Out of numerous causes of non-healing leg ulcers, ulcerative sarcoidosis is a very rare cause. In addition, in India, where tuberculosis is endemic, a diagnosis of ulcerative sarcoidosis can be easily missed unless specifically looked for. Histopathological examination is necessary in all cases of non-healing leg ulcers to reach at an accurate diagnosis.

Non-caseating, naked epithelioid granulomas are the most common histopathological findings seen in biopsies from ulcerative lesions, as seen in our case. Other histopathological findings described in the literature are hyaline degeneration and orceinophilic fibres in the granulomas, mixed inflammatory cell infiltrates with lymphocytes and neutrophils or histiocytes, suppuration with eosinophils and neutrophils in areas of necrosis, solitary sarcoidal granuloma, granulomatous vasculitis, necrotizing granulomas and periarteritis.[4]

Systemic corticosteroids and methotrexate were found to be effective in treating ulcerative lesions in sarcoidosis, and adalimumab was reported to improve ulcerative lesions that did not respond to corticosteroids.[5] Our patient responded well to prednisolone and methotrexate therapy, and the skin lesion completely healed in 8 weeks.

In conclusion, we report an exceptional case of ulcerative sarcoidosis presenting as a non-healing leg ulcer as the sole cutaneous presentation of chronic sarcoidosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Collision of basal cell carcinoma and atypical fibroxanthoma

Dear Editor,

A collision tumor is composed of two or more different cellular populations within the same lesion. Although this phenomenon is uncommon, a wide variety of either benign or malignant collisions have been described. We report a collision of a basal cell carcinoma (BCC) and an atypical fibroxanthoma (AFX) in an octogenarian woman.

BCC is the most common malignant tumour in humans while AFX represents a rarer neoplasm.[1,2] Occurrence of both these neoplasms at one site was first reported by Alves et al. in 2010.[3] Since then, only two other cases have been reported.[4,5] In this paper, we describe the clinical presentation, dermoscopy as well as pathological findings. We also discuss the different theories proposed in collision development.

An 84-year-old woman presented to our dermatology department for a slow growing tumor located at her temple, with episodes of spontaneous bleeding. Close inspection revealed a 3-cm diameter centrally ulcerated tumor [Figure 1a]. Polarized light dermoscopy demonstrated an amelanotic pinkish tumour with white structureless areas, irregular vessels, and a central serohaemorrhagic crust. We were able to identify a unique area with a large blue-grey ovoid nest [Figure 1b].

A wide margin excision was performed and sent for pathological study. Histological section revealed a dermal

How to cite this article: Perandones González H, Valladares Narganes LM, González Morán MA, Rodríguez prieto MÁ. Collision of basal cell carcinoma and atypical fibroxanthoma. Indian J Dermatol Venereol Leprol. 2024;90:500-3. doi: 10.25259/IJDVL_220_2021

Received: March, 2021 **Accepted:** May, 2023 **EPub Ahead of Print:** June, 2023 **Published:** June, 2024

DOI: 10.25259/IJDVL_220_2021 **PMID:** 37436024

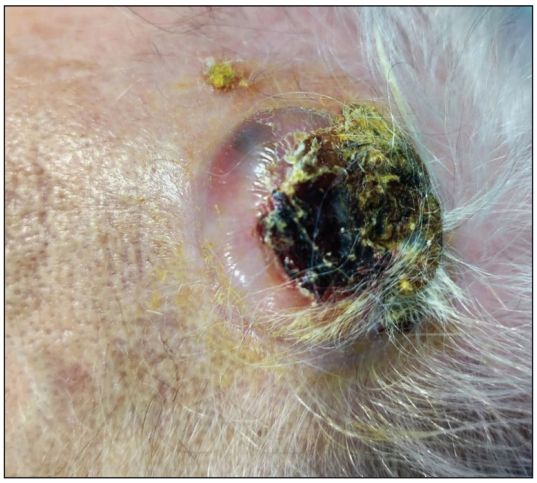

Figure 1a: A pink tumor with a central serohemorrhagic crust at the temple.

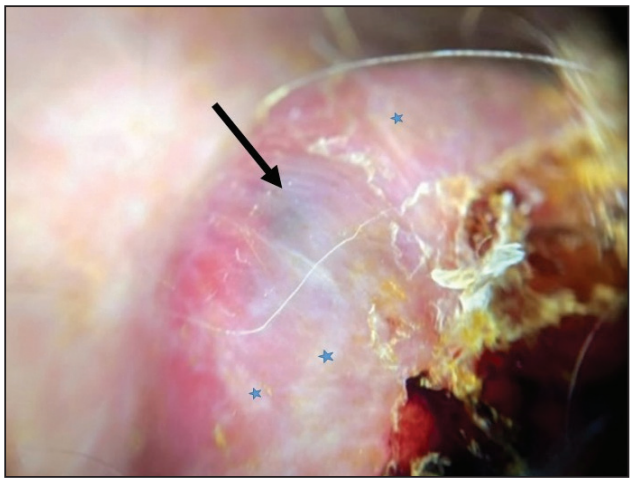

Figure 1b: Large blue-grey ovoid nest (black arrow) and structureless white areas (blue asterisk) on dermoscopy (Dermlite DL200 polarised).

tumour without involvement of epidermis and subcutaneous tissue which was composed of spindle-shaped cells with highly pleomorphic nucleus and abundant mitosis. Occasional giant multinucleated cells were also noted. We did not find necrosis. Another section revealed interconnected tumor islands budding from epidermis. These aggregates were composed of basaloid cells with peripheral palisading of the nuclei and a retraction artifact from a mucinous stroma [Figures 2a–2d]. These classic superficial BCC islands were surrounded by the neoplastic spindle cell proliferation in the dermis.

Immunohistochemical stain results were different in both neoplasms [Figure 3]. Vimentin, CD10, CD68, and CD99 were highly positive in the spindle cell tumor and negative in the BCC. Desmin, EMA, S100, and Melan A were negative in both tumors. BCC was positive for Ber EP4 and pancytokeratins AE1-3. Ki67 was highly positive in both neoplasms [Figures 3a–3d]. The final diagnosis was collision tumor of BCC and AFX. After complete excision, no recurrence was observed at one-year follow up.

Collision tumors are composed of two or more different neoplastic proliferations coexisting within the same lesion.[6]

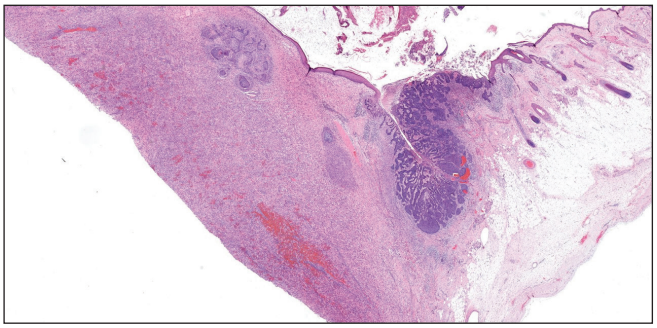

Figure 2a: Solar elastosis and two different proliferations present within the same lesion (Haematoxylin & Eosin, 10x)

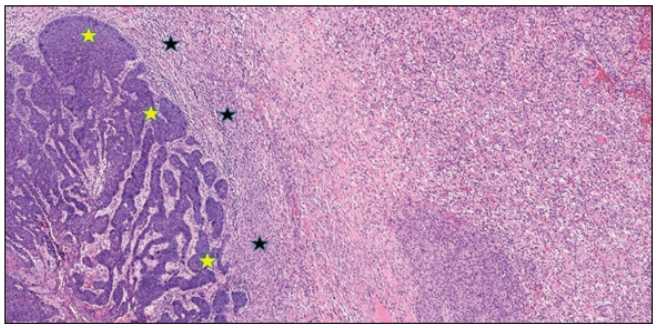

Figure 2b: BCC island within palisading artifact (yellow asterisk) in close contact with a spindle cell tumor in the dermis (black asterisk) (Haematoxylin & Eosin, 40x)

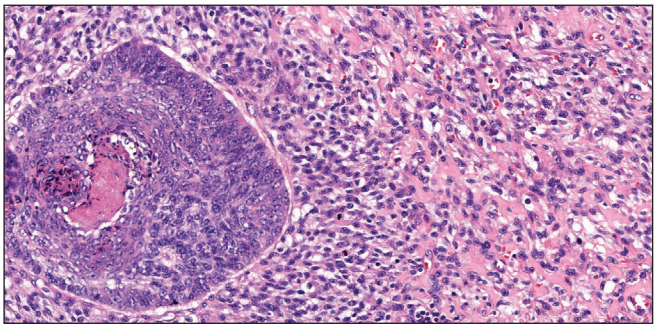

Figure 2c: Details of a combined tumor of a BCC and an AFX (Haematoxylin & Eosin, 200x)

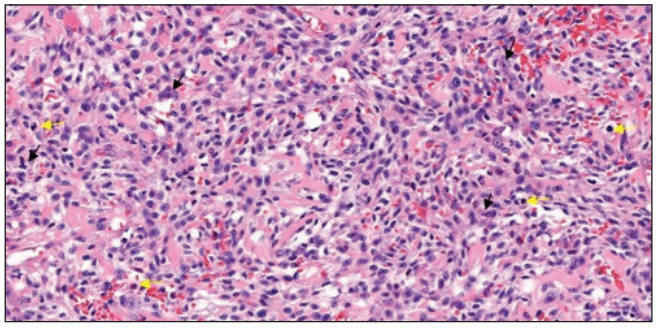

Figure 2d: Dermal proliferation composed of atypical spindle cells with pleomorphic nucleus (black arrows) and frequent atypical mitosis (yellow arrows) (Haematoxylin & Eosin, 200x)

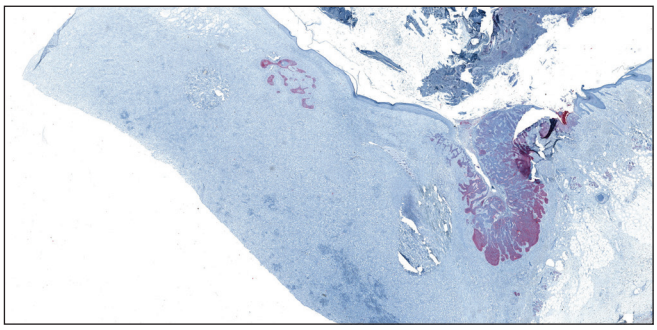

Figure 3a: Immunohistochemistry (10x). BCC showing positivity for BerEp4.

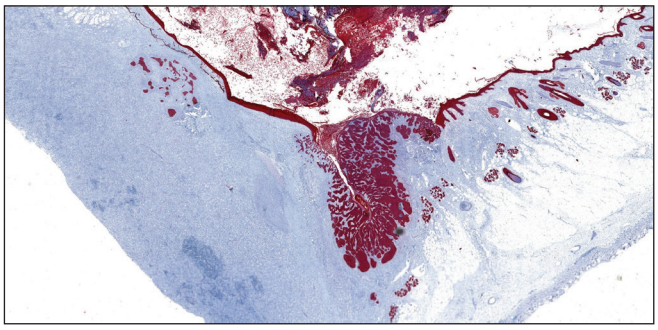

Figure 3b: Immunohistochemistry (10x). Strong positivity for pancitokeratins AE1-AE3 in BCC.

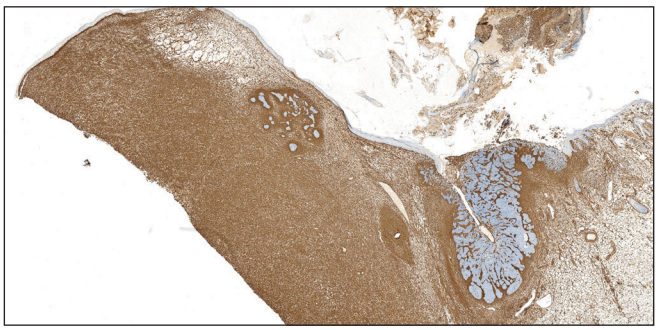

Figure 3c: Immunohistochemistry (10x). AFX showing strong and diffuse positivity to vimentin.

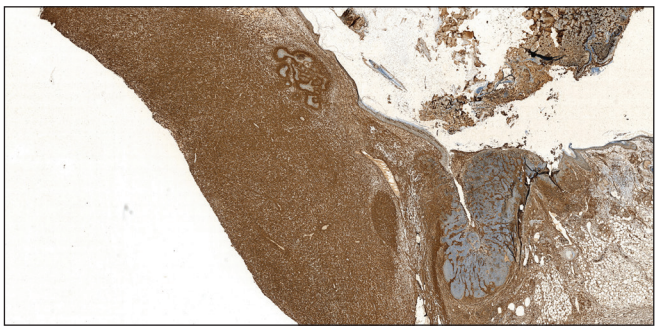

Figure 3d: Immunohistochemistry (10x). CD10 strongly positive in AFX and negative in BCC.

They represent an infrequent finding in dermatopathology (0.17% of 40,000 biopsies in a retrospective study) and their diagnosis can be challenging.[7] Recently, coexisting neoplasms have been classified based on their pathogenesis under the term "multiple skin neoplasm at one site."[8] This concept englobes the categories collision, colonization, combined and biphenotypic tumor defined by Satter *et al.*[9] Although this classification was designed for epithelial and melanocytic neoplasms, it can be useful in our case. Collision tumors have well-defined borders, unlike in our case where the border of both neoplasms is not sharp. Biphenotypic tumors arise from a common stem cell precursor differentiating into different populations. BCC develops from interfollicular epidermal stem cells whereas AFX is mainly thought to arise from mesenchymal lineage although an epithelial origin has also been suggested.[10,11] Colonization was defined as a permeation of a melanoma in situ into a BCC. BCC is a slow growing tumor, and it would be unlikely that is able to colonize the underlying faster growing and more aggressive AFX. Therefore, our case could fit into the combined tumor category, where the two populations are intertwined.[6,9]

AFX is a fibrohistiocytic tumor developing in highly sun-exposed areas and clinically presenting as a rapid growing neoplasia with the possibility of ulceration and bleeding.[14] Note that all the previously collision cases described presented ulceration, including ours.[3–5] AFX is not usually suspected on clinical presentation and histologic diagnosis is made by exclusion of other proliferations based on immunohistochemistry.[14] In our patient we could identify a blue-grey ovoid nest that histologically correlates with the dermal BCC tumor nests.[16]

In this report, we present the clinical, dermoscopy, and pathological features of a collision of BCC and AFX. It is interesting to describe more of these rarities to extract conclusions about their pathogenesis and different characteristics.

Declaration of patient consent

Patient's consent not required as the patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Squamoid eccrine ductal carcinoma: A frequently misdiagnosed entity

Dear Editor,

Eccrine carcinomas comprise around 0.01% of all tumours.[1] Squamoid eccrine ductal carcinoma (SEDC) is a rare tumour first described by Wong *et al*. in 1997, representing a lesion showing eccrine ductal differentiation along with a prominent squamous component.[2] It usually involves the head and neck area and only few cases have been reported in the English literature, including a series of 30 patients by van der Horst *et al*.[3] Accurate diagnosis is crucial given its aggressive nature, along with increased risk of local recurrence and metastasis.

An 89-year-old male presented to VA Medical Center, Pittsburgh with a non-healing lesion of unknown duration on right posterior shoulder. Clinical examination revealed a 2.0 × 1.0 cm ulcerated pink plaque with violaceous border [Figure 1]. Histopathology revealed epidermal ulceration with proliferation of infiltrating glandular structures composed of markedly pleomorphic squamoid cells containing intraluminal secretions [Figures 2a and 2b]. Abundant mitoses were noted, along with tumour cell necrosis and focal

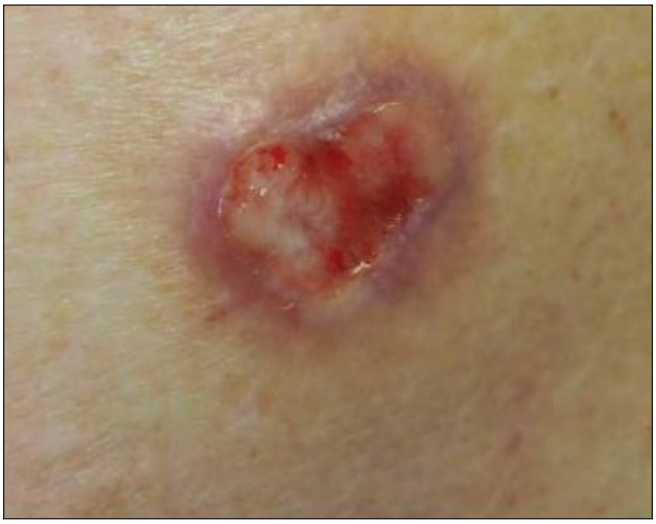

Figure 1: Ulcerated pink plaque (2.0 × 1.0 cm) with violaceous border on the right posterior shoulder.

How to cite this article: Srivastava P, Dando E, Doeden K. Squamoid eccrine ductal carcinoma: A frequently misdiagnosed entity. Indian J Dermatol Venereol Leprol. 2024;90:503-5. doi: 10.25259/IJDVL_310_2022

Received: March, 2022 **Accepted:** May, 2023 **EPub Ahead of Print:** June, 2023 **Published:** June, 2024

DOI: 10.25259/IJDVL_310_2022 **PMID:** 37436011