QUIZ CASES OPEN ACCESS

An Asymptomatic Lesion on the Penis

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1 | Case Presentation

A 28-year-old Caucasian man presented with an asymptomatic scaly macule on the left lateral penile shaft, present for and slowly enlarging over 3 months. He had trialled topical miconazole 2% and methylprednisolone aceponate 0.1% ointment without improvement. His past medical and family histories were otherwise unremarkable. He reported being sexually active with 3–5 casual male partners a year and was on tenofovir disoproxil fumarate 300 mg/emtricitabine 200 mg combination tablet once daily as pre-exposure prophylaxis against human immunodeficiency virus infection. A sexually transmitted infection screen 3 weeks before review was negative.

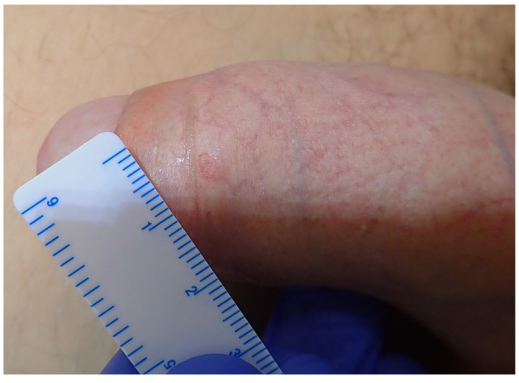

FIGURE 1 | .

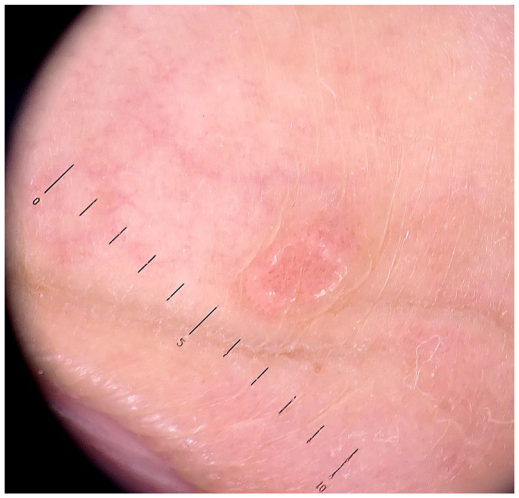

FIGURE 2 | .

Clinical examination revealed a well-defined 3 mm erythematous macule on the foreskin of the left lateral penile shaft, with slightly raised edges and a peripheral rim of scale (Figure 1). Dermoscopy revealed a keratin rim, central dotted blood vessels, and central reddish-brown globules (Figure 2). A 5 mm punch excisional biopsy was subsequently performed at the patient's request for complete removal of the lesion. The specimen was sent embedded in formalin for histological examination, which confirmed the diagnosis. There was no evidence of local recurrence or new genital lesions at follow-up 2 years later.

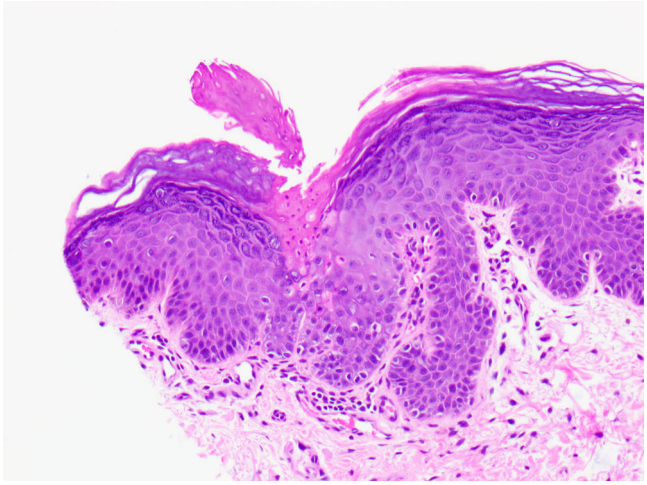

FIGURE 3 | .

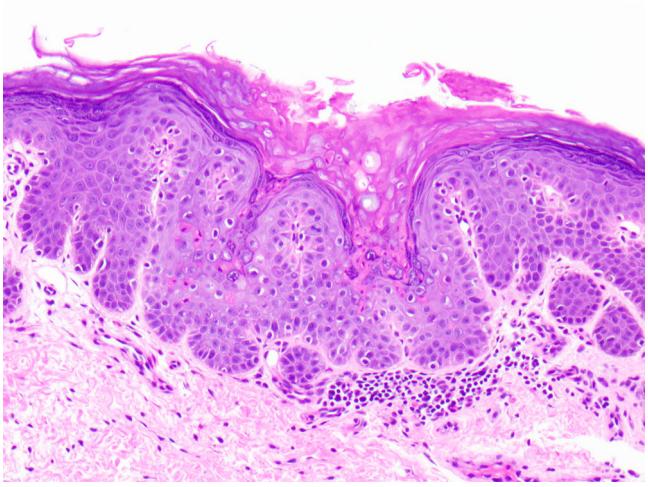

FIGURE 4 | .

2 | What Is the Diagnosis?

2.1 | Penile Porokeratosis

Histopathological examination (haematoxylin and eosin stain) demonstrated orthokeratin overlying a mildly acanthotic epidermis with hypergranulosis. There was no spongiosis or interface reaction. Two narrow epidermal invaginations were present, each comprising an angulated column of parakeratin overlying a small focus of epidermal thinning, hypogranulosis and superficial dyskeratotic cells (Figures 3 and 4). These findings are characteristic and diagnostic of porokeratosis.

3 | Discussion

Porokeratosis is a rare, heterogenous group of keratinisation disorders characterised by abnormal clonal proliferation of keratinocytes. Since porokeratosis confined to genitalia was first described in 1985 [1], fewer than 50 cases have been reported in the literature [2]. Although generalised forms can affect the genitogluteal region, primary porokeratosis in this area is extremely uncommon [3]. Multiple clinical variants of genitogluteal porokeratosis (GP) exist including classical porokeratosis of Mibelli-like, hyperkeratotic, ulceroproliferative, verrucous and penoscrotal porokeratosis (PP). GP typically manifests as extremely pruritic annular plaques with atrophic centres on the scrotum, penis, buttocks or proximal thighs, which may progress to plaques, nodules or ulcers [4].

The pathogenesis of porokeratosis remains poorly understood. Genetic mutations in the mevalonate pathway, ultraviolet radiation, immunosuppression and trauma (from friction and scratching) have been described, the latter mechanism being particularly implicated in GP [5]. Histologically, porokeratosis is characterised by the presence of a cornoid lamella, a compact thin column of parakeratotic cells overlying focal hypogranulosis. Other findings include lymphocytic infiltrate in the papillary dermis and dyskeratotic keratinocytes below the stratum spinosum [4]. Importantly, porokeratosis is considered a premalignant lesion given its hallmarks of clonal proliferation and abnormal maturation of keratinocytes, with well-documented cases of progression to malignancies such as squamous cell carcinoma (SCC), basal cell carcinoma (BCC) [5] and rarely, melanoma [4]. However, malignant transformation is rare in GP, with only one reported case of transformation into invasive SCC [6]. Although unclear, chromosomal instability, reduced immune surveillance and *p53* gene overexpression are thought to contribute to this risk [4].

The diagnosis of classical porokeratosis is easily achieved clinically given its typical appearance. However, GP closely mimics many other dermatoses and may be missed on visual inspection alone. Differential diagnoses include inverse psoriasis, tuberculosis verrucose cutis, viral warts, verrucous lichen planus, lichen simplex chronicus, lichen sclerosus, dermatophytic or candidal infection, secondary syphilis, acrodermatitis enteropathica, epidermal naevus, acantholytic disorders such as Hailey–Hailey disease and Darier disease, and extramammary Paget disease [4].

To date, multiple treatment options for porokeratosis exist, although primarily described in case reports. These include corticosteroids, cryotherapy, 5-fluorouracil, imiquimod, calcipotriol, antifungals, and physical therapies such as lasers [2]. There are currently no international guidelines, as no randomised controlled trials have been performed to evaluate these treatment modalities [7]. In a 2014 review of 55 cases of porokeratosis, surgical excision in seven cases led to no recurrence and was considered curative [8]. However, surgical removal is generally reserved for individuals with smaller and fewer lesions [7, 9].

Our case contributes several noteworthy observations to our current understanding of this specific subtype of porokeratosis. First, the patient's age aligns with the typical demographic for PP [2, 5, 10], whereas classical GP predominantly affects middle-aged men [2]. Interestingly, our patient was completely asymptomatic despite PP being commonly associated with pruritus [5]. This also emphasises the value of dermoscopy, as features observed in our patient (hyperkeratotic rim and brown globules) (Figure 1) were consistent with dermoscopic findings

in other reported cases [4]. The lack of response to topical corticosteroids observed in our case echoes the poor response of GP to various topical therapies seen in the literature. In our patient, a punch excision removed the lesion completely and proved to be both diagnostic and therapeutic. Importantly, our patient opted for complete excision despite the small theoretical risk of malignant transformation, highlighting the importance of shared decision-making and patient-centred care.

Author Contributions

Brandon Tan contributed to main writing, research and editing. Karen Cheung contributed to histopathology and article editing. Xin Liu contributed to research and editing. Alexis Daniel Lara Rivero contributed to overall supervision of writing, research and subject of the article. All authors have read and agreed to the published version of the manuscript.

Ethics Statement

All patients in this manuscript have given written informed consent for participation in the study and the use of their deidentified, anonymised, aggregated data and their case details (including photographs) for publication.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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