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Localised hypopigmentation:

clarification of a diagnostic conundrum

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INTRODUCTION

Various forms of skin hypopigmentation can occur spontaneously. When multiple forms of hypopigmentation occur simultaneously, the diagnoses may seem unclear. This article illustrates a patient who presented simultaneously with each of vitiligo, (idiopathic) guttate hypomelanosis (IGH), and a rarely noted hypopigmented variant of seborrhoeic keratosis. We outline distinguishing clinical features for clinicians to consider on encountering a patient with adult-acquired hypopigmentation. Subsequently, we present a useful approach to diagnosing common acquired forms of localised hypopigmentation seen in primary care.

CASE REPORT

A 61-year-old black female presented to her GP with white patches on her back (Figure 1a), and was diagnosed with vitiligo. Management included tacrolimus 0.1% ointment for daily use and referral to a dermatologist. She was also advised to avoid sun exposure to the affected areas. In the interim, the patient was exposed to ultraviolet radiation (UVR) while vacationing in Jamaica and noted improvement in the white patches. At her first dermatology visit, she displayed repigmenting patches of vitiligo with brown macules perifollicularly (Figure 1b). She queried whether new hypopigmented lesions were also vitiligo. Specifically, her back had light 'stuck on' papules and her arms and back had other 5-mm hypopigmented macules (Figure 1c and 1d). She queried why select back lesions improved after sun exposure despite being advised to avoid it. Her dermatologist explained that her initial back patches of vitiligo had repigmented due to UVR from sun and the use of tacrolimus ointment. In contrast, the newer dorsal arms and back lesions were two different benign hypopigmented conditions associated with ageing, neither of which respond to vitiligo treatments.

DISCUSSION

Due to the potential marked contrast between the affected skin and normal skin, hypopigmented skin conditions can result in cosmetic, and even psychological, challenges for patients, leading them to seek assessment. In rare cases, hypopigmentation can represent internal illness or even malignancy. Therefore, prompt, accurate diagnosis is preferable, and enables physicians to order appropriate investigations and timely treatment.

The patient's initial diagnosis was vitiligo (Figure 1b), an acquired polygenetic disorder with a predilection for acral sites, presenting as well-demarcated macules and patches that progress to complete depigmentation.[1] The prevalence of vitiligo is around 1% in Europe and the US, and more than 8% worldwide. An underlying genetic susceptibility to autoimmune diseases with aberrant encoding of the gene producing tyrosinase (an enzyme responsible for catalysing the rate-limiting steps in melanin biosynthesis) has been hypothesised as the aetiology of vitiligo.[1] Repigmentation occurred in this patient in a perifollicular pattern because repopulating melanocytes originate from the follicular bulge. Local vitiligo responds to treatment with topical corticosteroids and the topical calcineurin inhibitors (TCIs) tacrolimus or pimecrolimus. In this case, significant repigmentation of focal vitiligo was achieved with tacrolimus, likely due to a local immunosuppressive effect. More diffuse presentations may be treated with narrowband UVB phototherapy.

There are distinct clinical differences in the other benign forms of hypopigmentation that occurred in this patient. The seborrhoeic keratoses newly present on her back are a leading form of acquired skin change in adults.[2] Although they generally present in adulthood as a brown or black macule or papule, our patient's was a pale, hypopigmented papule with a variegated surface (Figure 1c). A clinical distinguishing feature of seborrhoeic keratoses is that they

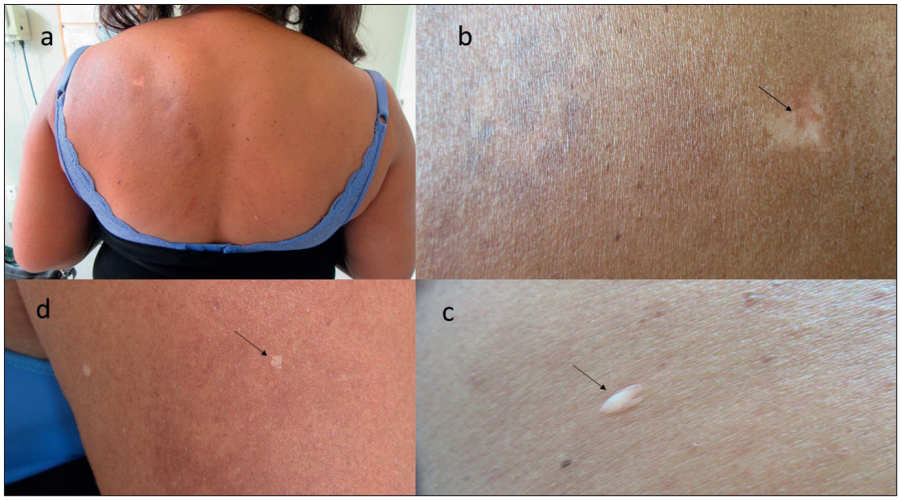

Figure 1. Patient's hypopigmentation:[a] vitiligo with repigmentation;[b] hypopigmented variant of seborrhoeic keratosis;[c] guttate hypomelanosis.[d]

Box 1. Diagnoses with hypopigmentation

Localised

- Face
  - — Pityriasis alba
  - — Vitiligo
- Limbs/torso
  - — Seborrhoeic keratoses
  - — Idiopathic guttate hypomelanosis

Diffuse

- Respondent to Rx
  - — Vitiligo
- Recalcitrant to Rx
  - — Sarcoidosis
  - — Mycosis fungoides

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exhibit a 'stuck on' appearance: the edge is palpable compared with the background skin, creating the illusion that the lesion was physically applied to the skin. Despite their capacity for growth, these lesions do not require medical treatment.[2]

Another common benign condition that presents with hypopigmentation is IGH, which affects approximately 50–80% of individuals aged >40 years. Its prevalence increases with age. IGH is characterised by multiple, discrete, circumscribed, porcelain-white macules that tend to favour sun-exposed sites,[3] but have been known to occur on covered areas as well. It was newly present on this patient's arms (Figure 1d). The precise pathogenesis of IGH is unclear, but cumulative sun exposure, genetic factors, and autoimmunity have all been suggested as causative factors.[3] Treatment is not required.

Other common hypopigmented conditions that may occur include pityriasis alba and pityriasis versicolor. The former presents as hypopigmented, non-scaly, round to oval patches with ill-defined border on the face, neck, and trunk.[4] It has a predilection for children or adolescents with brown or black skin. Lesions are more apparent during the spring and summer months contrasted against tanned skin. Medical treatment is optional using low-potency topical steroids or TCIs.[4] It tends to improve in autumn and winter, and generally resolves with puberty. Pityriasis versicolor is a common superficial yeast located in skin furrows of the neck, upper arms, and trunk. It is caused by *Malassezia spp.* yeasts and can be treated with topical -azole agents.[5] In its pathogenic mycelial form, the yeast causes mild inflammation of the skin. Its initial presentation of hyperpigmented scaling macules transforms into numerous hypopigmented macules due to *Malassezia*-produced azelaic acid that impairs melanocyte function.

Finally, two uncommon, yet notable, disorders that can present with hypopigmentation are sarcoidosis and mycosis fungoides (MF). Despite their rarity, they may be included in the differential diagnosis for the patient who has progressing hypopigmentation despite use of therapy (Box 1). Sarcoidal hypopigmented macules may occur over granulomas in the dermis or subcutaneous tissue.[6] Hypopigmented MF (HMF) generally occurs in dark-skinned young adults and is a cutaneous T-cell lymphoma with CD8+ phenotype. HMF is usually observed on the trunk and proximal extremities, especially the buttocks and pelvic girdle, and can present as scaly or non-scaly hypopigmented round patches.[6] In cases of suspected sarcoidosis or MF, it is important to confirm the diagnosis with a skin biopsy and consider the involvement of other physician subspecialties as deemed appropriate to help rule out systemic or lymphatic involvement, respectively.

CONCLUSION

This case highlights the need to consider different causes of acquired hypopigmentation in adults, particularly in brown- or black-skinned patients. Recognition of benign variants of hypopigmentation, such as seborrhoeic keratoses and IGH, can reassure patients and avoid inappropriate treatment. More broadly, the development of a short differential diagnosis for skin hypopigmentation allows primary care physicians to consider a range of plausible conditions and diagnose not only common benign entities but also less common conditions requiring further investigation.

Provenance

Freely submitted; externally peer reviewed.

Patient consent

The patient gave consent for publication of this case report and images.

Competing interests

The authors have declared no competing interests.

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