TREATMENT OF POROKERATOSIS PLANTARIS DISCRETA

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BASIC SKIN PATHOLOGY

The skin is composed of three layers, the epidermis, dermis and subcutis. The epidermis is formed by an ordered arrangement of cells called keratinocytes. The function of these cells is to produce keratin, a protein which serves a protective function. The dermis is composed of collagen protein. The subcutis is composed of fat cells. The epidermal layer is thickest on the palms and soles, measuring approximately 1.5 to 2.0 mm.

The epidermis may be divided into the following zones, beginning with the innermost layers: basal layer, malpighian or prickle cell layer, granular layer, stratum lucidum and stratum corneum. (Figure 1). These names reflect the changing appearance of the keratinocyte as it dif-

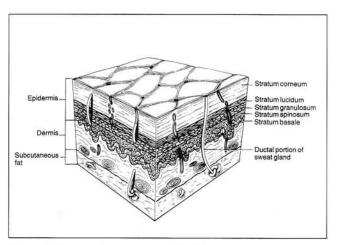


Figure 1. Cross section anatomy of the skin.

ferentiates into a cornified cell. Melanocytes are located in the basal cell layer of the epidermis, which is separated from the dermis by the basement membrane. The junction of the epidermis and dermis is formed by the basement membrane zone. The basement membrane zone is a semi-permeable filter, which permits the exchange of cells and fluid between the epidermis and dermis. It adds structural support to the epidermis and holds the epidermis and dermis together.

Eccrine glands and ducts are classified as adnexa of the skin. They originate embryologically as down-growths from the epidermis and are therefore of ectodermal origin. The eccrine gland originates from the subcutis near the junction of the dermis and subcutis. The straight, dermal portion of the duct is composed of cuboidal epithelial cells, while the intraepidermal component of the unit opens directly onto the skin surface. The distal portion of the duct, or the intraepidermal portion, is spiralled in configuration and is formed by keratinocytes. Cornification of the keratinocytes occurs within this portion of the duct. These eccrine sweat units are most abundant on the palms and soles.

The dermis is composed chiefly of collagen, a fibrous structural protein. Collagen fibers are loosely arranged in the upper or papillary portion of the dermis and are tightly bundled in a fascicle-like pattern within the lower or reticular portion of the dermis. The dermal vasculature consists of a superficial plexus and a deep plexus located respectfully in the papillary and reticular

portions of the dermis. Blood supply is greater in the superficial plexus. Associated with the vascular plexuses are the dermal lymphatics. The dermal layer is also rich in nerves.

PATHOPHYSIOLOGY

Porokeratosis Plantaris Discreta was described by Steinberg and Taub as an obstruction by pressure of the terminal portion of the plantar sweat duct. This presumably occurs by trauma, induced by mechanical imbalance. Pressure over the sweat gland orifice causes blockage of the duct and thus prevents the flow of sweat secretions, which results in marked dilatation of the duct, hypertrophy and cystic dilatation of the gland, and resultant pain.

Pathologically, the lesion is characterized by hyperkeratosis of the epidermal sweat duct with the formation of a coronoid lamella (keratin plug), absence of the stratum granulosum, acanthosis of the epidermis, and dilatation of the sweat gland apparatus in the corresponding deep dermis. Two diagnostic histologic hallmarks of the lesion are the coronoid lamella and absence of the stratum granulosum beneath the coronoid plug. Occasionally, hyperplasia and dilatation of the sweat gland units in the deeper portion of the dermis can be identified.

PREDILECTION

PPD lesions occur on the weight-bearing areas of the sole or heel, and are most commonly found beneath the metatarsal heads. Steinberg described the lesions as occurring directly beneath and lateral to weight-bearing surfaces.

The lesions occur more frequently in adults, age 22 to 25, with a 3:1 female to male predominance. Costello and Gibbs state that the lesions are more frequently found in black persons, and may also occur in areas not subject to pressure. The lesions may also be found in association with long-standing, thick calluses, if left untreated. The author notes that if the lesion is left untreated, it generally tends to increase in size and symptoms approaching 0.5 cm in diameter. Generally, the lesion is 1 to 2 mm in size, with a depth of approximately 1.5 cm. The lesions can be single or multiple, unilateral or bilateral, and a familial inheritance pattern has not been observed.

CLINICAL ASSESSMENT

The lesion is extremely painful to lateral and direct digital pressure. Only after the keratotic covering has been pared can these plugs be differentiated from a callus or verruca. The PPD lesions have a sharply marginated hyperkeratotic central core, which is rubbery-to-hard, opaque in appearance, white-to-yellowish in color, and devoid of blood vessels. (Figure 2)

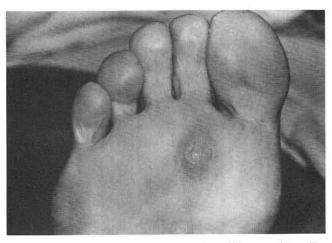


Figure 2. Clinically, PPD presents as a rim of hypertrophic callus surrounding a central plug (coronoid lamella) which is often recessed. No bleeding occurs on debridement and skin lines are absent.

In terms of a differential diagnosis, a callus has a more diffuse order and is not as sharply demarcated from the surrounding skin. In addition, calluses maintain skin lines. Verrucae have well-defined margins, adhere to the surrounding skin and have pinpoint bleeding upon surgical debridement. The PPD lesion has a central plug with a peripheral moat which separates the entity from the surrounding skin.

The PPD lesion is often, but not always, associated with other pathology such as a plantar exostosis, or biomechanically prominent metatarsal head. (Figure 3)

TREATMENT

Surgical treatment is often used to eradicate the lesion. The depth of lesion removal should include the lower dermis, so as to effectively remove the sweat gland apparatus.

The area surrounding the lesion is first anesthetized using a local anesthetic agent containing epinephrine (1:100,000 to 1:200,000 concentra-

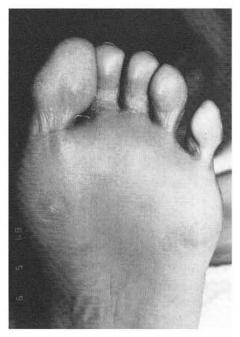


Figure 3. Distinct porokeratotic lesions beneath the first and fifth metatarsal weight-bearing areas in a patient with cavus foot. The patient had intense pain and was referred for cavus foot reconstruction.

tion). Sublesional injection of an anesthetic will help raise the lesion from its underlying bed. A #15 scalpel is then used to sharply circumscribe the lesion at its periphery. (Figure 4) As the dermis is incised, the lesion will separate from its bed. At times anesthetic fluid will flow from beneath the lesion. Care should be taken to avoid violating the underlying basement membrane of the dermis and the subcutis.

A large curette (4 mm) is helpful to bluntly separate and elevate the lesion from the underlying basement membrane, avoiding an inadvertent incision through the basement membrane of the dermis. (Figure 5A) The base of the defect is then sharply curetted and lightly electro-coagulated. (Figure 5B) The lesion is typically a hard, keratinized plug with underlying dermis. (Figure 6) A wide, superficial ulceration results.

Following excision, a topical antibiotic is applied with nonadherent gauze and a light compression dressing. The patient begins local wound care in 24 hours and continues to apply a topical antibiotic. Dispersive padding should be used throughout the healing process so that weight bearing is redistributed more effectively, thereby compensating for local biomechanical imbalances. Healing of the surgical defect is usually rapid,

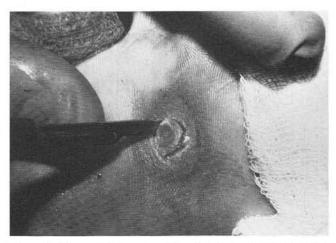


Figure 4. The central hyperkeratotic plug is circumscribed with a =15 blade debridement of the surrounding callus.



Figure 5A. Using a large curette, the lesion is bluntly excised, with the basement membrane intact.

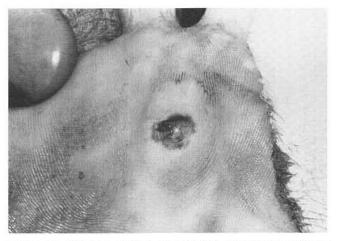


Figure 5B. Following excision of the lesion, the base and rim are further curetted, and electrocoagulation performed.

occurring over a 3 to 5 week period. (Figures 7A-D) At times, a small persistent scar may result. This scar usually resolves with time if padding and daily applications of Vitamin E are used. Recurrence of the lesion is possible, especially if the base has not been destroyed.

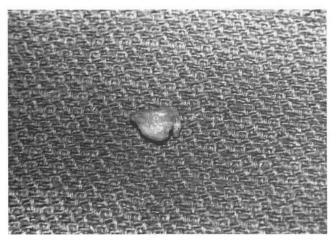


Figure 6. Typical appearance of an excised porokeratotic plug. Note the hyperkeratotic central plug with intact underlying dermis.

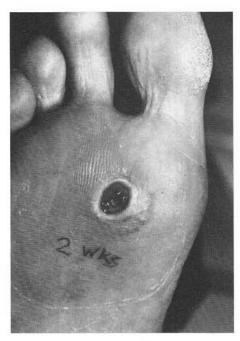


Figure 7B. Two weeks postoperative. Note the clean, healthy, superficial surgical wound.

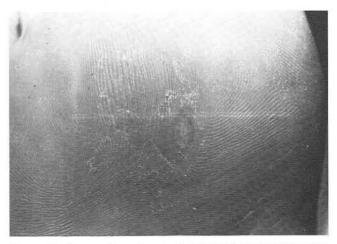


Figure 7A. Preoperative sub-second metatarsal porokeratotic plantaris discreta.



Figure 7C. Four weeks postoperative. Daily dispersion padding and topical antibiotics were utilized throughout the entire healing process.



Figure 7D. Three months postoperative, a small scar was apparent. The scar was treated with daily application of Vitamin E and continued dispersion padding.

Another method of treatment, advocated by Dribbon and Weisfeld, involves injecting the lesion with a sclerosing solution (4% absolute alcohol in Lidocaine with 1:100,000 epinephrine). The treatment consists of injecting 1 ml. per week until the pain disappears.

Limmer advocated the use of liquid nitrogen spray after the injection of a sublesional anesthetic, followed by sharp dissection of the plug. He reported a 90.5% cure rate in a series of 21 cases with an average follow-up of 22 months. Heiss and Gross reported a highly favorable response to topical application of .3% Vitamin A ointment twice a day with plastic occlusion during the night. Obvious clinical improvement, as well as marked symptomatic relief, were noted within two weeks.

Montgomery, as well as Steinberg and Taub, recommend complete surgical extirpation as the treatment of choice with mild electrocoagulation of the base of the lesion. A carbon dioxide laser can also be used, however, its use has not improved treatment outcome, or significantly shortened postoperative healing.

It has been stated that superficial treatment of PPD is often without benefit due to the depth of the lesion. However, the author has noted that early lesions which are 2 mm wide or smaller, may be initially treated with local application of 78% trichloroacetic acid, used in conjunction with biomechanical dispersion padding. This initial intervention may resolve the lesion in a limited number of clinical situations. In multiple, extensive PPD lesions, an initial trial of sublesional triamcinolone may be beneficial, again with the use of biomechanical padding. This treatment often results in shrinkage or resolution of the lesion.

Should the lesion fail to respond to this conservative therapy, then surgical extirpation should be considered.

CONCLUSION

The soles of the feet contain a large number of eccrine sweat glands per unit area. Weight bearing is a traumatic event which predisposes the sole of the foot to the development of PPD. These lesions are often encountered in clinical practice and represent, at times, a diagnostic dilemma. One should keep in mind the differential diagnosis as well as the biomechanical factors associated with these lesions.

Surgical extirpation or excochleation of the lesion under local anesthesia, coupled with light electrocoagulation of the base of the lesion, is often curative. The lesion can be recurrent, especially if the base of the lesion has not been removed.

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