Marjolin Ulcer: A Comprehensive Review

Kamran Khan, MD, Resident Physician, Division of Plastic & Reconstructive Surgery, University of North Carolina, Chapel Hill
Charles Schafer, DO, Resident Physician, Department of General Surgery, University of North Carolina, Chapel Hill
Jeyhan Wood, MD, Assistant Professor of Surgery, Director of Craniofacial and Pediatric Plastic Surgery, Program Director Residency Program Plastics, Division of Plastic & Reconstructive Surgery, University of North Carolina, Chapel Hill

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GENERAL PURPOSE: To provide a comprehensive review of Marjolin ulcer (MU) to assist clinicians in understanding the epidemiology, etiology, pathogenesis, diagnosis, and treatment of MU.

TARGET AUDIENCE: This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES: After participating in this educational activity, the participant will:

1. Describe the epidemiology, pathogenesis, and clinical manifestations of MU.
2. Summarize the diagnostic and treatment approaches for patients who have an MU.

ABSTRACT

This article aims to provide a comprehensive review of Marjolin ulcer (MU) to assist clinicians in understanding the epidemiology, etiology, pathogenesis, diagnosis, and treatment of MU. Marjolin ulcer presents with clear signs and symptoms of malignant degeneration in chronic wounds. It can be prevented by raising awareness and educating wound care providers appropriately about its signs and symptoms.

KEYWORDS: burns, malignancy, Marjolin, reconstruction, review, ulcer

ADV SKIN WOUND CARE 2020;33:629–34.
DOI: 10.1097/01.ASW.0000720252.15291.18

INTRODUCTION

Marjolin ulcer (MU) has classically referred to squamous cell carcinoma arising in burn scars; however, it is also used to describe aggressive malignant degeneration in any chronic wound.[1] The first recorded observation of malignant degeneration in burn scars was in the first century AD by the Roman encyclopedist Aurelius Cornelius Celsus.[1–4] However, it was not until 1828 that Jean Nicholas Marjolin, a French surgeon, described the ulcerative transformation of burn scars in the *Dictionnaire de Medecine*,[1,4] although they were not recognized as malignant at the time.[2,3]

The first manuscript describing the malignant transformation of burn scars was "Warty Tumours of Cicatrices," published in 1833 by Caesar Hawkins.[1,4] Six years later, a more complete description was made by Dupuytren, who wrote about a patient who developed a malignancy at the site of a previously healed sulfuric acid burn injury.[2–5] In the late 1840s, Byron and Smith correctly identified these previous descriptions as malignancies, with Smith calling them "warty ulcers" of Marjolin.[3,6] The current term was coined in 1903 by DaCosta: "The characterization of this condition as Marjolin's ulcer I think to be proper, because it was first carefully studied and accurately described by Professor Marjolin, of Paris, over 50 years ago."[6] The term was then consistently used and expanded by Da Costa and Fordyce.[1,3,5]

This article provides a comprehensive review to assist clinicians in understanding the epidemiology, etiology, pathogenesis, diagnosis, and treatment of MUs. Wound

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care providers need to understand the signs and symptoms of malignant degeneration in chronic wounds in order to adequately address this condition.

EPIDEMIOLOGY

Approximately 1.7% of chronic wounds undergo malignant transformation, and the incidence in burn scars specifically is 0.77 to 2.0%.[4,7,8] Marjolin ulcer is prevalent in all races and age groups, but the average age of diagnosis is in the fifth decade of life.[1–3,9] There appears to be a male-to-female predominance, with an estimated ratio of 2:1; however, some reports suggest a 3:1 or 1:1 ratio.[1,2,4,8,9] Because of the cultural and economic differences among developed and developing countries, the incidence of MU varies globally. For example, in certain Asian cultures, heating pads are frequently used for comfort. Asian individuals therefore may experience chronic burn wounds and, in turn, MU (with a reported incidence up to 6.8%). These have been described as "Kangri ulcers" in Kashmir, "Kairo burn cancer" in Japan, and "Kang ulcers" in China.[3]

The latency period between initial injury and subsequent malignancy may be as long as 32 years.[7,10,11] There appears to be an inverse relationship between patient age and the length of the latency period, with younger patients experiencing a longer latency.[7]

The most common histologic variant of MU is squamous cell carcinoma (71%); however, it constitutes only a small portion of all squamous cell carcinomas (2%).[1] Other manifestations include basal cell carcinoma, melanoma, angiosarcoma, fibrosarcoma, osteosarcoma, dermatofibrosarcoma protuberans, malignant fibrous histiocytoma, liposarcoma, leiomyosarcoma, and malignant schwannoma.[1]

Full-thickness burns that heal by secondary intention are predisposed to malignancy. In these situations, malignant degeneration occurs in approximately 0.77% to 2% of cases. The incidence of malignant degeneration in cases of chronic osteomyelitis is 0.2% to 1.7%, in contrast with a 2% incidence in burn scars.[9]

Marjolin ulcers make up 0.05% of squamous cell carcinomas arising in the lower extremities. Of all cancers that develop in leg ulcers, epidermoid carcinomas constitute 0.21% to 0.34%.[9,12] One in 300 leg ulcers can actually be attributed to malignancy, and patients with venous leg ulcers have a relative risk of 5.8% for the development of nonmelanoma skin cancer compared with the general population.[4,10,12]

Anatomic Sites of Occurrence and Predisposition

An MU most commonly occurs in the lower extremities (53.3%, and in one case series 81.5%), particularly the plantar foot; however, they can develop in several anatomical regions.[1,2,5,8,9] Sites of development in decreasing order of frequency include the head and neck (30%), upper extremities (18.7%), trunk (12.4%), and rarer locations (nose, eyelid, lip, foot, digits).[2,3,11,13,14] These lesions may be attributable to injuries that affect the arms and legs (trauma, burns, venous stasis ulcers, and osteomyelitis).[11,13] There may also be an association between excess joint movement and ulcer development, which could account for the frequent presentation of MU involving the knee region.[2] Scars present in flexion creases may be exposed to recurrent trauma leading to wound instability.[3] In addition, the high degree of lower extremity involvement may be because of the frequency of burns in this region.

Most MUs are seen in old burn scars (75%). However, the overall rate of malignant transformation in burn scars is less than 2%.[1] In addition to old burn scars, numerous chronic inflammatory conditions and unique experiences may predispose individuals to malignant transformation: chronic osteomyelitis, venous stasis ulcers, pressure injuries, diabetic foot ulcers, tropical ulcers, discoid lupus ulcers, vaccination sites, knife wounds, snakebites, frostbite, pilonidal abscesses, anal fistulae, cystostomy sites, amputation, skin grafts, hidradenitis suppurativa, dermatitis artefacta, Fournier gangrene, radiotherapy, and trauma wounds with delayed treatment.[1,4,7,10,12,14–16]

PATHOGENESIS

The typical time elapsed between trauma and the development of an MU is years; however, an accelerated onset of months to weeks is possible.[1] The latency period between initial injury and subsequent malignancy varies considerably in the literature, although 20 to 35 years is a common estimate.[2,4,12,15] Marjolin ulcers can be classified as acute (malignant transformation within 12 months) or chronic.[1] As previously stated, older adults often have a shorter latency period than younger individuals.[1,2] It is therefore possible that many patients diagnosed with an MU had childhood injuries that subsequently developed into a carcinoma over a period of 30 years or more.[8]

Numerous theories have been described as to the pathophysiology behind MU; however, there is no clear consensus. It is likely a multifactorial process with both a genetic and environmental component. It is known that these malignancies tend to develop in locations where there is constant inflammation and inadequate blood flow, such as burn scars. Therefore, repeated ulceration and poor healing from constant irritation are the most widely agreed-upon theory.[1,7] This concept is further strengthened by Virchow's hypothesis that chronic irritation is a factor in the initiation of carcinoma.[16] An increased rate of spontaneous mutations from prolonged inflammation and repetitive healing attempts has also been suggested.[13,17] In chronic ulcers, the point of most

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rapid cellular turnover is the site where malignant transformation usually occurs. Therefore, malignant transformation is more common at the wound edge.[10]

Cutaneous squamous cell carcinoma is characterized by the "two-hit" pathogenesis model, whereas two mutations in the p53 tumor suppressor gene are necessary for malignant transformation.[7] This pathogenesis model is likely one of numerous pathways that ultimately lead to MUs. It also has been hypothesized that there may be a deficiency of innate immunologic cells (natural killer cells) that normally counter malignancy in the epithelium of chronic ulcers. Neoplastic cells are subsequently capable of evading immunosurveillance, increasing the risk of metastasis.[13,17]

Further, MUs in burn scars have been shown to have mutations in the FasR (CD95) gene that controls programmed cell death, ultimately causing uncontrolled cellular proliferation.[1,7] The formation of scar tissue after a burn injury may obliterate lymphatics, leading to decreased immune surveillance.[7] Tumors can subsequently multiply to large sizes before breaching the burn-scar barrier.[15] However, burn scars increase tumor progression in existing neoplastic cells rather than increasing the rate of neoplastic development in cells; this is known as the co-carcinogen theory.[7,15]

The decreased vascularity and weak epithelium of granulation tissue itself may be predisposed to malignancy. For example, after a full-thickness burn, the eschar formed may cause a release of toxins from tissue hypoxia.[15] Autolysis of scar tissue may also release toxins that have a direct mutagenic effect on cells.[7,17] In addition, decreased circulation of lymphocytes may lead to an inadequate immunologic response and chronic infections.[1] Finally, any wound that healed by secondary intention is at risk of MU.

Clinical Indications of Malignant Degeneration in Chronic Wounds

First, providers must note that a lack of awareness of the signs and symptoms of malignant degeneration in chronic wounds leaves patients susceptible to MU. Wound care providers often encounter cases in which long-term wound care is needed, sometimes involving months or years of care. During this time, patients may change providers frequently, making the identification of any subtle wound changes difficult. Therefore, clear communication among providers and detailed documentation of wound characteristics in the medical record are crucial. It is also essential for wound care providers to be cognizant of the signs and symptoms of malignant degeneration in chronic wounds. In addition, patients with a history of extensive burn scars should not only be made aware of the possibility of malignant transformation but should also be educated on the signs and symptoms to look out for.

Any wound appearing decades after one of the predisposing factors of MU should evoke a high index of suspicion for malignancy, and a subsequent biopsy is warranted. Further, a nonhealing ulcer in an area of abnormal or scarred skin should be considered an MU until proven otherwise.

The most common presentation is a flat, ulcerative lesion with raised margins and surrounding induration.[15] The two major morphologic forms of MU include the well-differentiated exophytic form, which generally has a better prognosis, and the poorly differentiated ulcerative form, which often has a poor prognosis because of invasion.[2] One case series of 27 patients with MU developing in old burn scars documented an 85% incidence of the ulcerative variant, compared with a 15% incidence of the exophytic presentation.[8] In the same study, signs of the original burn injury (scarring) were present in 100% of participants.[8] Further, lymphadenopathy was present in 11.1% of patients at the time of diagnosis.[8]

Indications of malignant degeneration (Table) include chronic ulceration longer than 3 months, exophytic granulation tissue formation, everted or rolled margins, protracted wound course despite appropriate treatment, excess bleeding, malodorous discharge, spontaneous pain, and regional lymphadenopathy.[1,2,10,12,14,18–20] In the presence of any of these indications, a biopsy is warranted.

DIAGNOSIS

The current standard diagnostic test for MU is histologic analysis via biopsy. An excisional, incisional, or punch biopsy may be obtained. These should be taken from multiple locations of the ulcer including the margins to minimize false-negative results.[1,12] However, the most important aspect of MU diagnosis is patient history.

Table. CLINICAL INDICATIONS OF MALIGNANT DEGENERATION[1,2,10,12,18–20]

• Chronic ulceration longer than 3 months' duration
• Exophytic granulation tissue formation
• Everted or rolled margins
• Protracted wound course despite appropriate treatment
• Excess bleeding
• Malodorous discharge
• Spontaneous pain
• Regional lymphadenopathy
• Irregular margins
• Change in wound drainage

Note: A combination of these findings will likely be more reliable than single findings.

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Studies have shown that MU can be prevented with early wound surveillance and biopsy of any wound change.[7,15,21] Some authors even recommend annual biopsies of chronic ulcers.[10] Another recommendation is to consider a biopsy of any wound that does not heal within 1 to 3 months.[2,10]

The subsequent steps after histologic confirmation of malignancy include tumor staging. Computed tomography (CT) scan and MRI may provide the degree of soft tissue involvement; however, these are not required for diagnosis. Clinical examination of regional lymph node basins is necessary. The regional lymph nodes can be staged clinically or radiologically.[2] Further, if a diagnosis of MU is made and the patient is clinically node-negative, a sentinel lymph node biopsy is a reasonable consideration. Because of the high rate of MU metastasis, a distant metastatic workup with a positron emission tomography scan, chest CT scan, abdominal ultrasound, and brain CT scan should be obtained.[2,12]

TREATMENT

The best treatment of MU is prevention. The appropriate management of burns, chronic ulcers, and unstable scars is necessary to prevent malignant degeneration. Therefore, a measure such as excision with skin grafting or surgical flap coverage of a burn wound will go a long way in preventing potential complications.

Once the diagnosis of MU is made, the treatment process should involve an interprofessional team, including oncology, dermatology, and plastic surgery if necessary. Wound care specialists should also be involved for proper care, assessment, and prevention.

Surgical Intervention

The mainstay of treatment for MU is wide local excision; however, there is no clear consensus for resection margins. A review of the literature found no randomized controlled trials assessing resection margins for MU. However, there is general agreement in the range of 2- to 4-cm margins.[1,2,7,8,15,22] In areas in which primary closure is not possible, wound coverage with a skin graft or local flap (fasciocutaneous versus musculocutaneous) via adjacent tissue transfer is appropriate. This may ultimately require free tissue transfer at the discretion of the surgeon. Of note, an approach with cautery dissection is preferable to sharp dissection to prevent iatrogenic seeding of tumor cells.[2] An additional margin of tissue can then be obtained with sharp dissection to ensure proper healing and negative margins. Surgical margins should be confirmed intraoperatively by frozen section if possible.

Clinically or radiologically involved lymph node groups are treated with lymph node dissection.[2] A reasonable approach for patients with clinically node-negative MU is sentinel lymph node biopsy or regional nodal irradiation.[2] Lymphatic mapping may be useful, especially in patients with MUs at sites in which the lymphatic drainage is unpredictable.[15]

Chemotherapy and Radiotherapy

There is no established consensus on the adjuvant or neoadjuvant management of MU. That said, patients with widespread metastatic disease may benefit from palliative, adjuvant chemoradiation therapy.[1]

Ozek et al[23] proposed clear indications for radiotherapy in MUs: (1) inoperable regional lymph node metastasis; (2) grade 3 lesions with positive lymph nodes after nodal dissection; (3) MU diameter greater than 10 cm with positive lymph nodes present after node dissection; (4) grade 3 lesions with an MU diameter greater than 10 cm and negative lymph nodes after node dissection; and (5) MU of the head and neck with positive lymph nodes after lymph node dissection.[2,23]

Chemotherapy is often used in patients who are not appropriate surgical candidates. The regimen may consist of topical or systemic 5-fluorouracil in combination with cisplatin, methotrexate, and bleomycin.[2,24,25]

Last, hyperthermic intra-arterial limb perfusion has also been proposed, but this concept is beyond the scope of this review.

Prognosis

The subtle presentation of MUs often causes a delay in diagnosis and treatment. This consequently leads to a poor prognosis, and deaths from MU have been reported.[8,15,26–29] In fact, the overall mortality from MU is 21%.[2] The most important prognostic factor is histologic grading, with evidence of lymph node metastasis associated with the worst prognosis.[7]

Overall, MUs have a reported metastatic rate between 27.5% and 40%.[7,30] However, the metastatic rate of MU from pressure injuries may be as high as 61%, a rate much higher than of burn scars (38%) or osteomyelitis (14%).[11,13] At 3 years postdiagnosis, the overall survival for patients with MU is 65% to 75%, but this decreases to 35% to 50% if there is metastatic disease on presentation.[1] In addition, there is a 52% and 23% 5- and 20-year survival, respectively.[2]

Factors that predict a poor prognosis include palpable regional lymphadenopathy (predicts death within 2 years), lower limb involvement, tumor size greater than or equal to 2 cm, local invasion of the lesion, short latency period, recurrent MU, and distant metastatic disease.[1–3]

CASE STUDY

An 85-year-old White man with history of paraplegia had a nonhealing ulcer of his lower back for a period of 10 years. His wound care provider noted a rapidly

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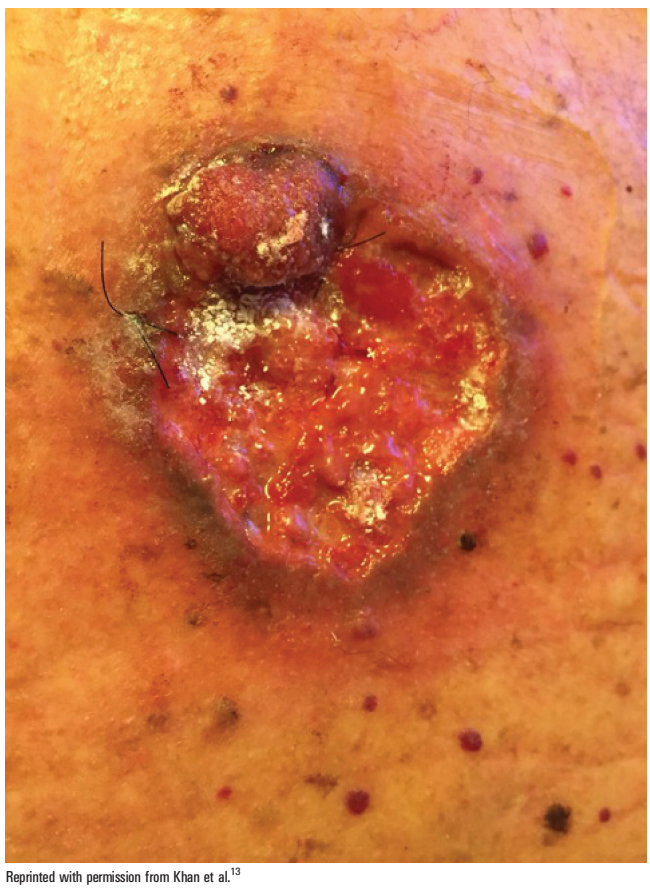
growing superimposed mass over a period of 4 months suggestive of malignancy. A punch biopsy was obtained at the periphery of the area of concern (Figure). Histopathologic examination revealed noninfiltrating, well-differentiated squamous cell carcinoma. The ulcer was excised with 2-cm margins, and a split-thickness skin graft was placed. Further workup did not reveal metastasis. The patient's wound has since healed, and there are no signs of recurrence at 3 years. Permission was obtained from this patient to publish the details of the case and associated image.

CONCLUSIONS

Marjolin ulcer describes the aggressive malignant degeneration in any chronic wound. It has a very poor prognosis, with a mortality of 21%. Multiple studies have shown that MU is preventable with early wound surveillance, and the timely assessment of any wound changes is necessary via biopsies. It is imperative that wound care providers are aware of the signs and symptoms of malignant degeneration in chronic wounds. This in turn will allow for swifter diagnosis and intervention prior to metastasis, improving patient outcomes.

Figure. SACRAL PRESSURE INJURY WITH A SUPERIMPOSED SQUAMOUS CELL CARCINOMA[13]



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PRACTICE PEARLS

- Old burn scars are the most common type of wounds predisposed to MUs, but other types of wounds including chronic osteomyelitis, venous stasis ulcers, pressure injuries, and diabetic foot ulcers can also lead to malignant degeneration.
- Any nonhealing, chronic wound in an area of previously injured or scarred skin should be considered an MU until proven otherwise.
- The current standard diagnostic test for MU is histologic analysis via biopsy; an excisional, incisional, or punch biopsy should be taken from multiple locations around the ulcer, including the margin, to minimize false-negative results.
- Common signs of malignant degeneration that should prompt a biopsy include chronic ulceration longer than 3 months, exophytic granulation tissue formation, everted or rolled margins, and regional lymphadenopathy.
- The latency period from initial injury to the development of malignancy is variable, but could be as long as 32 years.
- Wound care providers should be aware of the clinical signs and symptoms of malignant degeneration in chronic wounds. This will allow for swifter diagnosis and earlier intervention from the interprofessional team. ●

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