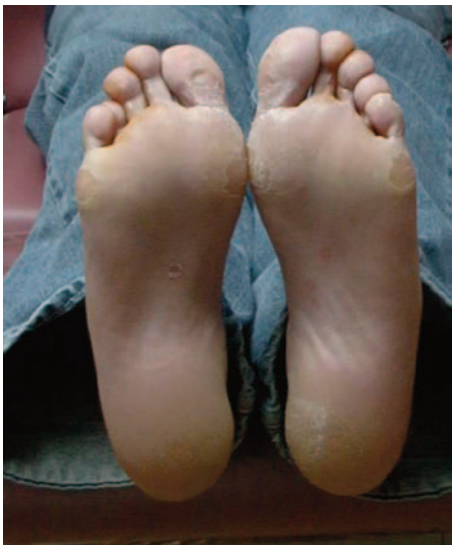

Figure 7. Five lesions including plantar heel.

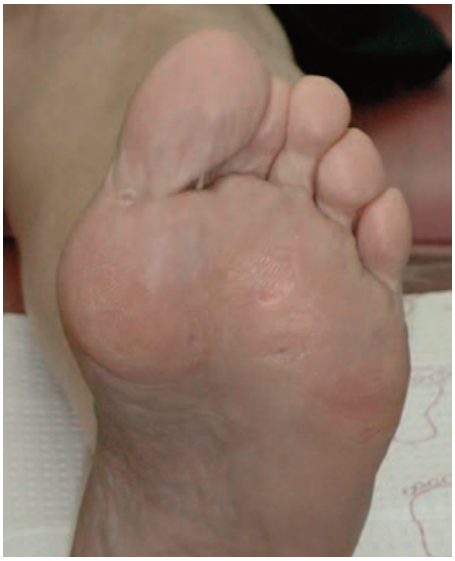

Figure 8. Multiple porokeratosis with plantar hyperkeratosis indicated tobacco use.

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