Image Letter

Trauma-Induced Bullous Pemphigoid After Coronary Bypass Surgery in a Patient Receiving Gliptin

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Case Presentation

A 63-year-old male with diabetes mellitus, hypertension, and coronary artery disease presented with tense and pruritic bullae on his trunk and left leg two months after coronary artery bypass graft (CABG) surgery. The saphenous vein had been removed from his left leg. One month after surgery, bullae appeared along the surgical scar and subsequently spread to the adjacent skin and sternal incision site (Figure 1A). Mucous membranes were unaffected. The patient was receiving gliptin therapy for diabetes.

Histopathology revealed subepidermal bullae with acantholysis and inflammatory infiltration rich in eosinophils in the upper dermis. Direct immunofluorescence revealed linear IgG and C3 deposition in the basement membrane zone, consistent with bullous pemphigoid (Figure 1C-D). The patient was treated with high-potency topical corticosteroids, resulting in complete resolution of the lesions within one month (Figure 1B). Given the temporal relationship and known risk, gliptin treatment was discontinued.

Teaching Point

This case demonstrates bullous pemphigoid triggered by localized trauma after coronary artery bypass grafting (CABG) surgery in a patient treated with gliptin. Both mechanical trauma and DPP-4 inhibitors are known inducers of bullous pemphigoid; their combined effects may synergistically trigger the disease [1,2]. Clinicians should consider bullous pemphigoid in the presence of postoperative blistering, particularly in patients receiving gliptin, and consider the benefits of medication adjustment in such cases.

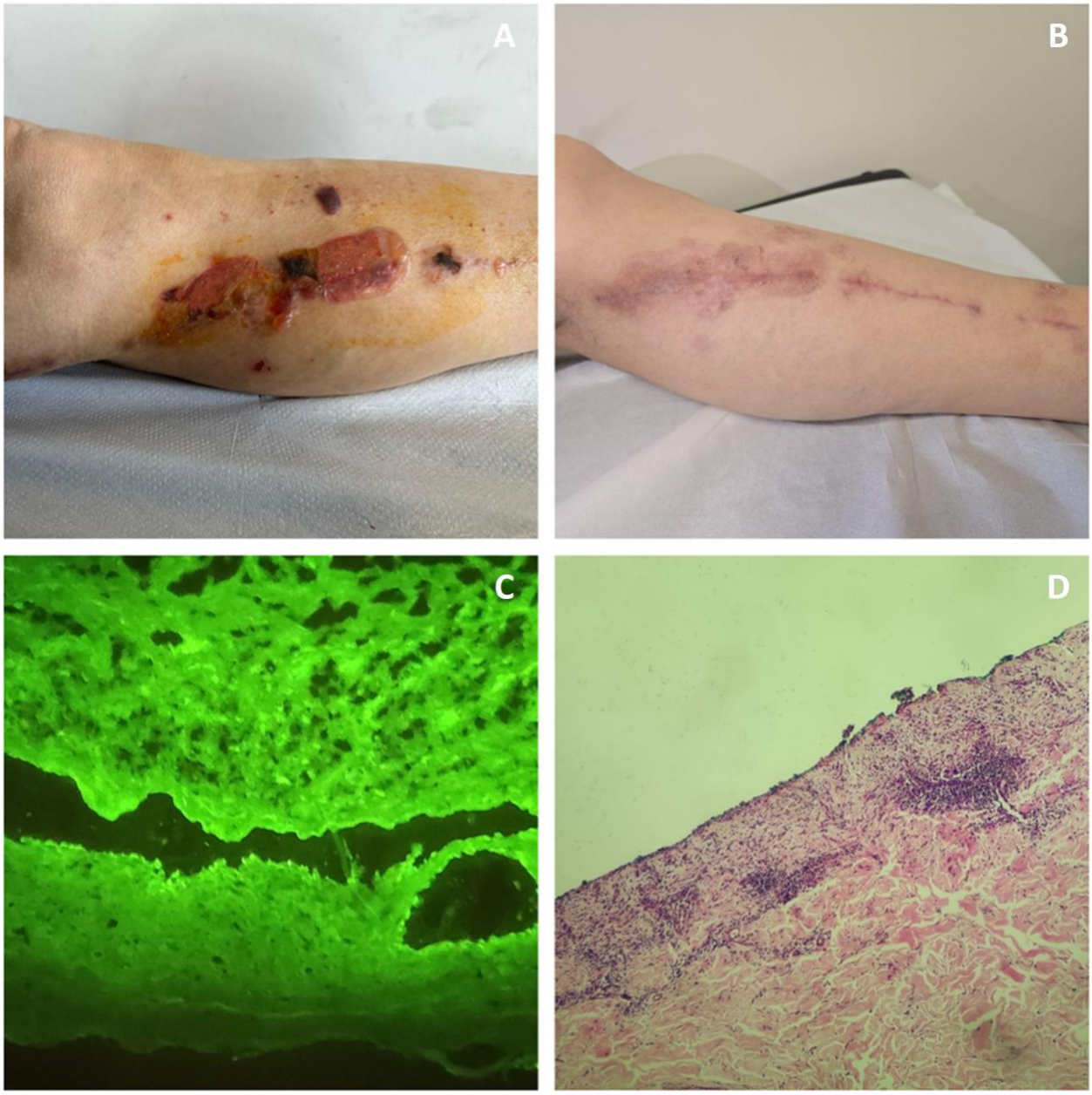

Figure 1. A) Multiple intact and ruptured bullae localized along the saphenous vein harvesting site on the left lower extremity; B) Marked clinical improvement with complete resolution of lesions five months after treatment; C) Direct immunofluorescence showing linear deposition of C3 and IgG along the basement membrane zone (40x); D) Histopathological examination revealing total epidermal loss due to dermoepidermal separation and inflammatory infiltration with eosinophils in the upper dermis (H&E, 40x).

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