

Chapter 49

Hyperkeratotic Plaques on the Scalp and Outer Ears



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Introduction

An 84-year-old male patient presented with multiple asymptomatic hyperkeratotic plaques on the scalp, outer ears, nose and cheeks (Figs. 49.1 and 49.2). He had been working in building trade for more than 40 years. His medical history was remarkable for hypertensive cardiac disease.

On physical examination we observed multiple verrucous and hyperkeratotic plaques on sun-exposed areas. The hyperkeratosis was variable in thickness and morphology. An erosive lesion was noted on the left cheek. The surrounding skin was atrophic with telangiectasias and pigmentary changes. His Fitzpatrick phototype was II. A calculation of lifetime ultraviolet radiation (UVR) exposure and profession-related UVR-exposure was performed. The professions-related exposure was >40% of lifetime exposure.

Based on the case description and the photographs, what is your diagnosis?

Differential Diagnoses

1. Seborrheic keratoses.
2. Basal cell carcinomas.
3. Field cancerization.
4. Actinic porokeratosis.
5. Human papilloma virus-induced warty keratomas.

Diagnosis

Field cancerization.

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Fig. 49.1 (a) Multiple hyperkeratotic plaques on the scalp. (b) Multiple hyperkeratotic plaques on the scalp

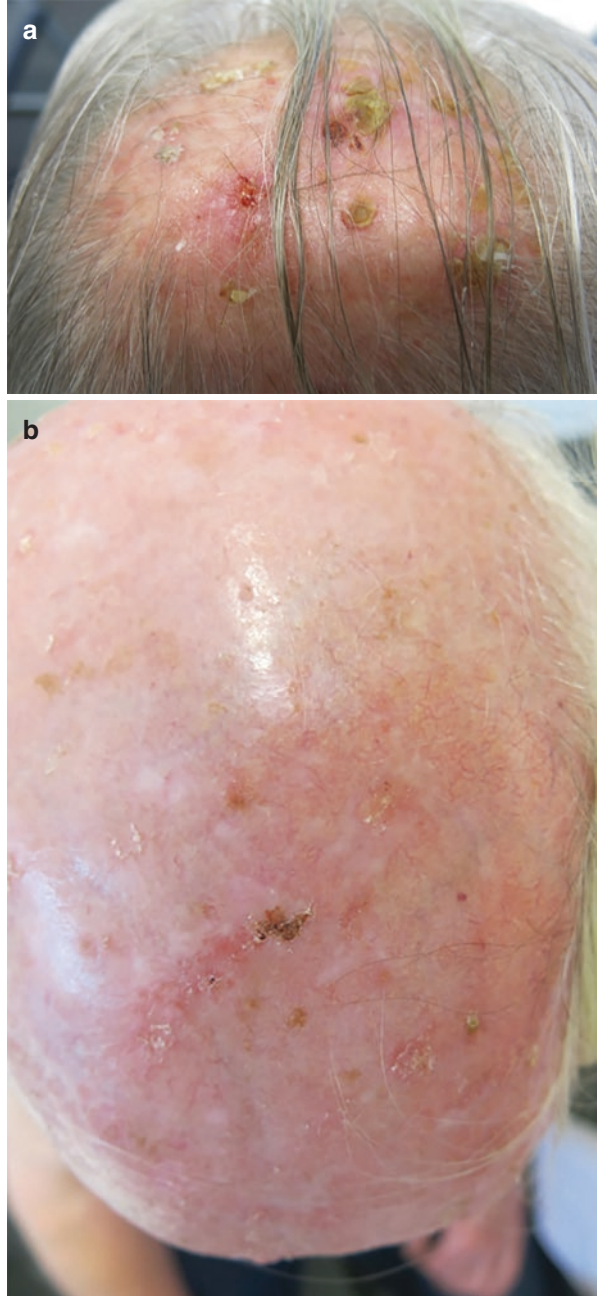


Fig. 49.2 Hyperkeratotic plaques and verruciform lesions on the outer ear



The ulcerated lesion on the cheek was surgically removed by delayed Mohs surgery. Histology showed features of SCC stage Ib which was completely removed. Defect closure was realized by skin advancement flap and two layered sutures.

For the field cancerization of the scalp with actinic keratoses Olsen grade I to II [1], photodynamic therapy was performed. The session started with roughing of the skin with a mono-filamentous fiber pad and disinfection. Thereafter, a gel containing 78 mg 5-aminolevulinic acid in a nanoemulsion was applied. The area was covered by aluminum foil for 3 hours. Irradiation was performed with a BF-RHODO LED® (Biofrontera) device emitting red light with a peak wavelength of 635 nm for 10 min (37 J/cm^2). During irradiation, the skin surface was treated by cooled air and ice-spray. After photodynamic therapy, we recommended skin care with a cream containing an extract of *Mahonia aquifolium* for at least two weeks [2]. In the presented patient, the occurrence of multiple actinic keratoses on UVR-exposed skin, outdoor profession and $\geq 40\%$ lifetime UVR-exposure by outdoor profession justify the recognition of occupational skin cancer [3].

Discussion

Actinic keratoses are common lesions of UVR-exposed skin in humans of Fitzpatrick type I or II. Their prevalence increases with age. In Central Europe, the prevalence is about 3% of the general population, while in Australia the prevalence increases from 7 to 74% with the age. A higher UVR-exposure leads to earlier development and more severe course of actinic keratoses [4].

Actinic keratosis is a potential precursor of squamous cell carcinoma. Therefore, treatment is recommended. Currently, the percentage of transformation from actinic keratosis to squamous cell carcinoma is unknown.

Single lesions can be removed by shaving, cryotherapy or ablative laser.

In case of field cancerization topical therapy with 3.75% or 5%, imiquimod 0.5% 5-fluorouracil with 10% salicylic acid, 5% 5-fluorouracil, 3% diclofenac in 2.5% hyaluronic acid is approved by the European Medical Association (EMA). Treatment time varies between two and 12 weeks.

Photodynamic therapy is approved for treatment of field cancerization. The following photosensitizers are available: methyl aminolaevulinic acid cream, 5-aminolaevulinic acid patch or nanoemulsified gel. Narrow-band irradiation was shown to be more effective than broad band light sources. Meta-analyses have shown that photodynamic therapy with narrow-band irradiation is more effective than all topical drugs except 5% 5-fluorouracil. PDT is also characterized by good cosmetic outcome [5]. The ulcerated lesion on the cheek was surgically removed by delayed Mohs surgery. Histology showed features of SCC stage Ib which was completely removed. Defect closure was realized by skin advancement flap and two layered sutures. For the field cancerization with actinic keratoses Olsen grade I to II on the scalp [1], PDT was performed. The session started with roughing of the skin with a mono-filamentous fiber pad and disinfection. Thereafter, a gel containing 78 mg 5-aminolevulinic acid in a nanoemulsion (Ameluz[®], Biofrontera AG, Leverkusen, Germany) was applied. The area was covered by aluminum foil for 3 h. Irradiation was performed with a BF-RHODO LED[®] (Biofrontera) device emitting red light with a peak wavelength of 635 nm for 10 min (37 J/cm²). During irradiation, the skin surface was treated by cooled air and ice-spray. After PDT, we recommended skin care with a cream containing an extract of *Mahonia aquifolium* for at least two weeks [2].

Key Points

- Actinic keratosis is caused by chronic ultraviolet irradiation.
- It is potential precursor of squamous cell carcinoma.

References

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