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CASE REPORT

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An enlarging scaly plaque localized on the previous keloid of the chest

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Abstract

Mycosis fungoides (MF) is the most common type of cutaneous T-cell lymphoma (CTCL). It may arise rapidly in a scar or keloid, presumably due to a long-standing proliferative state or autoimmune theory. There should be a low threshold for performing a skin biopsy if unusual lesions develop at those sites.

KEYWORDS

keloid, mycosis fungoides, scar, tumor

1 | INTRODUCTION

Mycosis fungoides (MF) is the most common type of cutaneous T-cell lymphoma, which may involve the nodes, blood, and viscera. The cause of MF is unclear. Genetic and epigenetic abnormalities may contribute to the development of MF.[1] In this report, we present a case of MF with unusual findings.

2 | CASE REPORT

A 61-year-old man known for hypertension and ischemic heart disease underwent coronary artery bypass graft (CABG) surgery about 7 years ago. A keloid developed at the site of the surgery scar. It was treated successfully by monthly intralesional triamcinolone injection, for 3 months, within the last 2 years. The patient complained from an enlarging pruritic erythematous scaly plaque on his chest for 5 months. The mentioned plaque emerged after 6 months of last intralesional triamcinolone injection. His drug history was positive for losartan and atorvastatin. There was no history of fever, weight loss, or anorexia.

On physical examination, an erythematous indurated scaly plaque was localized on the center of the chest extending to the left side. The plaque had sharp borders. Some discrete crusts were obvious on the upper portion of the lesion (Figure 1). No cervical or axillary lymphadenopathy was detected. No organomegaly was found. Routine laboratory tests, chest x-ray, and chest, abdomen, and pelvic CT scans were otherwise normal.

Digital dermoscopic examination with FotoFinder HD 1000 findings were follicular keratinization and plugging, yellow dots and scales, reticulated and linear blood vessels, multiple discrete ulcers, an elongated white fibrous band (Figure 2). Considering Piccolo's and colleagues' review, these dermoscopic signs are in favor of a lymphoproliferative disorder, especially mycosis fungoides.[2]

Skin biopsy was performed, with a differential diagnosis of cutaneous T-cell lymphoma (mycosis fungoides, pagetoid reticulosis), seborrheic dermatosis, Bowen's disease, keloid, and psoriasis. The biopsy specimen revealed a lymphoproliferative disorder with epidermotropism. Atypical lymphocytes infiltrated the whole dermis. Pathology was suggestive for cutaneous mycosis fungoides. Immunohistochemical

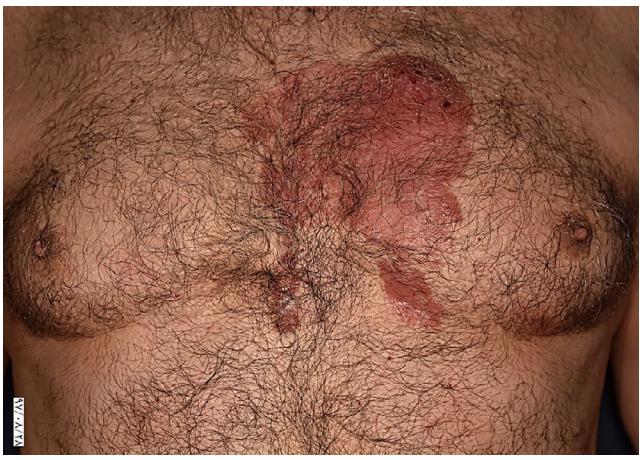

FIGURE 1 An erythematous scaly plaque with sharp borders at the site of CABG keloid extending to the left side of the chest

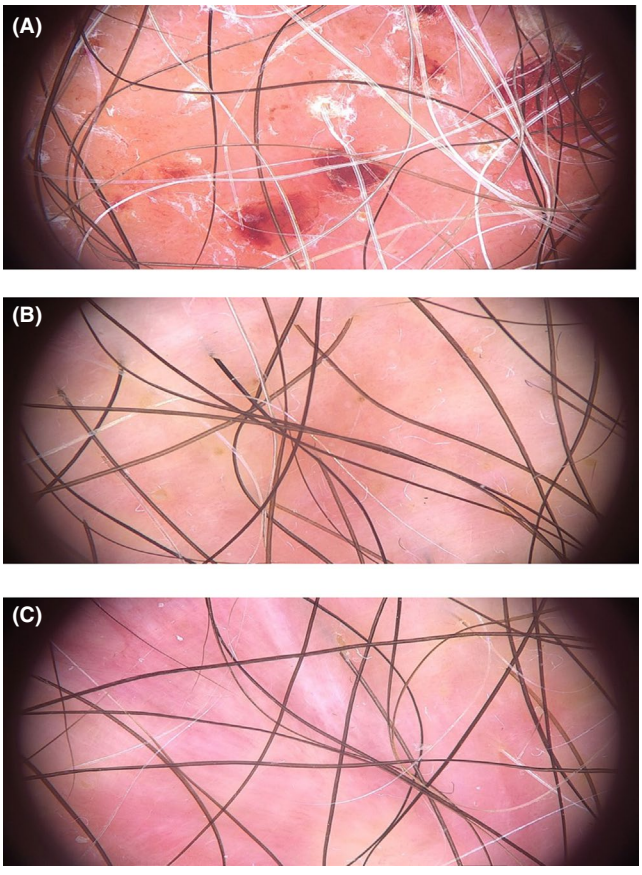

FIGURE 2 Follicular keratinization and plugging, reticulated and linear blood vessels, multiple discrete ulcers (A), yellow dots and scales (B), elongated white fibrous band (C)

analysis was positive for all CD3, CD4, CD8, and CD20 markers but negative for CD7 and CD30 (Figure 3). The important point was that lymphocytes with epidermotropism were negative for the CD20 marker. The serologic evaluation for HTLV1 was negative.

A final diagnosis of mycosis fungoides was confirmed. The patient referred for localized electron beam radiation. Unfortunately, the patient lost to follow-up, so the response to radiation therapy could not be determined.

3 | DISCUSSION

There are few reports of localized cutaneous T-cell lymphoma on skin trauma or previous surgery scars.[3-6] Huang and coworkers reported a 55-year-old man who developed zosteriform mycosis fungoides at the site of previous herpes zoster eruption.[1] They have been proposed that a Koebner-like phenomenon at a site of the skin with decreased cutaneous resistance, immunologic impairment and altered neural pathways, caused by the herpes virus may lead to a zosteriform pattern of MF.[1] A 72-year-old woman developed primary cutaneous CD4+ small-/medium-sized pleomorphic T-cell lymphoproliferative disorder at the site of a melanoma in situ excision scar which was excised 36 years ago, published by Aria et al.[4] Amoh and colleagues introduced a primary cutaneous anaplastic large cell lymphoma that developed at the site of the burning scar on the right arm of the middle-aged Japanese man.[5] Skin nodules emerged at the burning scar 4 months after the initial second-degree burning injury.[5] Paul et al[6] presented four patients who developed mycosis fungoides after a median time of 10 years of skin trauma. Exposures/ trauma included gravel embedded into the thigh of a runner, super glue inoculation of the right buttock, poison oak dermatitis of the right thigh, and a chronic hematoma of the right anterior lower extremity following a motor vehicle accident. The authors claim that MF occurrence at the sites of skin trauma after many years could be the sequence of persistent antigen stimulation that can evolve to clonal T-cell proliferation diagnosed as MF.[6] Gargallo and coworkers reported a primary cutaneous CD4+ small-/medium-sized pleomorphic T-cell lymphoproliferative disorder appearing at the scar of a biopsy of an old cutaneous lupus lesion.[3] They have been postulated that the immunologic dysregulation due to systemic lupus, the immunosuppressive effect of the medication and the proliferative stimulus due to the biopsy scar caused clonal proliferation of T cells, then may lead to the development of lymphoproliferative disorder.[3]

The pathophysiology of the appearance of lymphoproliferative disorders in scars or sites of skin trauma is unknown.[4] It has been postulated that proliferative stimuli secondary to a scar may lead to clonal proliferation and development of lymphoproliferative disorders.[6] Another hypothesis explains an autoimmune nature for cutaneous T-cell lymphoma. In this theory, skin trauma results in chronic antigen exposure and T-cell stimulation/accumulation.[4]

Phenotypic expression of CD20 in CTCL is rare according to the previous reports.[7] This phenotype aberrancy may

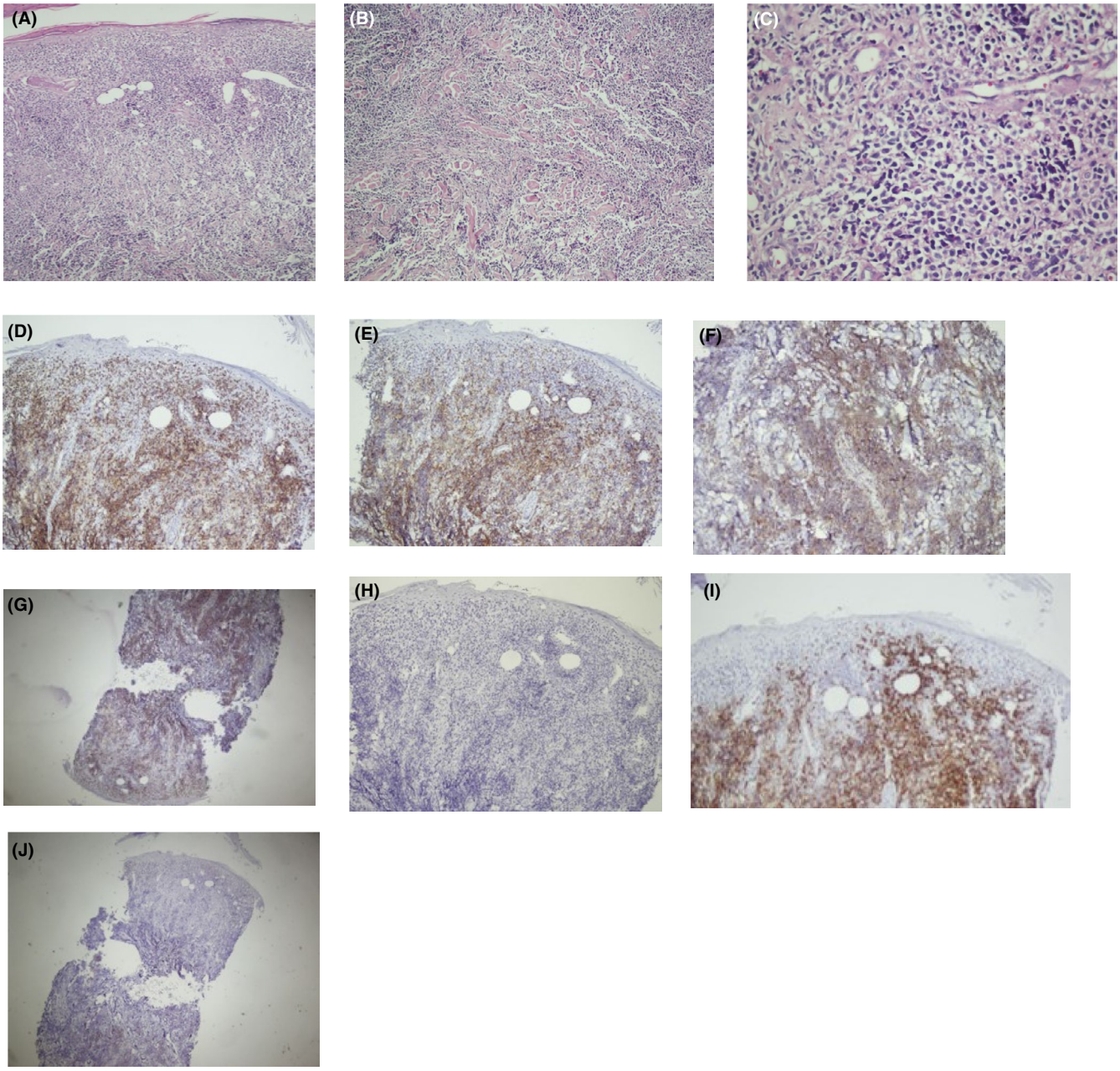

FIGURE 3 H&E staining, lymphoproliferative disorder with epidermotropism, atypical lymphocytes infiltrating the whole dermis (A). Atypical lymphocytes around collagen bundles (B). Higher magnification shows atypical lymphocyte cells with mitosis (C), CD3+ (D), CD4+ (E), CD8+ (F, G), CD7- (H), CD20+ (I), and CD30- (J)

emerge on different subtypes of T-cell lymphoma either systemic or localized disease. Today, several theories explaining the nature of the CD20 phenotype in CTCL have been introduced. The first hypothesis is that circulating CD20 positive T cells may develop neoplastic transformation. The second explanation is that sporadic reactive B-cell infiltrates may be present in the CTCL.[7,8]

The prognostic value of CD20 expression is unclear. CD20 may be a marker of B lymphocyte activation in tumor-stage MF. Only a few cases of CD20 CTCL have been treated with Rituximab with variable results.[9,10]

This report introduces a localized MF case that is extremely unusual due to localization on the previous surgical scar. Few previous reports introduce cutaneous lymphoproliferative disorders arising in a scar. The exact pathophysiology of the appearance of lymphoproliferative disorders in scars or sites of skin trauma is unclear. In our case, the keloid formation secondary to proliferative stimuli in scar successfully treated by intralesional triamcinolone. The autoimmune theory might be more helpful in explaining the relationship between CTCL and the scar. Another unusual point, in this case, is the aberrant presence of CD20

phenotype in dermal lymphocytes. Considering that epidermotropic lymphocytes could not exhibit the CD20 phenotype, it seems that reactive B-cell infiltrates may be present in this MF. CTCL may arise rapidly in scars or keloid, presumably due to a long-standing proliferative state or autoimmune theory. There should be a low threshold for performing a skin biopsy if unusual lesions develop at those sites. Further investigation is needed to explain the etiology and physiopathology of appearance lymphoproliferative disorders on the scars or skin trauma.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

ZR: wrote the initial draft of the manuscript. AHE: assisted in the preparation of the manuscript. KK-H: assisted in the preparation of the manuscript. PN: supported writing the manuscript. HM: assisted in the preparation of the manuscript. All authors: reviewed and approved the final manuscript.

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