

Chronic Ulcerative Lesion

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A 46-year-old man with a history of a left leg burn during childhood that was unsuccessfully treated with multiple skin grafts presented as a hospital follow-up for outpatient management of an ulcer. The patient had an ulcer that gradually increased in size over 7 years. Over the course of 2 weeks prior to the hospital presentation, he noted increased pain and severe difficulty with ambulation but remained afebrile without other systemic symptoms. Prior to the outpatient follow-up, he had been admitted to the hospital where he underwent imaging, laboratory studies, and skin biopsy, as well as treatment with empiric vancomycin. Physical examination revealed a large undermined ulcer with an elevated peripheral margin and crusting on the left lower leg with surrounding chronic scarring.

WHAT'S YOUR DIAGNOSIS?

- arterial ulcer
- Marjolin ulcer
- pyoderma gangrenosum
- ulcerative lichen planus
- venous ulcer

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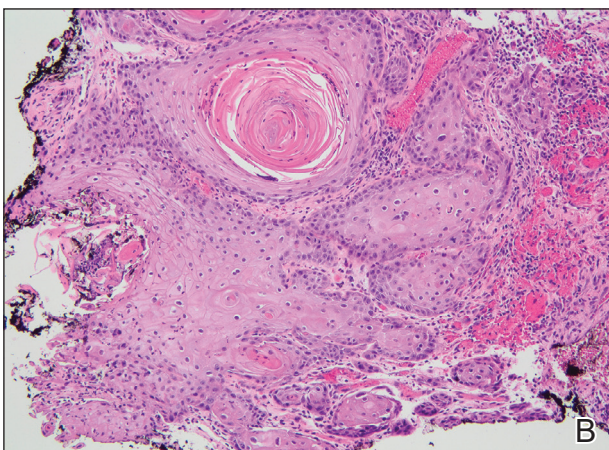
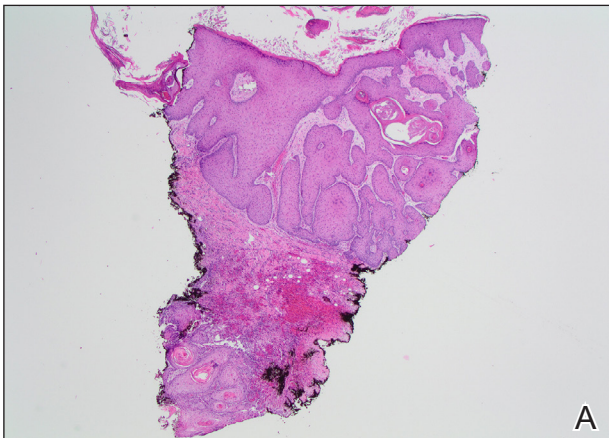
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THE DIAGNOSIS: Marjolin Ulcer

A skin biopsy during his prior hospital admission demonstrated an ulcer with granulation tissue and mixed inflammation, and the patient was discharged with close outpatient follow-up. Two repeat skin biopsies from the peripheral margin at the time of the outpatient follow-up confirmed an invasive, well-differentiated squamous cell carcinoma (Figure), consistent with a Marjolin ulcer. Radiography demonstrated multiple left iliac chain and inguinal lymphadenopathies with extensive subcutaneous disease overlying the left medial tibia. After tumor board discussion, surgery was not recommended due to the size and likely penetration into the muscle. The patient began treatment with cemiplimab-rwlc, a PD-1 inhibitor. Within 4 cycles of treatment, he had improved pain and ambulation, and a 3-month follow-up positron emission tomography scan



A and B, A punch biopsy revealed irregular lobules of pale-staining keratinocytes extending from a hyperplastic epidermis into the underlying dermis with infiltrative lobules of atypical keratinocytes with central keratinization in the deep dermis (H&E, original magnifications $\times 20$ and $\times 100$).

revealed decreased lymph node and cutaneous metabolic activity as well as clinical improvement.

Marjolin ulcers are rare and aggressive squamous cell carcinomas that arise from chronic wounds such as burn scars or pressure ulcers.¹ Although an underlying well-differentiated squamous cell carcinoma is the most common etiology, patients also may present with underlying basal cell carcinomas, melanomas, or angiosarcomas.² The exact pathogenesis underlying the malignant degeneration is unclear but appears to be driven by chronic inflammation. Patients classically present with a nonhealing ulcer associated with raised, friable, or crusty borders, as well as surrounding scar tissue. There is a median latency of 30 years after the trauma, though acute transformation within 12 months of an injury is possible.³ The diagnosis is confirmed with a peripheral wound biopsy. Surgical excision with wide margins remains the most common and effective intervention, especially for localized disease.¹ The addition of lymph node dissection remains controversial, but treatment decisions can be guided by radiographic staging.⁴

The prognosis of Marjolin ulcers remains poor, with a predicted 5-year survival rate ranging from 43% to 58%.¹ Dermatologists and trainees should be aware of Marjolin ulcers, especially as a mimicker of other chronic ulcerating conditions. Among the differential diagnosis, ulcerative lichen planus is a condition that commonly affects the oral and genital regions; however, patients with erosive lichen planus may develop an increased risk for the subsequent development of squamous cell carcinoma in the region.⁵ Furthermore, arterial ulcers typically develop on the distal lower extremities with other signs of chronic ischemia, including absent peripheral pulses, atrophic skin, hair loss, and ankle-brachial indices less than 0.5. Conversely, a venous ulcer classically affects the medial malleolus and will have evidence of venous insufficiency, including stasis dermatitis and peripheral edema.⁶

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