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The Uncommon Blister: Bullosis Diabeticorum Case Study

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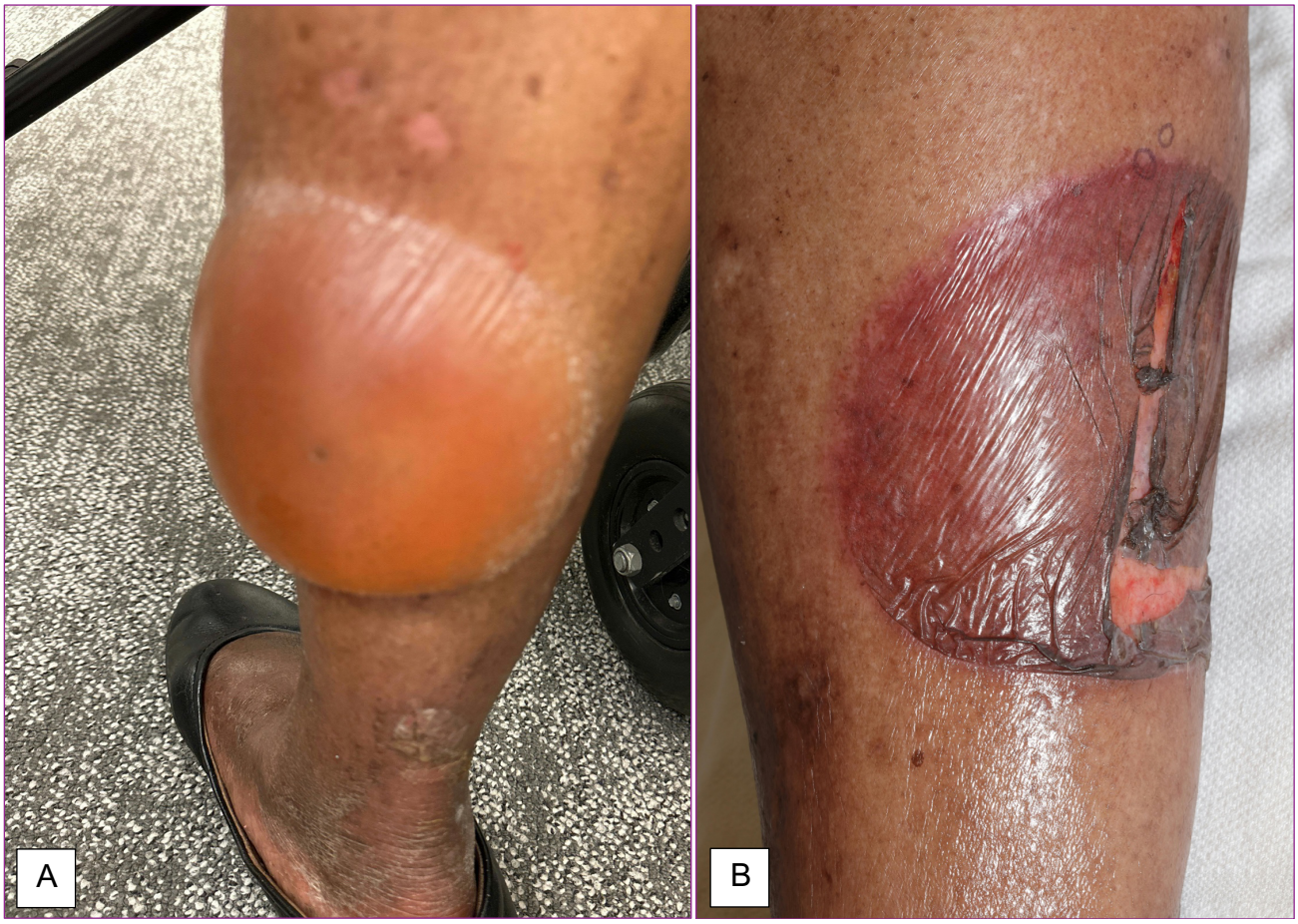

Figure 1. (A) Tense blister over the right medial lower leg
(B) Lanceted blister over the right medial lower leg

A 69-year-old woman with a history of type 2 diabetes mellitus (T2DM) presented to her primary care physician (PCP) for evaluation of a blister on her right lower extremity. The blister was mildly pruritic and had been present for one week. She denied preceding trauma to the area. Physical examination revealed a 14 cm x 10.5 cm tense blister on the right medial lower leg (**Figure 1A**). No other blisters were noted on the skin or mucosa. She was diagnosed by her PCP with presumed bullous cellulitis, who

subsequently lanced the blister (**Figure 1B**) and sent the fluid for gram stain and bacterial culture. The patient was later referred to dermatology.

The following day, the patient was seen in dermatology and diagnosed with presumed bullosis diabeticorum (BD). We performed punch biopsies for hematoxylin and eosin and direct immunofluorescence (DIF) studies. Histopathology revealed intraepidermal and focal subepidermal blistering with variable epidermal necrosis, spongiosis, and minimal inflammation, and DIF staining was negative, findings consistent with BD. Additionally, her hemoglobin A1C was elevated to 16.6. She was referred to a wound care specialist for management of her deroofed blister, as well as endocrinology, neurology, and ophthalmology for evaluation of potential T2DM complications.

BD, also known as diabetic bullae or bullous eruption of diabetes mellitus, is an infrequent blistering disorder associated with either type 1 or type 2 diabetes mellitus.[1] The prevalence of BD has been estimated to be 0.16%, though the true prevalence may be higher due to underreporting. It occurs more frequently in adults, and with a male to female ratio of 2:1.[2] It usually presents in patients with poor glycemic control and peripheral neuropathy or nephropathy, although it can also occur in well-controlled patients.

The exact etiopathology of BD remains elusive. Microangiopathic changes, such as vessel wall thickening and elastic fiber degeneration in the arterioles and dermis, have been linked to BD. Furthermore, UV light exposure with subsequent accumulation of plasma porphyrin has been hypothesized to occur in patients with diabetic nephropathy.[3] Histopathologic findings are nonspecific, revealing subcorneal or suprabasal intraepidermal blisters, subepidermal bullae, with pauci-inflammatory spongiotic or degenerative changes. DIF is commonly negative.[3] The intralesional fluid is protein-rich and sterile, while subepidermal blisters may be hemorrhagic.

Clinically, BD presents abruptly with a gradually expanding, asymptomatic or sometimes mildly pruritic, tense bulla. Occasionally, multiple bullae may be observed. There is no history of preceding trauma or pathergy.[2] While commonly found on the lower extremities, BD may also present on the upper extremities.[1] When assessing for BD, immunobullous and infectious conditions must be excluded. Once a diagnosis of BD is made, the clinician should evaluate for glycemic control and for potential complications of diabetes-associated microvascular disease. Treatment of BD is supportive, as most blisters are self-limited and will resolve within two to six weeks.[4] Although maintaining the integrity of the bulla remains a matter of debate, if a BD lesion becomes deroofed, it should be managed carefully, similar to a diabetic ulcer.

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