



Basal Cell Carcinoma Associated with Vitiligo: A Case Report and Literature Review

Dr. D. Jaadi^{1*}, Dr. H. Sqalli Houssaini¹, Dr. I. Boukhari¹, Dr. I. Moutahir¹, Dr. Z. Berjaou¹, S. Mazouz¹, J. Hafidi¹, N. Gharib¹, A. Abbassi¹

¹Department of Plastic and Reconstructive Surgery, Hand Surgery Unit, CHU Ibn Sina, Rabat, Morocco

*Corresponding author: Dr. D. Jaadi

Department of Plastic and Reconstructive Surgery, Hand Surgery Unit, CHU Ibn Sina, Rabat, Morocco

Article History

Received: 17-06-2025

Accepted: 24-08-2025

Published: 01-09-2025



Abstract:

Basal cell carcinoma (BCC) is the most common form of skin cancer, whereas vitiligo is a benign depigmenting disorder often regarded as protective against skin malignancies. Nevertheless, rare instances of BCC arising in vitiliginous skin have been documented. We present the case of a 74-year-old female patient with infiltrative BCC of the scalp arising within a vitiligo plaque, managed surgically at our department. This report also provides a comprehensive review of the literature to explore this uncommon association.

Keywords: Basal cell carcinoma, vitiligo, skin cancer, depigmentation, scalp.

Case Report

Copyright © 2025 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Basal cell carcinoma (BCC) accounts for over 75% of non-melanoma skin cancers. It is characterized by slow growth, local invasiveness, and a low risk of metastasis, resulting in a generally favorable prognosis when diagnosed early [1, 2]. Major risk factors include chronic ultraviolet (UV) exposure, fair skin phototype, immunosuppression, and certain genetic syndromes such as Gorlin-Goltz syndrome [3].

Vitiligo is an acquired autoimmune dermatosis marked by the progressive destruction of epidermal melanocytes, leading to depigmented macules. Some early studies suggested a potential protective effect of vitiligo against skin cancer, particularly BCC and squamous cell carcinoma (SCC), possibly due to enhanced immune surveillance mechanisms [4].

However, this hypothesis remains controversial. Recent data suggest the association may be more complex and context-dependent [5-7].

Case Report

A 74-year-old female patient, with a medical history of mastectomy for breast carcinoma (1980) and hysterectomy for uterine fibroid, presented with a progressively enlarging cutaneous lesion on the scalp. She had generalized vitiligo for over two decades without specific treatment.

Clinical examination revealed a 4 cm ulcerated and nodular lesion in the right occipital region, localized within a depigmented vitiligo plaque. The rest of the skin exhibited extensive vitiligo without suspicious lesions.

Cranial CT imaging demonstrated dermal and subdermal thickening in the occipital area, without bone invasion or metastasis. Surgical excision was performed in a single session, including periosteum resection with a 1 cm safety margin. Reconstruction was achieved using an occipital transposition flap and split-thickness skin graft over the donor site.

Histopathology confirmed an infiltrative, well-differentiated basal cell carcinoma, with clear

resection margins and no perineural or vascular invasion.

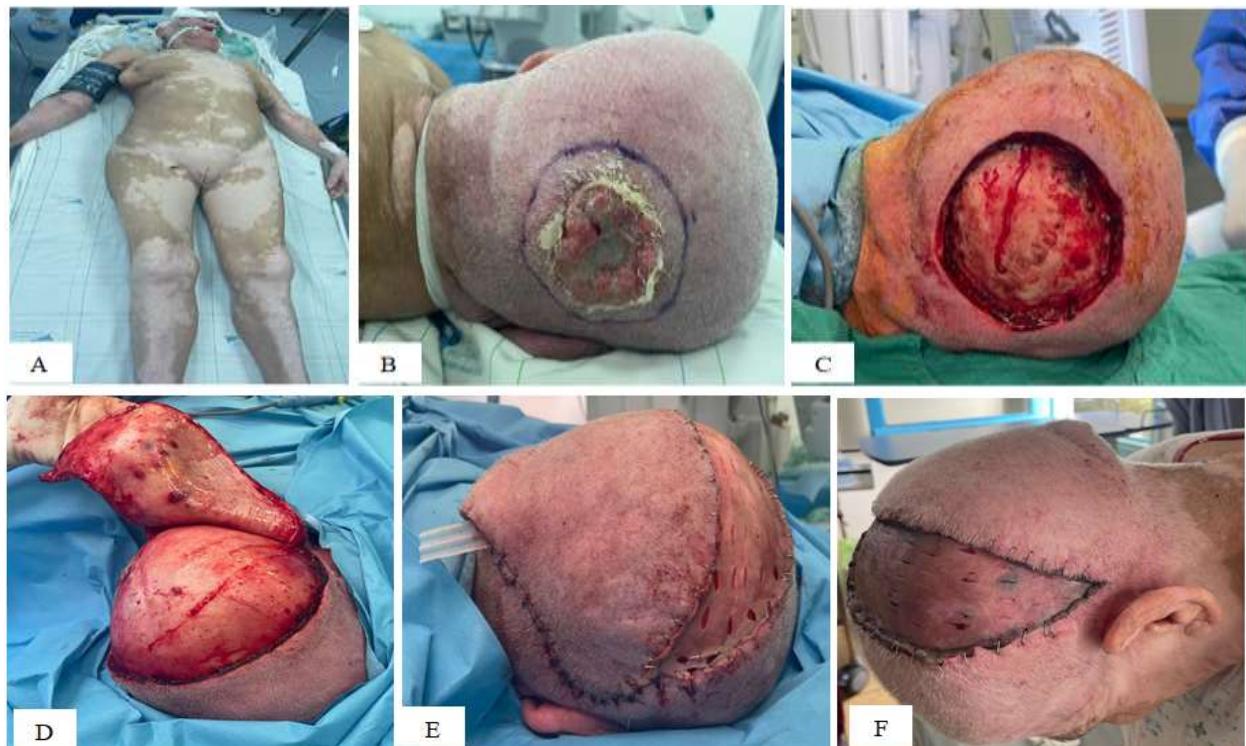


Fig 1: A) Patient with a BCC of the scalp plus vitiligo; B) BCC of the scalp with excision margin; C) Scalp skin graft after excision; D) Scalp flap raised; E) Flap placement over the skin graft + skin graft; F) Final appearance at 7 days

DISCUSSION

The relationship between vitiligo and the risk of skin cancer has long intrigued dermatologists and immunologists. Traditionally, it was believed that the autoimmune destruction of melanocytes and the associated upregulation of immune surveillance may confer protection against skin malignancies [4, 6]. Studies by Ongenae *et al.*, [4] and Gauthier *et al.*, [7] proposed that vitiligo patients exhibit increased anti-tumor immunity due to elevated p53 expression and heightened T-cell activity. However, more recent evidence suggests this assumption may not hold in all clinical scenarios.

Contrary to the protective theory, newer studies have reported cases of non-melanoma skin cancers developing in vitiliginous skin [5, 6, 8]. Mahajan *et al.*, [6] reviewed cases of BCC arising within vitiligo plaques and suggested that local immunologic alterations might not suffice to prevent tumorigenesis, especially in older individuals with long-term sun exposure.

One plausible mechanism includes the complete absence of melanin in vitiliginous patches, which eliminates the natural UV filtering capacity of the skin. Wu *et al.*, [10] emphasized that such areas are more vulnerable to ultraviolet-induced DNA damage. This damage, when combined with genomic instability, as shown in depigmented keratinocytes by Vargas-Luna *et al.*, [12], may drive oncogenic transformations.

Additionally, Dwivedi *et al.*, [8] and Bertolotti *et al.*, [9] highlighted the dual role of chronic inflammation in vitiligo: while it may trigger immune-mediated tumor clearance, it can also induce prolonged tissue stress, DNA repair impairment, and oxidative damage.

Importantly, none of the few published cases of BCC on vitiliginous skin involved prior phototherapy, which rules out iatrogenic UV exposure as a contributing factor [5, 6, 13].

Phototherapy, particularly narrowband UVB (nb-UVB), has not been conclusively linked to an increased risk of non-melanoma skin cancer in vitiligo patients. Hearn *et al.*, [14] conducted a systematic review demonstrating the safety profile of nb-UVB, reinforcing that intrinsic cutaneous alterations rather than external therapies may underlie tumor development.

In our case, the patient had generalized vitiligo and no history of phototherapy. The lesion was extensive, infiltrative, and located in a chronically sun-exposed area. Despite the aggressive clinical appearance, the patient had no metastatic spread, supporting previous findings of BCC's low metastatic potential [2].

This case adds to the limited literature on BCC in vitiligo skin and serves as a clinical reminder that:

- Vitiligo does not provide absolute protection against skin cancer.
- Depigmented areas, especially in elderly patients, require vigilant monitoring.
- A biopsy should be performed promptly for any evolving lesion, even if non-pigmented or appearing benign.

Future directions include large cohort studies to quantify the actual risk of BCC and SCC in vitiligo populations, incorporating genetic and environmental factors. Investigating molecular markers such as TP53 mutations, DNA methylation profiles, and cytokine expression in vitiliginous skin may also yield novel insights.

CONCLUSION

This case illustrates a rare but significant occurrence of infiltrative basal cell carcinoma arising in a vitiligo plaque. Although vitiligo has traditionally been considered protective against skin malignancies, emerging data suggest this is not universally the case.

Dermatologists and oncologists should maintain a high index of suspicion and promote vigilant, long-term surveillance, particularly in elderly vitiligo patients with chronic UV exposure.

REFERENCES

1. Madan V, Lear JT, Szeimies RM. Non-melanoma skin cancer. *Lancet*. 2010;375(9715):673–685.
2. Nehal KS, Bichakjian CK. Update on keratinocyte carcinomas. *N Engl J Med*. 2018;379(4):363–374.
3. Marzuka AG, Book SE. Basal cell carcinoma: pathogenesis, epidemiology, clinical features, diagnosis, histopathology, and management. *Yale J Biol Med*. 2015;88(2):167–179.
4. Ongena K, *et al.*, Increased prevalence of autoimmune diseases in vitiligo patients. *Acta Derm Venereol*. 2003;83(3):207–210.
5. Rustemeyer J, *et al.*, Basal cell carcinoma developing in a vitiligo lesion: a rare coincidence. *J Eur Acad Dermatol Venereol*. 2011;25(1):107–108.
6. Mahajan VK, *et al.*, Basal cell carcinoma in vitiliginous skin: coincidence or correlation? *Indian J Dermatol*. 2018;63(1):84–86.
7. Gauthier Y, *et al.*, p53 gene expression and mutation analysis in vitiligo. *Pigment Cell Res*. 2001;14(6):423–430.
8. Dwivedi M, *et al.*, Cytokine profile in stable and active vitiligo patients and its correlation with disease activity. *Clin Chim Acta*. 2011;412(23–24):191–194.
9. Bertolotti A, *et al.*, Activation of innate immunity in vitiligo. *Pigment Cell Melanoma Res*. 2014;27(1):12–20.
10. Wu CS, *et al.*, UV damage in vitiliginous skin: an underestimated oncogenic risk? *Exp Dermatol*. 2024;33(1):32–39.
11. Lee MJ, *et al.*, Incidence of Nonmelanoma Skin Cancer in Patients With Vitiligo: A Multicenter Korean Cohort Study. *J Dermatol Treat*. 2023;34(2):301–308.
12. Vargas-Luna J, *et al.*, Genomic instability in depigmented keratinocytes: new insights from single-cell sequencing. *J Transl Med*. 2024;22(1):55.
13. Huang X, *et al.*, Case report: Infiltrative BCC developing in sun-exposed vitiligo patch. *Clin Exp Dermatol*. 2025;50(1):89–92.
14. Hearn R, *et al.*, Risk of non-melanoma skin cancer with PUVA and NB-UVB: a systematic review. *Photodermatol Photomed*. 2020;36(4):246–254.
15. Krüger, C., & Schallreuter, K. U. (2012). A review of the worldwide prevalence of vitiligo in children/adolescents and adults. *International journal of dermatology*, 51(10), 1206-1212.