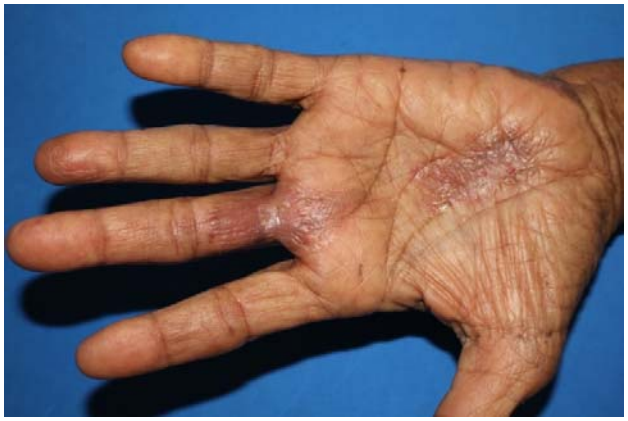
Case 13
An 84 year-old woman from Bangkok

Chief complaint: Solitary scaly hyperkeratotic erythematous plaque with minimal erosions on left palm for 1 year


(Fig. 13.1)

Present illness: 1-year history of solitary scaly hyperkeratotic erythematous plaque with minimal erosions on left palm. She had history of thorn injury at left palm but cannot related with the onset of skin lesion.

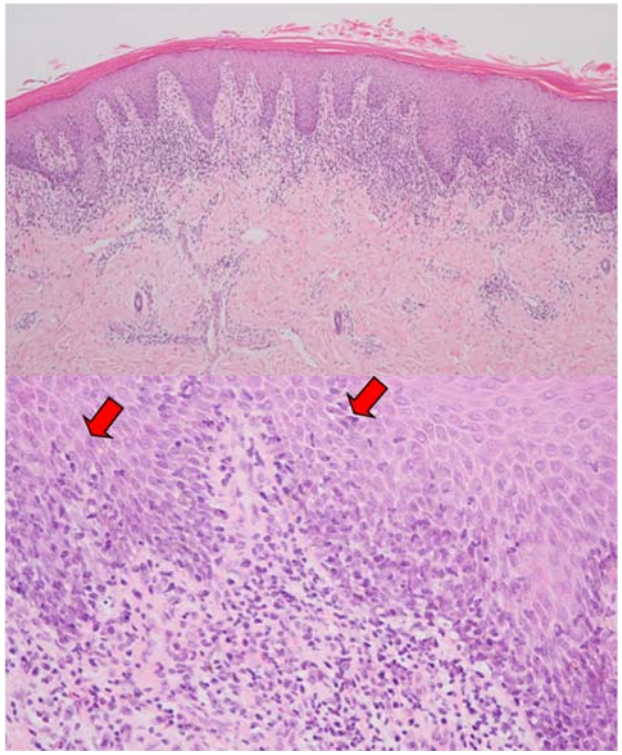
Past history: Hypertension

Family history: There was no family history of similar skin lesion.

Dermatological examination: Solitary scaly hyperkeratotic erythematous plaque with minimal erosions on left palm

Physical examination: Systemic examination other than skin revealed no abnormality (no lymphadenopathy and no hepatosplenomegaly)

Histopathology: (S16 - 40331, Left palm) (Fig. 13.2)


(Fig. 13.2)

- Epidermal hyperplasia and hyperkeratosis in associated with exocytosis of atypical lymphocytes
- Dense lichenoid cell infiltrate composed of mainly large convoluted dense nucleolar lymphocytes

Immunohistochemistry: CD3+, CD4+, CD5+, CD8-, CD20-, BF1+

T-cell receptor (TCR) gene rearrangement: Monoclonal TCR gene rearrangements
Skin culture for fungus: *Candida parapsilosis*

Skin polymerase chain reaction (PCR) for 18s ribosomal ribonucleic acid (rRNA): *Candida parapsilosis*

Skin culture for *Mycobacterium* spp., PCR for *Mycobacterium* spp., and 16s rRNA: Negative

Blood chemistry: Complete blood count, liver function test and chest X-ray revealed no abnormality, LDH 198 U/L

Diagnosis: Unilesional mycosis fungoides with cutaneous candidiasis on left palm

Treatment: Fluconazole 200mg/day for 2 weeks, then started PUVA 1-1.5 J/cm², 2 times/week for 6 months. Now, her clinical is improved, then the treatment is continued with PUVA 1.5 J/cm² once a week.

Presenter: Phatphitcha Jedee, MD

Consultant: Ploysyne Rattanakaemakorn, MD

Discussion:

Mycosis fungoides (MF) is the most common cutaneous T-cell lymphoma (CTCL) and accounts for more than 40% of all primary cutaneous lymphomas,[1] usually arising in mid-to-late adulthood (median age at diagnosis, 55–60 years) with a male predominance of 2:1.

Pathogenesis is the vast number of T cells residing in skin underlies a significant potential for neoplasia. Genetic factors, such as an individual's HLA type, may predispose some people to develop CTCL by inappropriate activation and accumulation of T cells via antigen presentation.[2] Environment may deregulate tumor suppressor or pro-oncogenic pathways include viral or other microbial pathogens (human T-cell lymphotropic virus type 1, Epstein-Barr virus [EBV], herpes simplex virus, *Staphylococcus aureus*, dermatophytes, *Mycobacterium leprae*, and *Chlamydia pneumoniae*).[3] Drug triggers (antihistamines, antiepileptics, antihypertensives, and selective serotonin reuptake inhibitors), and occupational/nutritional associations, including exposure to aromatic hydrocarbons or vitamin D deficiency, support an environmental role in the evolution of CTCL.[4-5]

Clinically, MF is categorized as being in the patch, plaque, or tumor stage, but patients may simultaneously have more than one type of lesion. Other MF variants are folliculotropic MF, hypopigmented MF, pagetoid reticulosis, granulomatous slack skin and unilesional MF (UMF).

Unilesional mycosis fungoides (UMF) is a variant characterized by a single lesion involving less than 5% of the body surface skin area.[6] Clinical feature is single erythematous plaque may occur anywhere on the body such as face, scalp,[7] breast,[8] trunk, buttock[7], groin,[9] forearm,[10] thigh,[11] leg,[8] palm[7] and sole.[1] UMF usually has an indolent course and good prognosis.[12]

Histopathology is similar to MF, there is atypical lymphoid infiltrate with epidermotropism, possible adnexal involvement.[12]

There are 3 cases report presented relationship between MF and koebner phenomenon. These 3 patients had history of stable MF

and developed recurrent MF on the trauma site area. The first, second and third patients developed recurrent MF on area of surgical excision, friction area that contact with crutch and belt, respectively.[13]

There are various therapeutic approaches to UMF, these therapeutic should be curative. Treatment modalities are radiotherapy, surgical excision, photodynamic therapy[1] and topical therapy (e.g., potent corticosteroids,[13] calcineurin inhibitors, imiquimod, carmustine, and nitrogen mustards).

In conclusion, this is a case of UMF on left palm that had history of thron injury before MF occurred and associated with fungal infection. After anti-candida treatment, she received topical PUVA and was improved after 44 treatment sessions.

References:

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