
Punctate keratoses of the palms and soles and keratotic pits of the palmar creases

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Punctate keratoses of the palms and soles and keratotic pits of the palmar creases are frequently confused. A prospective study of 283 patients revealed a prevalence of 11% and 3%, respectively, in a metropolitan county hospital dermatology clinic. Punctate keratoses of the palms and soles and keratotic pits of the palmar creases are similar in size, number of lesions per palm, aggravation by trauma, and predominance in blacks and in men. These entities are different in appearance, distribution, age at onset, prevalence, symptoms, and prognosis. Punctate keratosis of the palms and soles and keratotic pits of the palmar creases should be considered independent entities. To help eliminate confusion, we propose that punctate keratoses refer only to the hyperkeratotic papules scattered diffusely in the palms and occasionally in the soles and that keratotic pits of the palmar creases refer only the hyperkeratotic, conical depressions confined to the palmar creases. (*J AM ACAD DERMATOL* 1990; 22:468-76.)

Punctate keratoses of the palms and soles are common lesions that are frequently overlooked. Keratotic pits of the palmar creases are less common and are frequently confused with punctate keratosis of the palms and soles, primarily, we believe, because of the inadequate terminology for these disorders.

Punctate keratosis of the palms and soles has been referred to as "punctate keratoderma,"¹ "keratoderma punctata,"² "keratosis punctata palmaris et plantaris,"³ "keratoma hereditarium dissipatum palmare et plantare,"⁴ "keratoderma disseminatum palmaris et plantaris,"⁵ "palmar keratoses,"⁶ "palmar and plantar seed dermatoses,"⁷ and other terms.^{8,9} *Keratotic pits of the palmar creases* is a term we introduce to help differentiate this distinct condition from punctate keratosis of the palms and soles. Other terms for keratotic pits of the palmar creases have included "keratosis punctata of the palmar creases,"¹⁰ "punctate keratosis of the palmar creases,"¹¹ "keratosis punctata,"¹² "keratoderma punctata,"¹³ "hyperkeratosis penetrans,"¹⁴ "len-

ticular atrophy of the palmar creases," and "hyperkeratosis punctata of the palmar creases."¹⁵ "Keratotic pits of the palmar creases" is a better term in that it describes both the distinguishing characteristics of these lesions—pits rather than papules—and their distribution in the creases rather than diffuse distribution on the palms and soles.

PATIENTS AND METHODS

We prospectively examined all patients seen during a 4-month period in the adult dermatology clinic at the Hennepin County Medical Center. In patients with evidence of punctate keratosis of the palms and soles (PK) or keratotic pits of the palmar creases (KPPC), the lesions were mapped on a diagram of the palms and a more detailed history was obtained.

CASE REPORTS

Case 1: Punctate keratoses

A 56-year-old white man had an approximately 15-year history of slowly accumulating, hyperkeratotic papules. When he was nervous or hot, the papules caused a burning sensation. He was a waiter and a dishwasher, and his occupation did not aggravate his disease. He had a history of arsenic exposure (Paris green) and "sweaty feet" but no palmar hyperhidrosis. Previous x-ray treatments for the palm lesions temporarily decreased the number of papules. On examination he had numerous papules of PK (Figs. 1 and 2) and bilateral midline canaliform dystrophy of both thumbnails. A few lesions were present on the soles.

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Accepted for publication May 5, 1989.

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Fig. 1. Case 1. Punctate keratoses of palms.

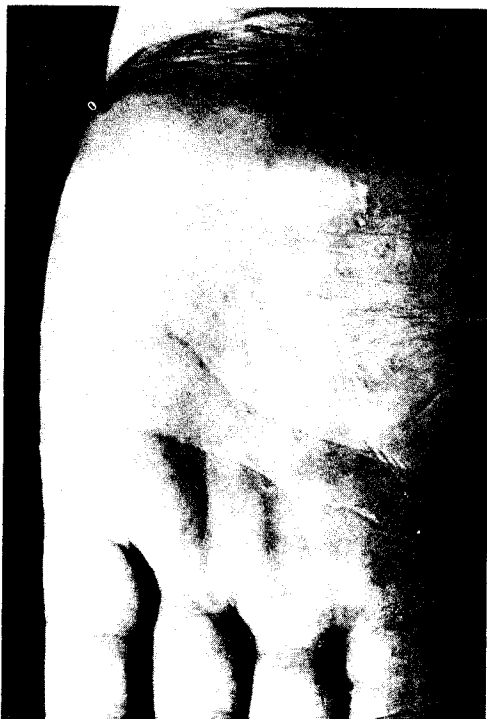


Fig. 2. Case 1. Punctate keratoses of palm.

Case 2: Keratotic pits of the palmar creases

A 34-year-old black man had a 3-month history of asymptomatic, craterlike depressions on the palms. He thought these depressions were warts and tried a nonprescription salicylic acid preparation without benefit. On examination, keratotic pits were found in the palmar creases of both hands, with a predilection for the proximal



Fig. 3. Case 2. Keratotic pits of palmar creases.

creases (Fig. 3). Treatment with topical tretinoin resulted in minimal improvement.

RESULTS

We examined 283 patients. Thirty-one patients (11%) had PK alone, 8 had KPPC (3%), and 13 had both (5%); a total of 52 persons (18%) were affected.

Blacks, as previously noted,^{10, 16-19} were predominantly affected; 41% of men and 27% of women had one or both lesions, compared with 13% of white men and 8% of white women (Table I). Some authors have noted a male predominance^{17, 20-22}; others have not.^{1, 5, 8, 10, 16, 23, 24} Of the affected patients, 43 had

Table I. Prevalence of PK and KPPC in clinic sample

| Race/Sex | No. of patients (%) | | | |
|-------------|---------------------|-------|--------|-------------|
| | PK | KPPC | Both | Total |
| B/M | 6(14%) | 4(9%) | 8(18%) | 18/44(41%) |
| B/F | 4(13%) | 2(6%) | 2(6%) | 8/30(27%) |
| W/M | 11(10%) | 1(1%) | 2(2%) | 14/111(13%) |
| W/F | 5(7%) | 1(1%) | 0(0%) | 6/74(8%) |
| Other race* | 5(21%) | 0(0%) | 1(4%) | 6/24(25%) |
| Total | 31(11%) | 8(3%) | 13(5%) | 52(18%) |

B, Black; W, white.

*Includes patients of either sex.

Table II. Numbers of PK and KPPC lesions in affected persons

| Sex | Total No. of PK | Total No. of KPPC | Total No. of PK and KPPC |
|-----|-----------------------------------|-----------------------------------|-----------------------------------|
| | No. of affected persons (Average) | No. of affected persons (Average) | No. of affected persons (Average) |
| M | 311/30 (10.4) | 127/16 (7.9) | 438/35 (12.5) |
| F | 54/14 (3.9) | 52/5 (10.4) | 106/17 (6.2) |

occupations involving manual labor (83%), and several noted that trauma exacerbated their condition, as previously reported.^{1, 5, 10, 16, 20}

The number of PK per person ranged from 1 to 41 (mean 8.3) whereas the number of KPPC per person ranged from 1 to 42 (mean 8.5). Previous authors reported the number of PK per person to range from 1 to 8 lesions (mean 1.9)¹⁶ to innumerable.^{24, 25} For KPPC the numbers of lesions per patient were 1 to 20 (mean 2.3).¹⁶

We found the average number of PK in men to be 2.5 times greater than in women, whereas the average number of KPPC was similar for men and women, as noted by others.^{10, 16, 20} The average total number of PK plus KPPC was approximately twice as great in men as in women (Table II). Patients had either a few or a great number of both lesions. Of the eight patients who had 10 or more lesions on both palms, six were black men.

Plantar keratoses were found in 18 of 36 affected persons (50%) whose feet were examined (50% of patients with PK, 30% of those with KPPC, and 60% of those with both PK and KPPC). The mean was three lesions per foot, without left or right predominance. Previous authors have reported few to hundreds of keratoses on the soles of patients with both conditions, with an incidence ranging between 1% to

85% of patients with PK^{16, 22} and from 14% to 45% of those with KPPC.^{10, 26}

PK were scattered on each palm, with an overall predilection for the left hand (left/right ratio 204:164) and the hypothenar eminence (Fig. 4). KPPC showed less of a predilection for the left palm (97:85) and were seen mainly in the proximal interphalangeal and distal medial transpalmar creases (Fig. 5). These observations have been noted by others.^{2, 13, 21, 27, 28} In our series, only one left-handed person was observed.

The age at onset for PK ranged from 15 to 68 years (mean 41 years), whereas for KPPC the age at onset ranged from 15 to 40 years (mean 25 years). This finding is consistent with previous observations.^{9, 19, 20} Thirty-nine percent of patients with PK previously noted the lesions, compared with 88% of patients with KPPC. Because these conditions may be asymptomatic, the stated age at onset may be overestimated whereas family history and prevalence are underestimated. Most patients denied knowledge of other family members with either process, and we did not attempt to examine family members. An autosomal dominant inheritance pattern has been reported for both PK^{1, 5, 9, 16, 20, 23, 24} and KPPC,²⁹ although inheritance has been disputed by others.^{8, 11, 19}

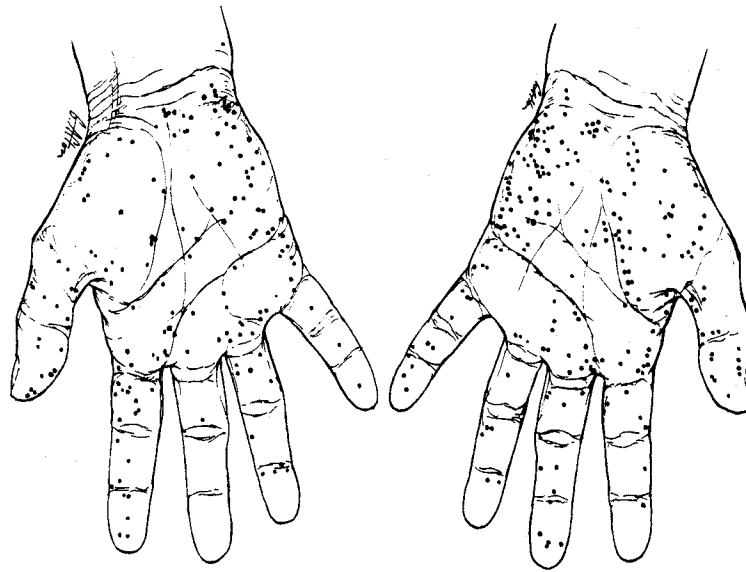


Fig. 4. Composite distribution of punctate keratoses on our patient's palms.

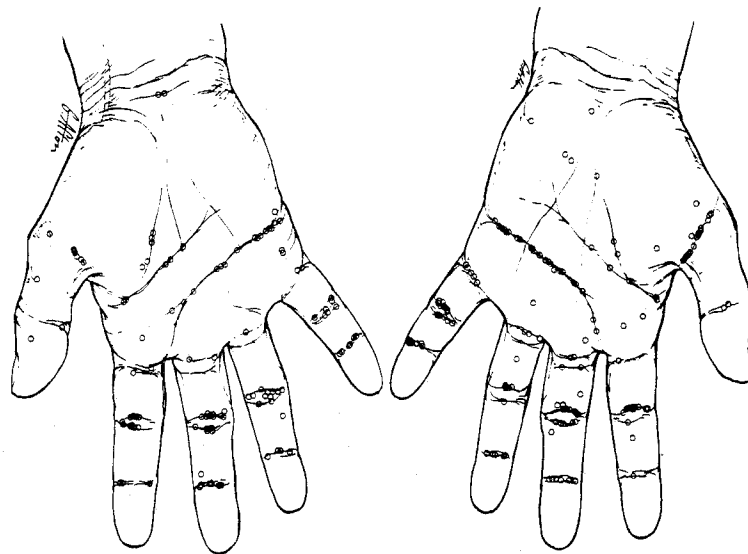


Fig. 5. Composite distribution of keratotic pits on our patient's palms.

Early observers related development of PK to tuberculosis, syphilis,⁸ hyperhidrosis or arsenic exposure,¹⁵ and internal malignancy,⁶ but these associations have not been substantiated.^{7, 16, 17, 20, 21, 27} Recently, however, there have been two reports of increased numbers of PK found in patients with lung carcinomas and particularly bladder carcinomas.^{22, 30} Reported associated physical findings include nail changes (longitudinal fissuring, onychogryphosis, onychauxis, and onychomadesis^{31, 32}), hypopigmentation,³³ hyperpigmentation

and blistering,³⁴ lichen nitidus,²⁶ trichilemmal cysts,³⁵ ichthyosis,²⁴ and knuckle pads and recalcitrant warts.²³ We found one patient with longitudinal fissuring associated with PK, and three of our patients had a history of arsenic exposure, none had a history of internal malignancy, tuberculosis, or syphilis.

KPPC has been reported to occur in association with Dupuytren's contractures,³⁶ pterygium inversum unguis,¹⁵ and dermatitis herpetiformis and psoriasis.²⁹ We found Dupuytren's contractures,

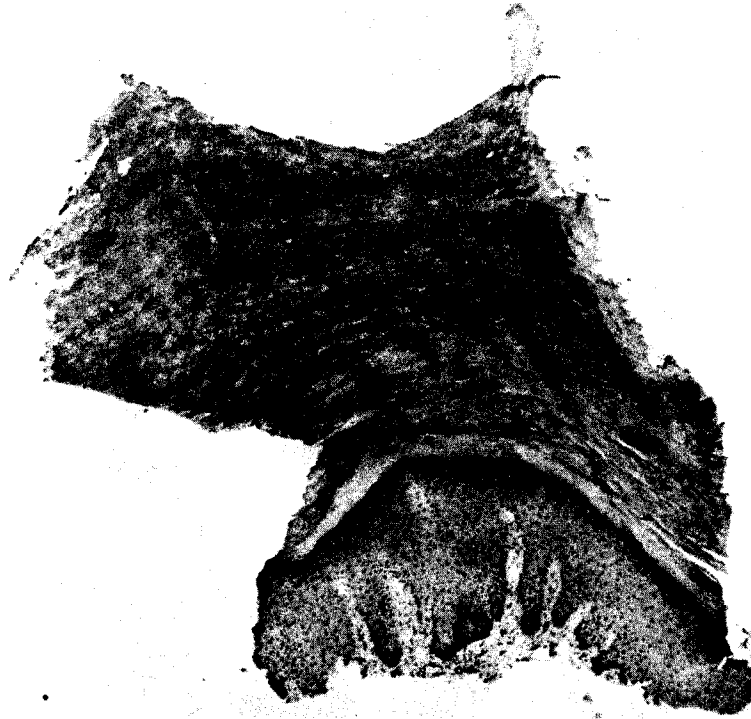


Fig. 6. Photomicrograph of punctate keratosis of palm. (Hematoxylin-eosin stain; $\times 10$.)

knuckle pads, and striate keratoderma to be associated with KPPC in our patients.

Histologically PK and KPPC are similar (Figs. 6 and 7). In both we observed compact hyperkeratosis without parakeratosis and a normal to mildly increased granular layer.^{20,22,25} Other authors* have noted thinning of the granular layer with parakeratosis in both PK and KPPC. Normal or central thinning and mild peripheral acanthosis of the malpighian layer were present in both.^{8,25,38} Pyknotic cells have been reported to cause vacuolization in PK.⁶ The basal layer was generally normal, but spongiosis² and nuclear hydrops⁵ in PK have been observed. In both, the dermis was normal without evidence of penetration by the stratum corneum, but flattened central papillae may be present.^{14,22,25,38} Occasionally a mild lymphocytic infiltrate was present in both.^{3,5,10,12,19,29,37} Some investigators have reported dilated and occluded blood²⁰ and lymph vessels² in PK. Some investigators have reported normal sweat ducts,^{5,10,16,20} whereas others have noted the ducts to be thickened, dilated, and apically occluded.³⁷ We observed normal eccrine ducts and glands, with ducts entering the lesions both centrally and peripherally (Figs. 7 and 8).

Sweat ducts were more frequently seen in KPPC than in PK. The only other histologic difference was the characteristic papular appearance of PK and the epidermal depression of KPPC. Electron microscopic examinations of PK have revealed only a thickened keratin layer and prominent nucleoli with a well-defined nucleolemma in the basal and spinosum layers,^{23,28} electron microscopic examinations of KPPC have been unremarkable.¹¹

DISCUSSION

PK are discrete, hard, scaling, 1 to 3 mm, round or oval, yellow to flesh-colored papules that are scattered on otherwise normal-appearing palms, the ventral aspect of the wrist, and, to a lesser extent, the soles.^{1-9,22,23,25} On the soles PK have generally been found to be larger than they are elsewhere^{22,37,39} and may cause difficulty in walking.²² Some authors^{5,23,27} have noted primarily a weight-bearing distribution, others^{7,8} have not. No evidence of systemic abnormalities⁴ or of similar lesions elsewhere on the body has been found.⁴⁰ Generally PK go unnoticed unless patients with symptomatic lesions note itch, burning, discomfort when the keratoses catch on clothing,^{25,28} or embarrassment when shaking hands.^{20,28} When patients remove the deep central portion to alleviate their discomfort, a kera-

*References 1-3, 10, 11, 14, 19, 37, 38.

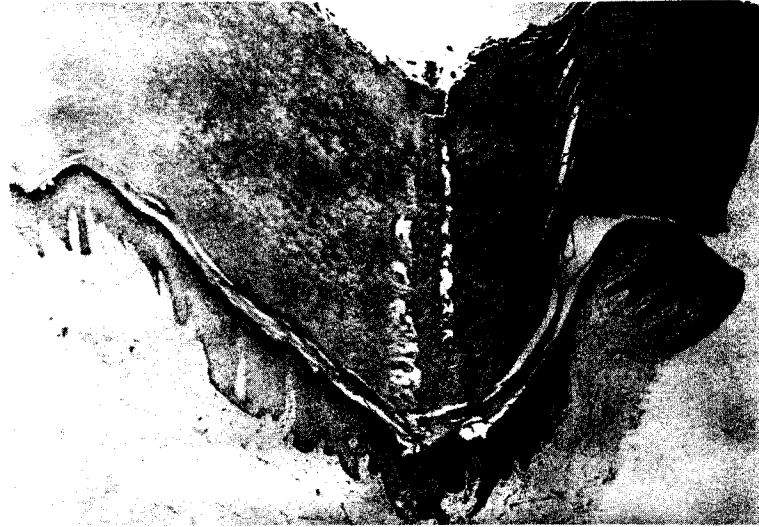


Fig. 7. Photomicrograph of keratotic pit of palmar crease. Note centrally located disruption of stratum corneum as result of eccrine duct. (Hematoxylin-eosin stain; $\times 10$.)



Fig. 8. Photomicrograph of keratotic pit of palmar crease shows peripheral eccrine duct. (Hematoxylin-eosin stain; $\times 10$.)

otic depression is left in which a PK will reform during a period of weeks.⁸

Once present, these lesions usually remain unchanged, although they may increase in size and number. * Only one report of spontaneous regression has been published²⁴; most accounts describe a chronic course.⁸ Attempted treatment modalities have included irradiation,^{5,22,25} lithium bromide,⁴

ammonium bromide,²⁵ cantharidin,²⁵ salicylic acid,¹³ cauterization,²² mechanical debridement, and excision, with only the latter achieving any permanent results.²⁰

KPPC are discrete, sharply margined, generally hyperkeratotic, 1 to 4 mm, circular, shallow, conically shaped depressions with occasional papules and are confined to the palmar creases. * KPPC may

*References 3, 5, 6, 8, 20, 21, 24, 25.

*References 10, 11, 13, 14, 16, 18, 19, 28, 38, 40.

Table III. Comparison of PK and KPPC*

| | PK | KPPC |
|-------------------------|---|---|
| Similarities | | |
| Size (mm) | 1-5 | 1-5 |
| No. [Average] | 1-41 (1-8) [8.3 (1.9)] | 1-42 (1-20) [8.5 (2.3)] |
| Race | Black > White | Black > White |
| Sex | M > F | M > F |
| Manual labor occupation | Yes | Yes |
| Scaling | Yes | Yes/No |
| Dissimilarities | | |
| Shape | Round to oval, dome-shaped papules | Round to oval pits |
| Sex ratio (M/F) | 2:1 | ~1:1 |
| Prevalence (%) | 11 (7-62) | 3 (0.05-56) |
| Sites of predilection | Left hand and hypothenar eminence | Medial transpalmar and proximal interphalangeal joint creases |
| Plantar lesions (%) | 50 | 30 |
| Age at onset (yr) | 15-68 (15-60) [41] | 15-40 (19-49) [25] |
| [Average (yr)] | | |
| Noticed (% of patients) | 39 | 88 |
| Symptoms | Itch | Pain |
| Prognosis | Stable | More likely than PK to increase in No. and size |
| Genetic pattern | Sporadic, (autosomal dominant) | Uncertain |
| Histologic differences | Papule (pyknotic, vacuolated epithelium; basal layer spongiosis, nuclear hydrops; dilated, occlusion of sweat ducts, blood vessels and lymph vessels) | Pit, more frequently associated with sweat ducts |
| Electron microscopy | (Nucleolemma in prominent nucleoli of basal and spinous layers) | (Unremarkable) |
| Associations | Longitudinal nail dystrophy (onychogryphosis, onychomadesis, hyperpigmentation and blistering, hypopigmentation, lichen nitidus, knuckle pads, and warts) | Dupuytren's contracture, striate keratoderma, knuckle pads (pterygium inversum unguis, dermatitis herpetiformis, psoriasis) |

*Parentheses denote information from previous authors.

be found concurrently with PK of the palms,¹⁶ soles,^{19,27} and with comedo-like lesions on the feet.¹⁰ Occasionally they can become scaling, ill-defined, oval craters with peripheral hyperpigmentation. One author³⁸ has reported a prodromal "ache" at the site where a pit would subsequently develop, followed by the development of a painful, scaling papule. No evidence of systemic abnormalities^{12,38} or of lesions elsewhere on the body has been found.¹⁴ KPPC may

remain unchanged but frequently increase in size and number. They are rarely more than an annoyance, but the tenderness³⁸ or the pain¹⁹ associated with KPPC may be so severe as to require surgical intervention if the process is localized^{14,19} or if a change of occupation if more diffuse.³⁸ Therapy with keratolytic agents or with retinoids has improved the condition only temporarily.^{10,13,40}

The differential diagnosis varies for each condi-

tion. For PK the differential diagnosis includes arsenic keratoses, verrucae vulgaris, lichen planus, secondary syphilis, linear epidermal nevus, keratosis follicularis, basal cell nevus syndrome, clavi, calluses, psoriasis, Cowden's disease, acrokeratosis verruciformis of Hopf, acrokeratoelastoidosis costa, lipid proteinosis, prurigo nodularis, pachyonychia congenita, epidermodysplasia verruciformis, hyperkeratosis lenticularis perstans, scabies, benign familial pemphigus, tyrosinemia, eccrine poromas,²⁸ yaws, keratoma plantare sulcatum, hyperkeratosis follicularis et parafollicularis in cutem penetrans, angiokeratoma,³ punctate palmoplantar keratoses acuminata,⁴¹ hereditary painful calluses,⁴² punctate porokeratotic keratoderma,⁴³ punctate porokeratosis of the palms and soles,^{28, 44, 45} focal acral hyperkeratosis,⁴⁶ and gouty tophi.⁴⁷

For KPPC the differential diagnosis includes basal cell nevus syndrome, porokeratosis of Mibelli, acrokerato elastoidosis,²⁸ focal acral hyperkeratosis,⁴⁶ punctate xanthoma,⁴⁸ and keratosis follicularis.

The cause of PK and KPPC remains unknown. Several etiologic theories have been proposed for PK. One theory previously advanced was an abnormal keratinization of the sweat duct. The hyperkeratinization was thought to be due to an inherent process in the ducts³⁷ or due to a drug.^{2, 21} The sweat ducts in our specimens were histologically normal, however, and not every PK or KPPC was associated with a duct. We were unable to elicit a consistent history of any possible offending drug. A viral cause has been proposed because of the possible relationship to virally associated carcinomas (lung and bladder) and because of a history of recalcitrant warts in some patients.^{21, 23, 30} Although PK may resemble verrucae clinically, no histologic evidence supports this theory.^{23, 28} Specific analysis for viral DNA content will be necessary to exclude this possibility. A suboptimal level of provitamin A or β -carotene has been proposed as a link between the development of cancer and PK, because of the involvement of vitamin A in cellular differentiation and β -carotene's ability to quench singlet oxygen.⁴⁹ If such a systemic abnormality were present, it would be difficult to explain why a healthy population is generally afflicted with PK and KPPC and why these processes are so localized. Finally, both PK and KPPC have been regarded by some in-

vestigators as an abnormal variant of callus formation.^{16, 18}

PK and KPPC seem to be due to an abnormal localized, hyperproliferative response in predisposed persons induced possibly by trauma. Our reasoning includes the clinical observation of their association with manual labor and their characteristic distribution on the palms and soles. The predominance of lesions on the ulnar half of the palm in both entities may be anticipated because it is the more powerful half of the hand. The dominant hand (the right hand in most people) is usually responsible for fine motor coordination, whereas the nondominant (usually the left hand) is the stabilizing hand. This predisposes the left hand, particularly the ulnar portion of the palm, to more trauma.^{50, 51} The overrepresentation of men in occupations involving manual labor may account for the difference in occurrence between the sexes. A hypermetabolic reactive process may be deduced from the histologic observations of hyperkeratosis with occasional parakeratosis and, in PK, an increased nucleolemma.

PK and KPPC are dissimilar enough that they should be considered independent entities (Table III). To help eliminate confusion, we propose that *punctate keratoses* refer only to the hyperkeratotic papules that are seen scattered diffusely on the palms and occasionally the soles, whereas *keratotic pits of the palmar creases* refer only to the hyperkeratotic conical depressions confined to the palmar creases.

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