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Original Research

Porokeratosis - A Non-Specific form of Cornification? Porokeratosis-Like Features Presenting Especially in Seborrheic Keratosis with Regression in A Series of 133 Patients

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A B S T R A C T

Objective: Porokeratotic-like columns in the stratum corneum may occur in a variety of circumstances apart from authentic forms of porokeratosis.

Methods: In a retrospective histopathological study, we investigated specimens in which a porokeratotic column was present.

Results: In 133 patients (68 women, 65 men) a porokeratotic-like column was especially seen in seborrheic keratosis (n = 112), the majority of them in advanced regressive forms (n = 81). Criteria for seborrheic keratosis in regression were a slight basket weave to compact orthohyperkeratosis, a flattened epidermis with effacement of rete ridges and frequently a lichenoid infiltrate (n = 56) with (n = 30) or without (n = 26) pigment incontinence. 31 cases showed features of more obvious seborrheic keratosis with a basket weave hyperkeratosis, acanthosis, infundibular "pseudocysts" and more early regressive features with the formation of cytoid bodies. In a few cases there was a porokeratotic column in prurigo (n = 2), epidermal nevus (n = 1), squamous cell carcinoma (SCC), type keratoacanthoma (n = 2), early SCC (n = 1), SCC in situ, type Bowen (n = 3), respectively. In 12 further cases these features were associated with SCC, type actinic keratosis.

Conclusion: In our series porokeratotic columns occurred in different conditions, most frequently however in seborrheic keratoses. We therefore consider the presence of a porokeratotic column in biopsies from patients without clinical evidence of any form of the known porokeratoses commonly to be within the histopathological spectrum of seborrheic keratosis, mostly the regressive lichenoid type.

Keywords: Seborrheic keratosis, Porokeratosis, Squamous cell carcinoma, Actinic keratosis, Bowen´s disease

1. Introduction

Seborrheic keratosis (SK) is one of the commonest skin lesions predominantly presenting in the elderly population[1-3]. Clinically, lesions present as slightly raised and rough (velvet-like) papules or plaques of varying size and pigmentation. Those which present on the eyelids, neck, axilla and groin frequently have a skin-tag-like appearance. Lesions on the trunk commonly follow the lines of Blaschko or present in the intertriginous areas. Especially those cases associated with solar exposure reveal histopathological alterations over time beginning as solar lentigo, gradually forming a reticulated SK. Sudden onset of multiple lesions may be an indicator of internal malignancy (Leser-Trélat sign). Thus, the etiology of SK varies considerably. In histopathology, different variants are known as well. These include the acanthotic, reticulated, clonal, papillomatous exophytic fibroma-like, inverted, stukko, melanotic, desmoplastic, keratoacanthoma-like[4], tumor of follicular infundibulum-like[5], large and clear cell acanthoma, apocrine and sebaceous[6], adamantanoid, transient acantholytic acanthoma-like, dyskeratotic acanthoma-like, and finally epidermolytic acanthoma-like variant[1-3]. In many cases there is histopathologic overlap of various variants of SK. The different histopathologies may be in part attributed to the multipotency of stem cells in the outer root sheath, but also interpreted as a secondary phenomenon when different parts of the hair follicle are stimulated by an inflammatory or neoplastic process which is also frequently seen in dermatofibromas[5]. SK may undergo regression which is signified by a lichenoid tissue reaction with the formation of cytoid bodies, pigment incontinence and over time a flattening of the epidermis[7]. In these later stages the diagnosis which is usually readily established may be challenging, clinically and histopathologically. - It is the purpose of this histopathologic study to stress that in these regressive forms of SK a porokeratotic column simulating authentic porokeratosis is frequently evident.

2. Materials and methods

Excisions from 133 patients were investigated. 71 cases were gathered from the files of the Dermatohistopathological Laboratory, Department of Dermatology, Medical University of Innsbruck, Austria over a period of 24 years (from 1998 to end of 2022). 62 further cases came from the files of the Private Institute of Pathology in Zams (from 2022 until fall of 2025), where two of us (B.G.Z. and B.Z.) are acting as consultants. Specimens have been processed in 4% formalin and stained with hematoxylin and eosin.

3. Results

Of 133 patients there were 68 women and 65 men with a mean age of 63,6 (range 29 - 87) and 65,1 (range 27 - 86) years, respectively. Clinical information was obtained from the records. Accordingly, 88 patients presented with a solitary lesion, the remaining 45 with two or multiple ones. In the latter group, if more than one lesion has histopathologically been investigated, only the lesion contributory to this study has been included. Specimens were from the head (n = 14), chest (n = 3), back (n = 3), abdomen (n = 2), shoulder (n = 1), upper arm (n = 7), lower arm (n = 33), hand (n = 6), buttock/hip (n = 3), upper leg (n = 16), knee (n = 4), foot (n = 2) and most commonly on the lower leg (n = 39). The clinical diagnosis varied from folliculitis, psoriasis, pityriasis lichenoides, pityriasis rosea, lichen planus, porokeratosis, viral wart, seborrheic keratosis, melanocytic nevus, prurigo nodule, squamous cell carcinoma (SCC) invasive, type keratoacanthoma, to in situ, types actinic keratosis or Bowen´s disease to trichoblastic carcinoma (basal cell carcinoma). Thus, the overall clinical appearance was quite variable. A number of lesions (50/133 (37,6%)) showed clinical features suggestive of porokeratosis, among them (9/133 (6,8%)) of the disseminated superficial actinic keratosis variant. The case of a solitary lesion of seborrheic keratosis in regression presenting as a roundish pale flat papule with a circular rim **(Figure 1A)** may serve as an example that the clinical presentation of the individual lesion may be indistinguishable from authentic porokeratosis. **(Figure 1B and 1C)** show an example of multiple lesions with an erythrosquamatous appearance. For comparison an authentic (multiple) form, namely linear porokeratosis **(Figure 1E)** is illustrated. In our series there was no evidence for a generalized disease process suggestive of a drug reaction which might be considered from histopathology showing the features of interface epidermatitis with cytoid bodies. However, one patient presented with eruptive small papular lesions on the arms and thighs and another on the knee associated with a thymoma and lymphoma (c-ALL), respectively, indicative of the Leser-Trélat sign **(Figure 1D)**.

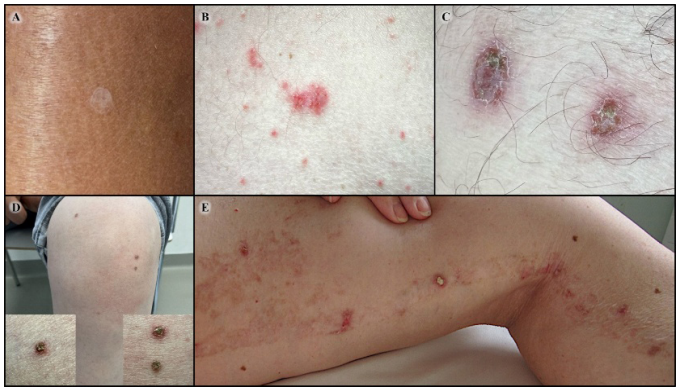


Figure 1A: Solitary flat, annular lesion on the shin in a 33-year-old female. Note the circular rim and paleness of the lesion simulating porokeratosis (case no. 82).

Figure 1B,1C: Multiple erythrosquamatous lesions on the trunk and upper leg in a 60-year-old male. In (B) the process is acute, in (C) in a more advanced stage. Note the scale and redness especially evident in (C) (case no. 94).

Figure 1D: Eruptive lesions on the left knee in a 29-year-old male suffering from acute lymphocytic leukemia for two years. Small reddish, scaly papules on the left knee are evident (case no. 96).

Figure 1E: For comparison, a 45-year-old female presenting with linear porokeratosis on the left leg. Multiple erythrosquamatous, annular lesions are evident.

Histologically, we classified most of our porokeratotis-like lesions as examples of SK in regression (n = 112), 81 of them with evidence of advanced regression. Criteria for this diagnosis were a slight basket weave to compact orthohyperkeratosis, a flattened epidermis with effacement of rete ridges and frequently a lichenoid infiltrate (n = 56) with (n = 30) or without (n = 26) pigment incontinence. In addition, all these cases showed remnants of SK with “dirty feet” and/or “dirty line”. There was no suspicious melanocytic component. In all cases in serial sections

a porokeratosis-like cornoid lamella was present, occasionally multiple ones **(Figure 2)**. 31 cases showed features of more obvious SK with a basket weave hyperkeratosis, acanthosis and infundibular "pseudocysts". In these earlier regressive lesions prominent cytoid bodies sometimes directly below the cornoid lamella were evident **(Figure 3)**. A cornoid lamella was directly located within the follicular ostium in 22 cases **(Figure 4)**. A porokeratosis-like pattern was also evident in a few cases in the context of prurigo (n = 2), epidermal nevus (n = 1), squamous cell carcinoma (SCC), type keratoacanthoma (n = 2), early SCC (n = 1), and in 3 cases Bowen´s disease, respectively **(Figure 5)**. In 12 cases a porokeratotic-like column was associated with SCC, type actinic keratosis (AK) signified by hyperparakeratosis, basal cell atypia and dyskeratosis; in 4 of them porokeratosis has clinically been considered.

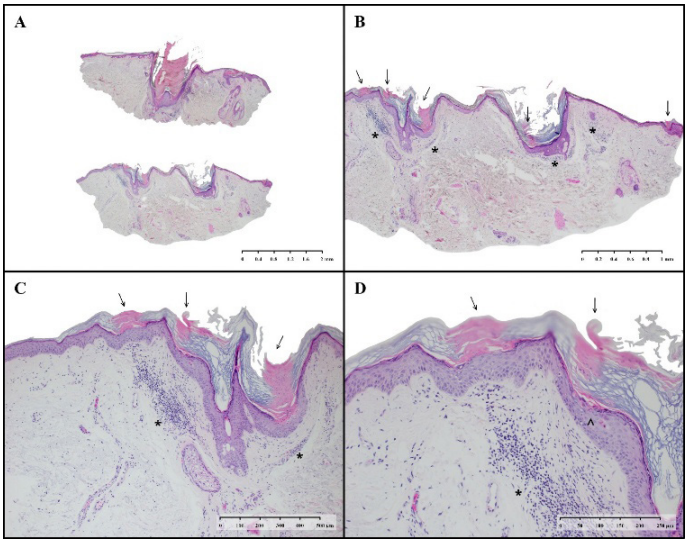


Figure 2: Seborrheic keratosis, late regressive stage, keratoacanthoma-like. Nearly already flat status of seborrheic keratosis in regression of a clinically typical seborrheic keratosis with signs of regression. Remnants of minimal/partial lichenoid lymphocytic infiltrate on the right side of upper part as well as around the infundibulum of a prominent hair follicle with porokeratotic columns on lower part (asterisk), thereby imitating keratoacanthoma. High power reveals porokeratotic columns (arrows) with remnants of cytoid bodies (arrowhead) and a lichenoid papillary infiltrate; H&E, individual scales inserted 2mm, 1mm, 500µm, 250µm (case no. 94).

4. Discussion

Similar to SK, porokeratoses represent a group of disorders with predominantly epidermal alterations. Based on the clinical presentation with different distribution of lesions, various forms of the disease have been established and comprise disseminated superficial actinic porokeratosis (DSAP), porokeratosis of Mibelli (PM), linear porokeratosis (LP; **Figure 1E**), porokeratosis palmaris, plantaris et disseminata (PPPD) / punctate porokeratosis (PuP), verrucous porokeratosis (VP, porokeratosis ptychotropica)[8-14]. Of note, there sometimes is considerable clinical overlap of the different forms of porokeratosis[8-10,15]. The hallmark of the individual lesion is a roundish macule with a hyperkeratotic circular rim (the cornoid lamella) and a slightly depressed center sometimes with a reddish appearance. Thus, in the right clinical setting its diagnosis is usually straightforward. In contrast, eruptive forms can manifest as bullous lesions[16] which may impede a rapid diagnosis as do cases of porokeratosis ptychotropica[8-10,17,18]. The most common form of porokeratosis is DSAP. Apart from the inherited variant, sporadic cases may be seen as well and then usually present later in life, around 35 years of age. Sporadic cases and an autosomal dominant mode of inheritance with variable penetrance are also seen in other forms of porokeratosis such as PM, LP, PPPD, PuP and VP[8-10]. In addition, porokeratosis, especially the eruptive variant, may be associated with immunosuppression, autoimmune disease and hematologic or other internal malignancy[8-10]. Interestingly, many of these latter patients had a history of previous porokeratosis, most commonly disseminated superficial porokeratosis[8], and the lesions may regress upon discontinuing the immunosuppressive treatment[9]. Malignant transformation especially to squamous cell carcinoma has been reported predominantly in the linear variant and in part been attributed to p53 overexpression[8-11,19-24].

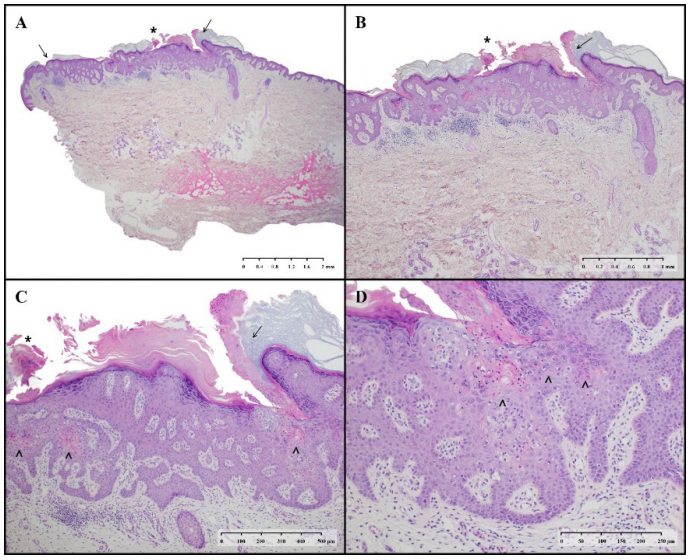


Figure 3: Seborrheic keratosis, reticular variant with prominent perivascular to early nodular lymphocytic infiltrate in the papillary dermis. Note silhouette of the lesion which is limited on right side with a hair follicle within which a porokeratotic column is evident (arrow). On the opposite side the lesion ends close to the excision margin without a porokeratotic column, but with a dense nodular dermal lymphocytic infiltrate (arrow). A second porokeratotic column is seen in the middle of the lesion (asterisk). Higher power reveals prominent cytoid bodies with early mass necroses not only on the bottom of the porokeratotic column but also in the midst of the lesion indicating ongoing regression of seborrheic keratosis (arrowheads); H&E, individual scales inserted 2mm, 1mm, 500µm, 250µm (case no. 87).

Histopathologically, porokeratosis typically reveals a circular column of hyperparakeratosis (cornoid lamella) extending vertically or an oblique angle accompanied by large vacuolated keratinocytes with loss of stratification devoid of a granular layer. Mild pleomorphism of keratinocytes with dyskeratoses can be a feature. This may be accompanied by a lichenoid lymphocytic infiltrate with the formation of cytoid bodies. Interrelation between the different forms of porokeratosis is also mirrored by molecular investigations since the various forms of porokeratoses reveal aberrations in the mevalonate-isoprenoid pathway[8-10,25-29].

Authentic forms of porokeratosis must be differentiated from inflammatory diseases with epidermal alterations such as the various forms of eczema, psoriasis, lichen planus, prurigo and tinea[11]. Clinicopathological correlation leads to the correct

diagnosis in most cases. On the other hand, a porokeratotis-like pattern may histopathologically incidentally be observed in infectious, inflammatory and inherited conditions, such as viral warts, HIV-associated epidermodysplasia verruciformis[30], psoriasis, lichen planus[31], prurigo[32], epidermal nevus, seborrheic keratosis[33], some ichthyoses[10,11], and, as documented herein, in squamous cell carcinoma in situ (type actinic keratosis as well as Bowen´s disease), and of note especially in regressive SK. Porokeratotic-like alterations in SK may clinically resemble authentic porokeratosis **(Figure 1A)**, and it may well be that examples previously designated as porokeratosis with a benign keratosis-like appearance fall into this spectrum[34]. This is likely also the case in those patients in our series clinically suspicious for porokeratosis since the histopathology in most of these cases sowed the features of SK in regression. Although the overall clinical appearance of regressive SK with porokeratotic features in our patients was non-diagnostic, frequently a circumscribed reddish and scaly lesion is present as shown in **(Figure 1B and 1C)**.

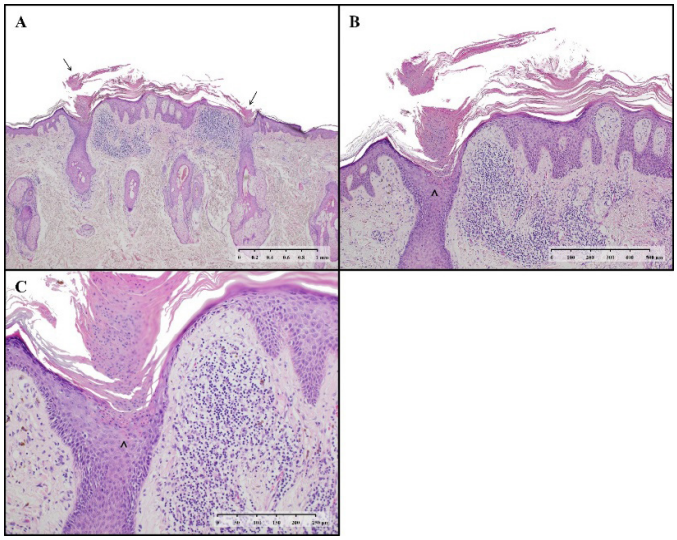


Figure 4: Seborrheic keratosis, hyperkeratotic variant with prominent lichenoid infiltrate with pigment incontinence in the papillary dermis. Note at periphery of this lesion on both sides porokeratotic columns within a hair follicle are present (arrows). Higher magnification reveals numerous cytoid bodies below the porokeratotic area (arrowhead); H&E, individual scales inserted 1mm, 500µm, 250µm (case no. 86).

Histopathologically, in our series, early stages of regressive SK frequently show the silhouette of a typical SK in which a cornoid lamella has formed accompanied by numerous cytoid bodies and a lichenoid inflammatory infiltrate. This phenomenon is occasionally located within the hair follicle similar to cases of follicular porokeratosis[8-11]. In later stages the silhouette of the lesion may become irregular and the epidermis flattened with only remnants of cytoid bodies and a patchy lichenoid inflammation with or without pigment incontinence. Not only one, but multiple porokeratotic columns may be evident **(Figure 2)**.

A porokeratotic pattern in SCC in situ, type AK likely represents one form of parakeratosis present in these cancerous lesions in early “embryonic” stages leading to a keratotic horn with alternating orthohyperkeratosis and hyperparakeratosis over time. Of note, 4/12 of these latter cases were clinically suspicious for porokeratosis as well. Porokeratosis-like epidermal alterations were also rarely seen in the context of prurigo, epidermal nevus, squamous cell carcinoma, type keratoacanthoma, in 3 cases of Bowen´s disease, respectively **(Figure 5)**, confirming the notion of an overall non-specific epidermal reaction pattern[10,11,30-33]. Parenthetically, it must be mentioned that a porokeratotic column may occur as a coincidence of authentic porokeratosis with other lesions. In this respect, clinicopathological correlation was unsuspicious in all our cases.

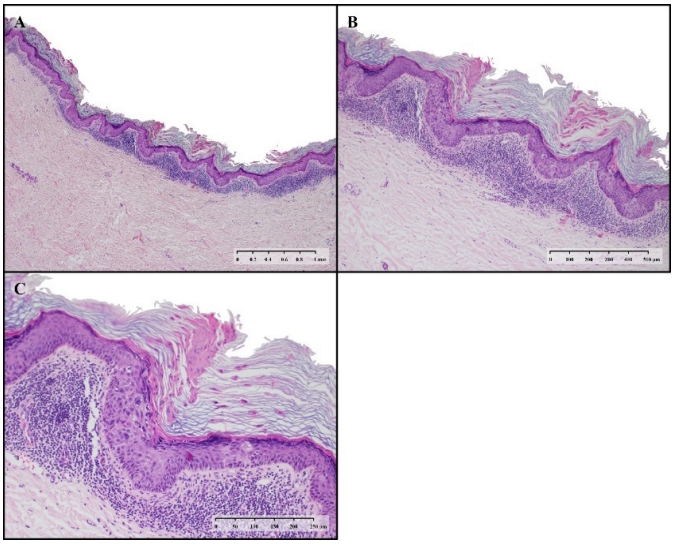


Figure 5: Squamous cell carcinoma in situ, type Bowen´s disease with a thickening of the epidermis and a prominent lichenoid papillary dermal lymphocytic infiltrate. High power reveals parakeratosis simulating a porokeratotic column above full thickness epidermal atypia; H&E, individual scales inserted 1mm, 500µm, 250µm (case no. 111).

In daily histopathological practice, especially small biopsies from solitary lesions in question histologically showing a lichenoid tissue reaction must be carefully screened for differential diagnosis. Thus, in view of the data provided herein a porokeratotic column in itself should not be considered to point towards porokeratosis as suggested[35]. Instead, due to the pigmentation phenomena and pigment incontinence especially seen in late stages of regressive SK, an underlying melanoma in situ, type lentigo maligna must be ruled out - this especially in view that in a previous study 34% of specimens in DSAP showed pigment incontinence as well[36] and in another investigation 25,8% of porokeratosis cases revealed melanocytic hyperplasia, leading to the designation “pigmented porokeratosis”[37].

In summary, in our series porokeratotic-like alterations were present in prurigo, epidermal nevus, squamous cell carcinoma, type keratoacanthoma, squamous cell carcinoma in situ, types actinic keratosis and Bowen´s disease and most commonly SK, the majority of the latter showing advanced type of regression (“benign lichenoid keratosis”). We therefore consider the presence of a porokeratotic-like column in biopsies from patients without clinical evidence of any form of the known porokeratoses frequently to be within the histopathological spectrum of SK, mostly the regressive lichenoid type.

5. Declarations

5.1. Ethics approval and consent to participate: This study represents a solely histopathological laboratory investigation and as such has not been presented for approval to an ethics commission.

5.2. Consent for publication: Yes.

5.3. Availability of data and materials: Yes.

5.4. Conflict of interest: None.

5.5. Funding: None.

5.6. Authors contributions: All authors contributed to this work

5.7. Acknowledgement

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6. References

1. Barthelmann S, Butsch F, Lang BM, et al. Seborrheic keratosis. J Dtsch Dermatol Ges, 2023;21: 265-277.
2. Gorai S, Ahmad S, Raza SSM, et al. Update of pathophysiology and treatment options of seborrheic keratosis. Dermatologic Therapy, 2022;35: 15934.
3. Wollina U. Recent advances in managing and understanding seborrheic keratosis. F1000Res, 2019;8: 1000.
4. Laimer M, Kraus L, Zelger B. Verrucous seborrheic keratosis with keratoacanthoma-like features, a pitfall in differential diagnosis. J Dtsch Dermatol Ges, 2015;13: 911-913.
5. Wilk M, Zelger BG, Zelger B. Tumor of follicular infundibulum - reappraisal in a series of 28 patients with critical review of the literature. J Dtsch Dermatol Ges, 2024;22: 223-235.
6. Hayani KM, Zelger BG, Kind P, et al. Seborrheic keratosis with sebaceous differentiation, a series of 8 cases and critical review of the literature. Am J Dermatopathol, 2021;43: 801-810.
7. Morgan MB, Stevens GL, Switlyk S. Benign lichenoid keratosis. A clinical and pathologic reappraisal of 1040 Cases. Am J Dermatopathol, 2005;27: 387-392.
8. Vargas-Mora P, Morgado-Carrasco D, Fustà-Novell X. Porokeratosis: a review of its pathophysiology, clinical manifestations, diagnosis, and treatment. Actas Dermosifiliogr, 2020:111: 545-560.
9. Kostopoulos-Kanitakis KA, Kanitakis J. Porokeratoses: an update on pathogenesis and treatment. Int J Dermatol, 2025;64: 62-71.
10. Pietkiewicz P, Korecka K, Salwowska N, et al. Porokeratoses - a comprehensive review on the genetics and metabolomics, imaging methods and management of common clinical variants. Metabolites, 2023;13: 1176.
11. Biswas A. Cornoid lamellation revisited: apropos of porokeratosis with emphasis on unusual clinicopathological variants. Am J Dermatopathol, 2015;37: 145-155.
12. Badea MA, Tatu AL, Saleva-Stateva M, et al. Five years follow-up of a patient with porokeratosis ptychotropica and MVK gene mutation. J Dtsch Dermatol Ges, 2024;22: 112-113.
13. Lammer M, Schmuth M. Scaly macules and papules on the extremities and a therapeutic clue. J Dtsch Dermatol Ges, 2024;22: 1442-1444.
14. Eicher L, Flaig MJ, Senner S, et al. Lineare Porokeratose. Der Hautarzt, 2020;71(1): 554-556.
15. Verma SB. A rare case of porokeratosis ptychotropica and coexistent linear porokeratosis in a 10-year-old boy. Clin Exp Dermatol, 2009;34: 501-502.
16. Rigo RS, Finnin CY, Schneiderman PI, et al. A case of bullous eruptive disseminated porokeratosis. J Cut Pathol, 2018;45: 968-969.
17. Yeo J, Winhoven S, Tallon B. Porokeratosis ptychotropica: a rare and evolving variant of porokeratosis. J Cut Pathol, 2013;40: 1042-1047.
18. Yang X, Jian X, Huang Q, et al. The Brownish verrucous plaques on buttocks: challenge. Am J Dermatopathol. 2023;45:e26. And answer. Am J Dermatopathol, 2023;45: 277-278.
19. Abbott-Frey AM, Coromilas AJ, Niedt GW, et al. Recurrent squamous cell carcinoma arising within a linear porokeratosis. J Drugs Dermatol, 2020;19: 205-206.
20. Saleva-Stateva M, Hess M, Technau-Hafsi K, et al. Molecular characterization and natural history of linear porokeratosis: a case series. J Am Acad Dermatol, 2021;85: 1603-1606.
21. Sasson M, Krain AD. Porokeratosis and cutaneous malignancy. A review. Dermatol Surg, 1996;22: 339-342.
22. Chang SE, Lim Y, Lee H, et al. Expression of p53, pRb, p16 and proliferating cell nuclear antigen in squamous cell carcinomas arising on a giant porokeratosis. Br J Dermatol, 1999;141: 575-576.
23. Rongioletti F, Rebora A. Disseminated porokeratosis with fatal metastatic squamous cell carcinoma. Am J Dermatopathol, 2002;24: 144-148.
24. Busch DA, Berking C, Schliep S, Erfurt-Berge C. Multiple squamous cell carcinomas arising in linear porokeratosis. Dermatologie (Heidelb), 2026;77: 33-36.
25. Kubo A, Sasaki T, Suzuki H, et al. Clonal expansion of second-hit cells with somatic recombinations or C>T transitions form porokeratosis in MVD or MVK mutant heterozygotes. J Invest Dermatol, 2019;139: 2458-2466.
26. Zhang Z, Li C, Wu F, et al. Genomic variations of the mevalonate pathway in porokeratosis. eLife, 2015;4: 06322.
27. Wang X, Ouyang X, Zhang D, et al. Two novel and three recurrent mutations in the mevalonate pathway genes in chinese patients with porokeratosis. Clin Cosmet Investig Dermatol, 2024;17: 191-197.
28. Atzmony L, Khan HM, Lim YH, et al. Second-hit, postzygotic PMVK and MVD mutations in linear porokeratosis. JAMA Dermatol, 2019;155: 548-555.
29. Jägle S, Juratli HA, Hickman G, et al. Porokeratosis plantaris, palmaris et disseminata caused by congenital pathogenic variants in the MVD gene and loss of heterozygosity in affected skin. Acta Derm Venereol, 2021;101: 00397.
30. Champagne C, Moore L, Reule R, et al. Cornoid lamella-like structures in HIV-associated epidermodysplasia verruciformis: a unique histopathologic finding. Am J Dermatopathol, 2015;37: 929-932.
31. Dhanta A, Kansal NK, Durgapal P, Divyalakshmi, C. Porokeratotic lichen planus. J Dtsch Dermatol Ges, 2019;10: 1063-1065.
32. Kang BD, Kye YC, Kim SN. Disseminated superficial actinic porokeratosis with both typical and prurigo nodularis-like lesions. J Dermatol, 2001;28: 81-85.
33. Asawanonda P, Noppakun N, Huiprasert P. Seborrheic keratosis-like porokeratosis: a case report. Dermatol Online J, 2005;11: 18.
34. Kim MS, Ha JM, Cho EB, et al. Porokeratosis presenting with a benign lichenoid keratosis-like appearance. Ann Dermatol, 2015;27: 778-779.
35. Foster C, Tallon B. Porokeratosis: a differential diagnosis to consider in benign lichenoid keratosis. Int J Clin Pathol, 2022;15: 56-62.
36. Shumack S, Commens C, Kossard S. Disseminated superficial actinic porokeratosis. A histological review of 61 cases with particular reference to lymphocytic inflammation. Am J Dermatopathol, 1991;13: 26-31.
37. Tan TSP, Tallon B. Pigmented porokeratosis. A further variant? Am J Dermatopathol, 2016;38: 218-222.