

# A Clinical Image of Basal Cell Carcinoma in Universal Vitiligo

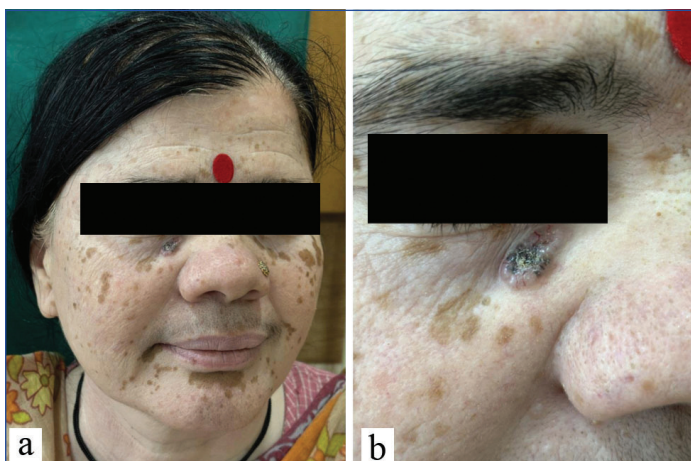
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A 53-year-old woman presented to dermatology OPD with an abnormal growth under her right eye since one year. The patient first developed white patches on the fingertips at the age of 13. Similar lesions appeared over her toes and perioral region and then progressed to involve approximately 90% of the body surface area over the next 15 years, which was consistent with universal vitiligo. She did not undergo any treatment for vitiligo during this time. She denied prior use of phototherapy, topical corticosteroids, calcineurin inhibitors and systemic immunosuppressive agents. The disease remained stable for over a decade prior to the onset of the current lesion.

Physical examination was notable for a solitary nodule with an irregular surface, translucent rolled-out border, pigmented areas, telangiectasias and central ulceration overlying vitiliginous skin [Table/Fig-1]. Dermoscopy revealed blue ovoid nests with a white veil, arborising vessels, short fine telangiectasias, brown-grey globules and central focal ulceration with scaling. Patches of normal skin with regular reticulate pigmentary pattern could be appreciated [Table/Fig-2]. A diagnosis of nodular Basal Cell Carcinoma (BCC) was made based on the characteristic clinical and dermoscopic findings.



**[Table/Fig-1]:** Clinical image showing a solitary papule with rolled-out borders, central ulceration and telangiectasias located on vitiliginous skin.

The patient refused all invasive procedures despite being repeatedly counselled for biopsy to confirm the diagnosis. Therefore, a diagnosis had to be made based on the characteristic dermoscopic features. The decision was supported by substantial evidence from recent literature, which demonstrated a high diagnostic accuracy of dermoscopy for BCC. Dermoscopy showed a sensitivity of 92.2% and specificity of 96.0% for BCC diagnosis, with especially high accuracy in facial and pigmented lesions, in a study of 934 patients [1]. Similarly, a systematic review and meta-analysis evaluating over 2,000 BCCs reported sensitivity and specificity of 91.2% and 95.0%, respectively, for dermoscopy-based diagnosis [2]. These findings support the reliability of dermoscopy as a non-invasive diagnostic tool when histopathological confirmation is not feasible.



**[Table/Fig-2]:** Dermoscopy of the lesion showing blue ovoid nests, arborising vessels, brown-grey globules, white veil and central ulceration.

The patient was counselled regarding treatment options, including Mohs micrographic surgery; however, she declined all surgical interventions. As an alternative, topical imiquimod 5% cream was initiated, to be applied as a thin layer on the lesion on alternate days. The patient returned for follow-up after three weeks, at which time partial regression of the lesion was observed, with crusting at the centre and reduced telangiectasia. However, she was subsequently lost to follow-up.

The absence of melanin in vitiligo-affected skin has traditionally raised concerns regarding heightened susceptibility to ultraviolet-induced skin cancers. However, a large-scale meta-analysis [3] revealed that patients with vitiligo have a significantly lower risk of developing keratinocyte cancers, including BCC, compared to the general population. This finding suggests that immunological and molecular factors may offer cancer protection in vitiligo skin.

One such mechanism involves the dysregulation of microRNA (miRNA) networks. miR-31-5p, miR-31-3p, and miR-194-3p are three upregulated miRNAs identified in lesional vitiligo epidermis through transcriptomic and miRnome analysis [4]. These miRNAs target and downregulate key Oncogenic Transcription Factors (OTFs) implicated in BCC pathogenesis, such as FOXC1, AR, SP1, YY1, GLI2, TP53, and RARA, which disrupts tumourigenic signalling cascades. This interaction may help explain the lower prevalence of non-melanoma skin cancers seen in vitiligo by contributing to an anti-skin cancer molecular signature.

All of these results point to the possibility that vitiligo skin, even in the absence of melanin, may have a downregulated oncogenic transcriptome, improved immune surveillance and changed miRNA regulation-all of which work together to lower the risk of BCC.

The genetic and autoimmune profile of vitiligo patients protective against skin cancers is known as the “white armour” of vitiligo [5]. However, Non Melanoma Skin Cancers (NMSCs), including BCCs, are infrequently reported in patients with vitiligo. Even rarer are BCCs developing on vitiliginous skin. A case of sclerodermiform BCC on a vitiliginous patch over the cheek of a 33-year-old female, with minimal sun exposure and absence of conventional risk factors, was reported, highlighting the possibility of malignancy even in younger individuals with limited sun exposure [6]. In another study, BCC occurring within a vitiliginous area of the nasolabial fold was observed in an elderly male with generalised vitiligo [7]. Additionally, a separate report described the development of BCC in a patient with vitiligo, although clinical details were sparse [8].

This may denote a chink in the white armour, suggesting the need for increased vigilance towards carcinogenesis and use of photoprotective measures that are often overlooked when treating patients with vitiligo.

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