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Management of a Large Necrotic Abdominal Wall Ulcer Following Repeated Insulin Injections in a Patient with Diabetes Mellitus: A Case Report

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ABSTRACT

Background: Large abdominal wall ulcers resulting from repeated insulin injections are exceptionally uncommon, with only isolated reports describing progressive soft-tissue necrosis arising from chronic injection-related microtrauma. Diabetic patients are particularly vulnerable to such complications due to underlying microangiopathy, impaired immunity, neuropathy, and diminished tissue resilience. This case is reported to highlight an unusual presentation of progressive abdominal wall necrosis in a long-standing insulin-dependent diabetic patient and to emphasize key principles of assessment and management.

Case Presentation: A 69-year-old woman with long-standing type 2 diabetes mellitus presented with a progressively enlarging ulcer in the left upper abdominal quadrant, initially developing at a habitual insulin injection site. Examination revealed a large necrotic defect with extensive subcutaneous tissue loss but preserved fascial integrity. Laboratory evaluation showed hyperglycemia without systemic inflammatory response. Imaging and intraoperative assessment did not support necrotizing fasciitis. The patient underwent staged surgical debridement followed by negative pressure wound therapy to optimize the wound bed. Once robust granulation developed, definitive coverage was achieved with a split-thickness skin graft, resulting in complete graft take and uneventful recovery.

Conclusion: This case underscores the potential for repeated insulin injections to precipitate extensive soft-tissue injury in diabetic patients, even in the absence of overt infection or necrotizing fasciitis. Early recognition, meticulous debridement, and timely initiation of negative pressure wound therapy are essential to preventing disease progression and enabling successful reconstruction. Clinicians should maintain a high index of suspicion for injection-site complications and reinforce preventive education on rotation techniques and site inspection in vulnerable patients.

Keywords: Diabetic wound, Insulin injection site, Abdominal wall necrosis, Negative pressure wound therapy, Split-thickness skin graft

Introduction

Diabetic wounds represent one of the most challenging complications of chronic hyperglycemia and continue to impose a substantial burden on global health systems.[1,2] Long-standing diabetes mellitus impairs multiple components of the wound-healing cascade, including leukocyte function, microvascular integrity, angiogenesis, collagen turnover, and cellular immunity.[1,2] These factors predispose patients to delayed healing, progressive tissue breakdown, and increased susceptibility to infection even after minor trauma.[1-3] Chronic wounds in diabetes fre-

quently exhibit histopathologic features such as subcutaneous necrosis, fibrosis, and impaired granulation, reflecting the complex interplay between neuropathy, ischemia, repetitive pressure, and immunologic dysfunction.[1-4]

Although diabetic lower-extremity ulcers have been extensively characterized, atypical wound locations—including the abdominal wall—may also be affected when microvascular compromise, neuropathy, or repeated local trauma are present.[1-3] Repeated subcutaneous insulin injections can induce lipohypertrophy, fat necrosis, and regional ischemia, creating a vulnerable microenvironment predisposed to ulceration.[1,5] While minor injection-site reactions are relatively common, severe necrotizing complications are exceedingly rare and are poorly represented in the literature.[5,6] Subacute and progressive soft-tissue necrosis following insulin administration has been described, particularly in patients with poorly controlled diabetes or significant microangiopathy, highlighting the potential for small injection-related injuries to evolve into extensive defects when tissue perfusion is compromised.[2,5]

Extensive abdominal wall necrosis is more commonly associated with necrotizing soft-tissue infections, postoperative complications, or ischemic adipose tissue injury.[7-10] However, a subset of noninfective or limited-infection necrotic wounds may arise in diabetic patients from relatively minor insults that would otherwise heal uneventfully in healthy individuals.[1,3,5] These lesions often require aggressive surgical debridement and prolonged wound care, mirroring management principles used for necrotizing fasciitis or complex traumatic wounds.[1,7,9,11] Early recognition and intervention are critical, as delays in debridement are known to worsen the depth and extent of necrosis and increase the risk of systemic complications.[7,11,12]

Advanced wound-care modalities, particularly negative pressure wound therapy (NPWT), have transformed the management of complex diabetic wounds and soft-tissue defects.[1,13,14] NPWT improves perfusion, promotes granulation tissue formation, reduces interstitial edema, and streamlines bacterial bioburden control.[13-15] It has become an integral component of diabetic wound management, traumatic wounds, abdominal wall defects, and necrotizing soft-tissue infections, serving both as a bridge to definitive surgical closure and a bolster mechanism to enhance graft take.[1,14-18] When combined with meticulous debridement, NPWT facilitates the creation of a well-vascularized wound bed suitable for split-thickness skin grafting (STSG), which remains the standard reconstructive option when fascia and deeper layers are preserved.[8,14,17,19,20]

Despite the widespread use of NPWT and STSG in complex wound care, reports of extensive abdominal wall necrosis directly attributable to repeated insulin injections are sparse. Given the aging diabetic population and the lifelong necessity of insulin administration in many patients, awareness of this rare but significant complication is essential. This case report describes the successful management of a 20-cm necrotic abdominal wall ulcer at an insulin injection site using staged surgical debridement, NPWT, and STSG. The report aims to highlight the underlying mechanisms, surgical decision-making, and evidence-based rationale for this approach, thereby contributing to the limited literature on severe injection-related soft-tissue injury in diabetes mellitus.

Case Presentation

A 69-year-old woman presented to the surgical clinic with a chronic, foul-smelling ulcer involving the left upper quadrant of the abdominal wall. She reported that the lesion had originated several months earlier as a small, indurated area at a routinely used insulin injection site and had progressively enlarged despite conservative wound care, ultimately evolving into a large necrotic ulcer measuring approximately 20 cm in diameter. Over time, the lesion became increasingly painful and developed a thick, dark eschar.

Her medical history was notable for a 10-year diagnosis of type 2 diabetes mellitus and known coronary artery disease. She was receiving subcutaneous insulin therapy consisting of a single daily dose of 10 IU neutral protamine Hagedorn (NPH) insulin, self-administered by the patient. Although she reported using a new needle for each injection, she acknowledged inconsistent adherence to recommended injection-site rotation practices. There was no history of peripheral vascular disease, immunosuppression, prior abdominal infections, or systemic symptoms such as fever, chills, or malaise.

On admission, her height was 1.62 m and body weight was 90 kg, corresponding to a body mass index (BMI) of 34.3 kg/m^2, consistent with obesity. Physical examination revealed a large ulcer characterized by extensive necrotic plaques, erythematous and indurated margins, and purulent discharge (Figure a–c). There was no crepitus, fluctuance, or palpable subcutaneous emphysema, and deep palpation suggested that the necrosis was confined to the subcutaneous tissue without fascial involvement.

Laboratory evaluation demonstrated a serum glucose level of 216 mg/dL and a glycated hemoglobin (HbA1c) level of 6.9%. The white blood cell count was mildly elevated, and C-reactive protein levels were increased, consistent with localized infection. No biochemical or clinical indicators of systemic sepsis were present. Wound cultures yielded mixed skin flora without multidrug-resistant organisms.

Under general anesthesia, the patient underwent thorough surgical debridement, during which the thick eschar and devitalized subcutaneous tissues were excised until healthy, bleeding tissue was encountered (Figure c). The underlying fascia was intact, confirming that the necrosis had not extended beyond the subcutaneous fat. Following debridement, negative-pressure wound therapy (NPWT) was initiated using standard continuous suction parameters (Figure d). NPWT promotes wound healing by applying controlled subatmospheric pressure that removes exudate, reduces tissue edema and bacterial burden, enhances local perfusion, and mechanically stimulates granulation tissue formation. Dressings were changed at appropriate intervals, resulting in progressive reduction of exudate and rapid development of uniform granulation tissue across the wound bed.

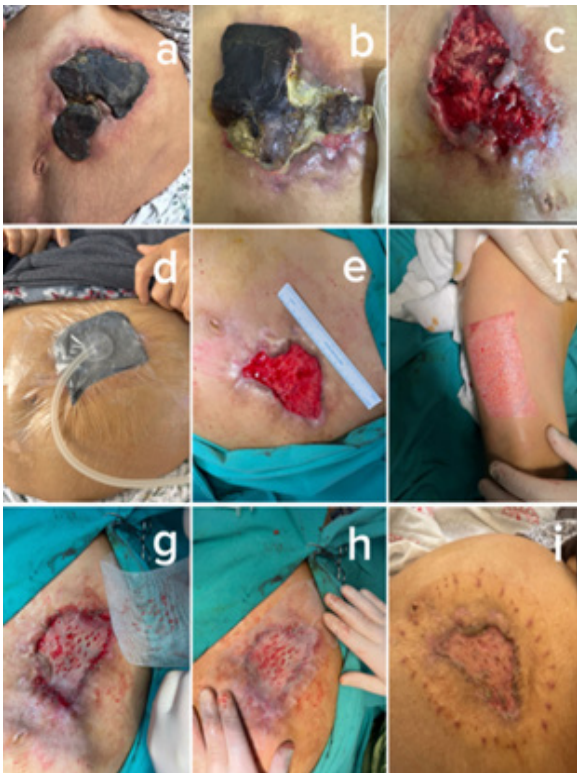

Figure 1. Sequential clinical course, surgical management, and reconstruction of an extensive abdominal wall ulcer arising from an insulin injection site.

(a) Initial presentation showing a large necrotic eschar occupying the left upper abdominal quadrant with surrounding induration. (b) Progression to wet gangrenous necrosis with malodor and peripheral erythema prior to surgical intervention. (c) Intraoperative appearance after the first radical debridement demonstrating extensive subcutaneous tissue loss with preserved fascial integrity. (d) Application of negative pressure wound therapy (NPWT) following initial debridement to promote granulation and control exudate. (e) Wound appearance after repeated NPWT cycles, showing a well-vascularized granulation bed suitable for grafting. (f) Harvesting of a split-thickness skin graft (STSG) from the lateral thigh donor site. (g) Placement of the meshed STSG over the optimized abdominal wound bed. (h) Immediate postoperative appearance following graft fixation and reapplication of NPWT as a bolster dressing. (i) Final outcome demonstrating complete graft integration and stable scar formation at follow-up.

Over sequential therapy sessions (Figure e), the wound demonstrated progressive contraction, improved vascularity, and no areas of residual necrosis.

Once a robust granulation bed was achieved, definitive closure was performed using a split-thickness skin graft harvested from the lateral aspect of the thigh (Figure f). The graft was secured and supported with NPWT used as a bolster dressing to enhance graft adherence and minimize shear forces (Figure g–h). The postoperative course was uneventful. By postoperative day 5, the graft demonstrated full viability without evidence of hematoma, seroma, infection, or partial loss. The patient was discharged with detailed instructions regarding glycemic control, wound care, and appropriate insulin injection-site rotation. At the 1-week outpatient follow-up, the graft showed complete integration and continued healing without complications (Figure i).

Written informed consent was obtained from the patient for publication of this case and accompanying images.

Discussion

Large abdominal wall ulcers arising from repeated insulin injections remain exceptionally rare, with only isolated reports documenting similar patterns of progressive necrosis.[5] In the present case, several factors intrinsic to diabetes mellitus likely contributed to the severity of soft-tissue destruction. Chronic hyperglycemia results in microangiopathic changes, impaired immune function, neuropathy, and defective collagen synthesis, all of which diminish the skin's resilience and ability to repair after repetitive microtrauma.[1–3] Injection-site lipohypertrophy or fat necrosis may further compromise perfusion, creating poorly vascularized tissue planes that are highly susceptible to breakdown.[5] Infections arising at injection sites, particularly with Staphylococcus aureus, have been documented as triggers for subacute necrotizing processes, underscoring the importance of early recognition in diabetic patients presenting with localized pain, induration, or discoloration.[5,7,12]

The progressive enlargement of the ulcer over several weeks in this patient mirrors the clinical trajectory of both necrotizing soft-tissue infections and ischemic adipose tissue necrosis described in the literature.[7–9] Although this lesion was not consistent with necrotizing fasciitis, the management principles remain similar: prompt and adequate debridement is central to halting progression and facilitating recovery.[7,11,12] Serial debridement has been repeatedly emphasized as a cornerstone of therapy in both infectious and noninfectious necrotizing soft-tissue injuries, particularly in diabetic or immunocompromised hosts, where necrosis often extends beyond clinically apparent margins.[7,11,12] In our case, layered debridement revealed preservation of the fascia, enabling a less invasive reconstructive strategy while ensuring complete removal of devitalized tissue.

NPWT played a pivotal role in wound bed preparation, consistent with extensive evidence demonstrating its benefits in complex diabetic wounds, abdominal wall necrosis, and postoperative wound complications.[1,8,14,17,21] By enhancing angiogenesis, promoting granulation, modulating wound exudate, and stabilizing the wound microenvironment, NPWT serves as an ideal bridge between initial debridement and definitive closure.[13–15] The use of NPWT as a bolster dressing for STSG application is also supported by multiple reports, with improved graft adherence and reduced shear forces facilitating successful outcomes.[14,19,20] In our patient, continuous NPWT resulted in rapid development of a robust granulation bed that supported complete graft integration without complication.

The choice of STSG as definitive reconstruction aligns with best practices for large superficial abdominal wall defects in which deeper fascial structures remain intact.[8,19,20] STSG has been reliably used for wounds resulting from abdominal necrotizing fasciitis, postoperative fat necrosis, and extensive debridement for chronic wounds.[7,8,10,20] More complex defects involving the muscular or fascial layers often require flap-based reconstruction, including free flaps or component separation techniques; however, these approaches carry added morbidity and are unnecessary when vascularized granulation tissue is present.[8,22–24] The favorable outcome in this case reinforces the principle that STSG, when preceded by meticulous wound bed optimization, can achieve durable closure for extensive soft-tissue defects in diabetic patients.

This case also highlights the critical role of preventive education. Many patients with long-standing diabetes habitually inject insulin into the same localized region, increasing the risk of lipohypertrophy, local ischemia, fat necrosis, and ulceration. Repeated injections into compromised tissues can precipitate the kind of progressive necrosis observed here.[5] Structured counseling on injection-site rotation, needle reuse avoidance, and inspection of injection areas may significantly reduce the incidence of such complications, especially in elderly or visually impaired patients.

Proper insulin injection technique and systematic site rotation constitute essential components of optimal diabetes management and are strongly recommended in international guidelines to minimize local injection-site complications.[25] Repeated insulin administration to the same anatomical region has been consistently associated with the development of lipohypertrophy, a frequent yet often underrecognized condition, with reported prevalence rates ranging from approximately 20% to over 60% among insulin-treated patients.[26,27] Lipohypertrophic tissue is characterized by abnormal adipocyte proliferation, altered tissue architecture, and impaired local microcirculation, resulting in unpredictable insulin absorption and increased susceptibility to local tissue injury. [26,28]

In the present case, the patient's history of long-standing diabetes and repeated self-administered insulin injections to a single abdominal site likely played a central role in the pathogenesis of the lesion. Inadequate site rotation may have led to cumulative microtrauma, progressive disruption of subcutaneous tissue integrity, and compromised local perfusion. These pathological changes may create a vulnerable microenvironment that predisposes the affected tissue to ischemia and subsequent necrosis rather than a benign inflammatory reaction.

Although rare, ischemic and necrotic complications related to insulin injections have been previously reported, particularly in patients with diabetes-associated microangiopathy, obesity, or impaired wound healing capacity.[29] Several mechanisms have been proposed, including repetitive mechanical trauma from needle insertion, localized pressure effects, insulin-induced alterations in local blood flow, and secondary infection, all of which may act synergistically to promote tissue ischemia.[28,29] In this context, the extensive subcutaneous necrosis observed in our patient likely represents the end result of chronic local injury superimposed on systemic metabolic vulnerability.

The differential diagnosis of a rapidly progressive necrotic ulcer of the abdominal wall is broad and includes necrotizing soft tissue infection, pyoderma gangrenosum, calciphylaxis, cutaneous vasculitis, and malignant soft tissue tumors. Necrotizing fasciitis was considered unlikely due to the absence of systemic toxicity, crepitus, or fascial involvement during surgical exploration. Pyoderma gangrenosum was excluded based on the lack of characteristic violaceous, undermined borders and the absence of associated systemic inflammatory disease. Calciphylaxis was deemed improbable given preserved renal function and the absence of vascular calcifications. Importantly, the clear clinical history of repeated insulin injections at the same site, together with intraoperative findings demonstrating isolated subcutaneous necrosis with preserved fascia, strongly supported an injection-related ischemic etiology.

This case highlights a rare but clinically significant complication of improper insulin injection practices and underscores the importance of continuous patient education regarding correct injection technique and strict site rotation. Increased awareness among clinicians may facilitate earlier recognition of atypical injection-site lesions, prevent diagnostic delay, and reduce the risk of severe local tissue damage requiring surgical intervention.

In summary, the extensive abdominal wall necrosis in this patient illustrates a severe yet underrecognized complication of repeated insulin injections in the context of diabetes mellitus. The staged approach used—early and serial debridement, NPWT to optimize the wound bed, and STSG for definitive reconstruction—is strongly supported by contemporary literature on diabetic wound management and complex soft-tissue defects. Awareness of this complication and adherence to evidence-based wound care principles can facilitate timely diagnosis, appropriate intervention, and excellent functional outcomes in similar cases.

This case report has several inherent limitations that should be acknowledged. First, it represents the clinical course of a single patient, which substantially limits the generalizability of the findings and precludes any causal inference regarding insulin injection practices and the development of extensive abdominal wall necrosis. Second, the follow-up period was relatively short, limited to approximately one week after definitive wound closure, and therefore does not allow assessment of long-term outcomes such as graft durability, scar quality, recurrence of ulceration, or late infectious or ischemic complications. Third, the absence of longitudinal follow-up data restricts evaluation of whether modifications in insulin injection technique and site rotation resulted in sustained prevention of similar lesions. Finally, detailed histopathological analysis of the debrided tissue was not performed, which may have provided additional insight into the underlying microvascular or ischemic mechanisms contributing to tissue necrosis.

Conclusion

Extensive abdominal wall necrosis secondary to repeated insulin injections is a rare but significant complication. A structured approach consisting of radical debridement, NPWT, and subsequent split-thickness skin grafting can achieve successful wound closure. Optimal perioperative glucose management and long-term patient education remain essential to prevent recurrence and ensure durable healing.

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