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Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Marjorin ulcer (squamous cell carcinoma) in a temporal artery biopsy wound

It is well recognized that postoperative and traumatic wounds and scars are susceptible to malignant degeneration, most commonly to squamous cell carcinoma (SCC).[1] This phenomenon can occur months to decades after the inciting trauma.[1] We describe a case arising from a temporal artery biopsy (TAB) site, demonstrating the importance of considering skin malignancy in any nonhealing incision. Informed consent for publication of this patient's case was obtained, adhering to the Declaration of Helsinki principles.

An 82-year-old female presented with acute painless vision loss OS and was diagnosed with a central retinal artery occlusion. Erythrocyte sedimentation rate, C-reactive protein, and platelets showed significant elevation. The patient was 1 week postoperative from hip surgery, interfering with interpretation of the inflammatory markers for possible giant cell arteritis (GCA). The patient had no other symptoms or signs of GCA. She was started on 60 mg oral prednisone (1 mg/kg) until we had results from the TAB performed a few days later. A full vascular work-up also was initiated. Her medical history was significant for mild chronic obstructive pulmonary disease, for which she used puffers occasionally. The patient had no history of diabetes, immunosuppression, or skin cancer or other cancers and was not taking any other medications.

A 1.5 cm TAB specimen was obtained. The surgeon's report did not mention visible skin changes or textural anomalies warranting concern for underlying malignancy at the biopsy site. The results were negative for vasculitis. The neuro-ophthalmologist quickly tapered the patient off prednisone in the following 3 weeks and followed with repeat bloodwork to ensure her inflammatory markers were trending down. Five months after TAB, the previously well-healed biopsy site started to gape open with crusting and erythema (Fig. 1A). Cephalexin was started orally for 1 week, followed by punch biopsies, wound debridement, and dehiscence repair in the operating room. Pathology reported the biopsies as acute and chronic inflammation with fibrosis and increased vascularity consistent with granulation tissue.

In the first month after dehiscence repair, the site seemed to heal well. However, 2 months later the patient presented again with a dehisced wound, tender and red, this time with prominent green mucopurulent discharge (Fig. 1B) that grew *Candida parapsilosis* on culture. The infectious disease team became involved and ordered a computed tomography scan to assess the underlying bone, which showed no osteomyelitis. The team arranged for wound care consisting of 1 month of ceftriaxone and metronidazole and 2 weeks of fluconazole. Despite initial reduction of the discharge, the wound continued to flare with persisting erythema. The team then suggested that the ophthalmology department reevaluate the patient for active GCA, knowing that active vasculitis can impair wound healing. A repeat assessment

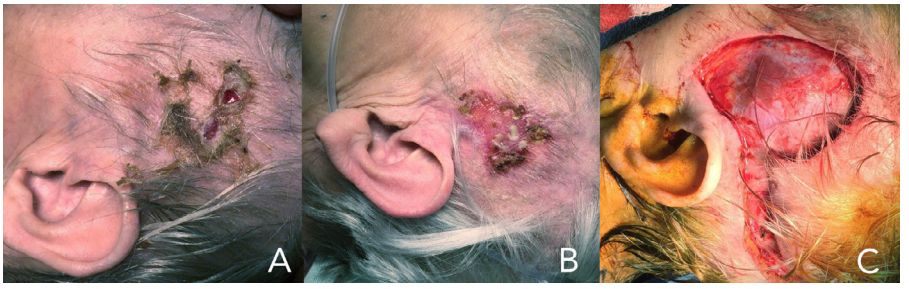

Fig. 1—(A) Gaping nonhealing wound 5 months after temporal artery biopsy; (B) 2 months after wound debridement demonstrating mucopurulent discharge cultured as *Candida parapsilosis*; (C) reconstructive flap used by plastic surgery team.

and noncontributory inflammatory markers did not substantiate this line of thinking.

Now, 1 year after the original negative TAB, the decision was made to rebiopsy the site of the nonhealing incision. This time the biopsy showed SCC in situ. The patient was referred to the plastic surgery department for reconstruction of the left temple parietal area with a large rotation flap (Fig. 1C). There was no invasive SCC in the final specimen after reconstruction.

Malignant degeneration of wounds and scars is a well-accepted phenomenon, even though the pathogenesis remains unclear. The term *marjorin ulcer* or *scar carcinoma* is often used to refer to malignancy arising from chronic wounds or scars.[1] The most common malignancy to arise from a scar is SCC, although other malignancies such as basal cell carcinoma, melanoma, and sarcoma have been reported less frequently.[2] The scar-related SCC are known to have a more invasive course and a greater tendency to metastasize.[3] Therefore, they are usually treated more aggressively (i.e., wider margins, consideration of adjuvant therapies),[3] as in our patient.

Most reported cases of scar carcinomas are from thermal burns and are thought to be related to poor vascularization of scar tissue [4] Although there was no burn wound involved in our case, we considered the possibility that the combination of intraoperative cautery and a decreased blood supply in the area of the excised temporal artery could have simulated the same predisposing milieu.

The majority of scar carcinomas are chronic with a mean latency period of 32 years.[2] Acute cases are defined as arising within 1 year.[5] In the eyelids, Pratt et al. showed that the majority of eyelid burn scar carcinomas were more commonly basal cell carcinomas, with a latency period within 2 years.[5] In our patient, the suspicion of malignancy manifested just 5 months after TAB and would be classified as acute. Notably, some authors are skeptical that acute scar carcinomas are a true phenomenon because it is hard to conceptualize malignant transformation happening in such a short time frame. An alternative narrative to explain the seemingly acute onset of malignancy in a scar is that the injured state accelerated an already preexisting process of carcinogenesis.[1] In our patient, no note was made by the TAB surgeon of a suspicious lesion at the site of the biopsy, and the procedural approach for TABs at our centre emphasizes avoiding "suspicious" skin changes for the selected site of incision. Yet, without a preoperative photograph, we cannot rule out the possibility of more subtle actinic skin changes in this white patient with blue eyes (Fitzpatrick 1−2), who inherently has a higher risk of skin cancer. Theoretically, the scarring process after biopsy could have accelerated transformation into malignancy.

Overall, to our knowledge, this is the first case of SCC arising at the site of a TAB incision. It is one of a handful of scar carcinoma cases that did not arise from a thermal burn. There are a few key takeaway points to highlight from this case. First, malignancy should always be considered in a nonhealing wound or scar or a scar that has changed in character. There should be a low threshold to biopsy and rebiopsy a wound not healing as expected. Second, it is possible for skin malignancies to become secondarily infected and obscure the clinical picture, as was the case here when the culture revealed the organism *C. parapsilosis*. Although treatment for the infection is warranted, one must still consider an underlying malignancy even when an organism is identified. It is also valuable to involve the appropriate interdisciplinary team when carrying out definitive treatment.

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