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DERMA TODAY

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Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "Derma Today" which contains latest and interesting news in the field of Dermatology.

This issue contains:

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Looking forward to your valuable feedback.

Best Regards,

Medical Team, Micro Labs Limited.

Dendritic cells may aid in distinguishing between discoid lupus erythematosus alopecia and lichen planopilaris.

Introduction:

Primary cicatricial alopecia (PCA) is a diverse set of inflammatory illnesses characterised by the fibrous tissue replacement of hair follicle components. The most prevalent causes of scarring alopecia with lymphocytic infiltration are discoid lupus erythematosus (DLE) and lichen planopilaris (LPP). [1] Patch alopecic regions with follicular plugs, perifollicular scaling, and perifollicular erythema are all symptoms of LPP. [2] The absence of follicular openings, peripilar casts and scaling, perifollicular erythema, and white cicatricial patches are all seen on trichoscopy of LPP patients. [3] Although the pathophysiology of PCA is unknown, the position of the peri-follicular inflammatory infiltrate, which in PCA is focused around the bulge region, may hold the key to understanding it. Immune privilege exists at this area as a result of multiple immunosuppressive mechanisms, such as the reduction of dendritic cell (DC) activity. Plasmacytoid DCs (pDCs), myeloid/conventional DC1 (cDC1), myeloid/conventional DC2 (cDC2), Langerhans cells (LCs), and monocyte-derived dendritic cells (MDDCs) are the five major subsets of DCs (moDCs). Immune response initiation and regulation necessitate a diverse set of mechanisms and responses. LCs are involved in both the maintenance of commensal tolerance and the responses to specific infections. Natural type I interferon (IFN type I) producers are pDCs. While cDC1 and cDC2 are critical for antigen processing and presentation, Mo-DCs are mostly active at the site of an active illness and release inflammatory cytokines. [4]

Methods:

The location of dendritic cells was mapped and quantified in a retrospective cohort study involving 51 individuals with LPP and DLE. Using particular monoclonal antibodies to specific subpopulations of dendritic cells, cell count in lesional skin was performed using immunohistochemistry. Based on histological and clinical evaluations, all patients were divided into two groups: LPP and DLE patients (Figure 1).

Results:

Both PCA groups had a similar distribution of CD123+ cells. The CD123+ cell infiltration was the smallest of all the DC subsets, and it was predominantly seen in the perifollicular area. In the epidermis, single CD123+ cells were discovered. CD11c+ cells showed a similar

pattern of distribution, with no distinction between DLE and LPP groups. In both groups, CD206+ cells were seen in the perifollicular area as well as the dermis. CD1a/CD207+ cells were typically found in the epidermis, but they were also prevalent in the hair follicles of DLE patients. The infiltration of DCs in the bulge region of the hair follicle was less, and the investigation found no changes between the two entities studied.

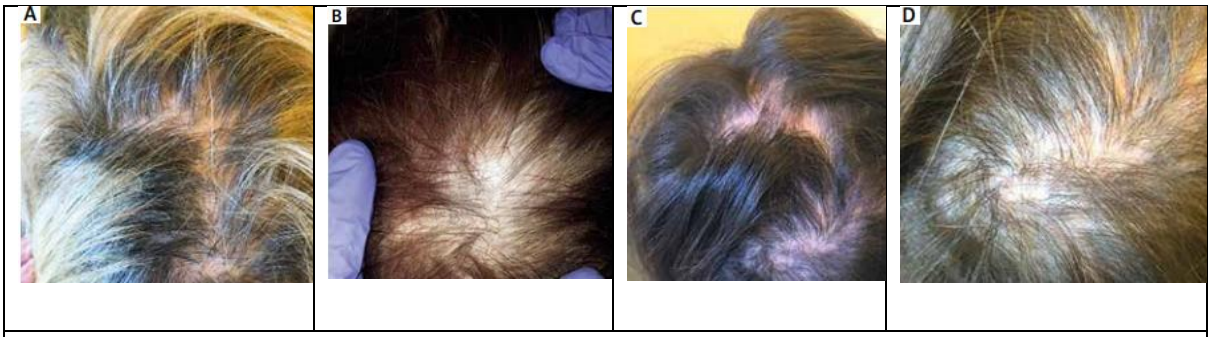

Figure 1: Clinical presentation of LPP (A, B) and DLE (C, D)

Discussion:

pDCs are the primary makers of type I interferon in response to pathogenic pathogens, and they play a critical role in innate immunity. pDCs are bone marrow-derived cells that are normally lacking in healthy skin and are initially found in reactive lymphoid organs. [4] This is the first study to reveal an increase in monocyte-derived DCs in DLE patients' lesional skin when compared to LPP. Both groups had a similar pattern of distribution. Inflammation speeds up the recruitment and activation of moDCs. As a result, they're often referred to as "inflammatory dendritic cells." Langerhans cells are specialised DCs located in the epidermis's basal and squamous layers. LCs lose their connections with the epithelial layer and move into the afferent lymphatics during inflammation. Although LCs are vital for epidermal health and commensal tolerance, they can also present mycobacterial glycolipid and trigger CD8 T lymphocytes.

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Conclusion:

In comparison to lichen planopilaris patients, practically all subpopulations of dendritic cells were substantially expressed in lesional skin of discoid lupus erythematosus patients in the

present study. Dendritic cells could be used as an additional indication in the differential diagnosis of PCA in light of this result.

Dendritic cells could be used as an additional indication in the differential diagnosis of PCA in light of this finding.

Reference:

1. Kłosowicz AD, Pastuszczak M, Dyduch G, Łukasik A, Kozicka K, Englert K, Kaleta K, Wojas-Pelc A. Dendritic cells may help differentiate discoid lupus erythematosus alopecia from lichen planopilaris. Advances in Dermatology and Allergology/Postępy Dermatologii i Alergologii. 2022 Apr;39(2):298.
2. Errichetti E, Figini M, Croatto M, Stinco G. Therapeutic management of classic lichen planopilaris: a systematic review. Clinical, cosmetic and investigational dermatology. 2018;11:91.
3. Waśkiel A, Rakowska A, Sikora M, Olszewska M, Rudnicka L. Trichoscopy in lichen planopilaris: an update. Dermatology Review/Przegląd Dermatologiczny. 2018;105(1):63-75.
4. Collin M, Bigley V. Human dendritic cell subsets: an update. Immunology. 2018 May;154(1):3-20.

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A SEER Population-Based Study of Second Primary Malignancies Following Cutaneous Angiosarcoma

Introduction:

Conic and colleagues use the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database to conduct an important epidemiologic analysis of cutaneous angiosarcomas (CAS). [1] Based on age, disease severity, and treatment, they reflect incidence rates and survival outcomes. However, after initial CAS, the likelihood of secondary primary cancers is a relevant epidemiologic issue (SPMs). SPMs may have an impact on surveillance and prognosis following a CAS diagnosis. [2]

Methods:

The Surveillance, Epidemiology, and End Results (SEER) database of the National Cancer Institute was used in this investigation. Initial CAS cases were extracted and examined using standardised incidence ratios (SIR) and excess absolute risks (EAR) for SPMs in comparison to a control population matched by sex, race (white/unknown, black, other), age group (5-year interval), and calendar year (5-year interval). The EAR was calculated for per 10,000 people. A statistically significant P-value of 0.05 was used.

Results:

Patients with CAS had a higher rate of new malignancies when compared to a matched group from the general population. Soft tissue malignancies and non-epithelial skin malignancies other than melanoma/basal cell/squamous cell carcinoma were shown to be more common.

Discussion:

The mechanisms behind CAS's relationship with SPMs are still unknown. Radiation therapy, as well as exposure to arsenic, polyvinyl chloride, and viral hepatitis, are recognised to be underlying risk factors for CAS.

These are, however, common risk factors for a variety of different malignancies. It's worth noting that in our study, non-epithelial skin malignancies had the highest incidence of SPMs in patients with initial CAS. We believe that treatment-related complications, such as chemotherapy and radiation, may play a role in increased risk. Surgical excision appears to be the cornerstone of treatment for CAS, and some patients also get adjuvant radiation therapy or chemotherapy. 4 SPMs may be induced by these indefinite treatments. [3]

Immunotherapy/immunemodulating medicines have recently been utilised to treat CAS; nevertheless, immunosuppression may raise the incidence of SPMs. [3] Furthermore, angiosarcomas may be linked to certain mutations and pathophysiologic pathways, according

to the literature (e.g. KDR, TP53, and PIK3CA. PIK3CA). [4] These pathways are likely to be involved in SPMs in individuals with first CAS because they act as a hereditary predisposition that can become unmasked, especially when the SPM is vascular in nature. Collaboration with translational researchers to elucidate the genetic sequencing and molecular characterization of malignant tissue in these individuals via biobanking might be beneficial in future investigations. This may also lead to more tailored therapy options for the treatment of this difficult cancer. For instance, the Angiosarcoma Project is an example of such a project. [4]

Conclusion:

SPMs have a variety of causes, including genetic vulnerability, treatment-related complications, lifestyle/environmental variables, and shared risk factors. SPMs can be induced by long-term therapy. Immunotherapy/immune-modulating medicines have recently been utilised to treat CAS4; but, immunosuppression may raise the risk of SPMs. SPMs could potentially be explained by a common aetiology (i.e. blood vessel or soft tissue-derived neoplasms). Importantly, even after complete resection, CAS is linked to a high rate of recurrence.

Patients who have been diagnosed with CAS should be followed up on not only for recurrence but also for SPMs after their original diagnosis and therapy regrowth patterns in AA patches are influenced by treatment techniques and patch size.

Reference:

1. Ramachandran V, Loya A, Phan K. Second Primary Malignancies After Initial Cutaneous Angiosarcoma: A SEER population-Based Study. SKIN The Journal of Cutaneous Medicine. 2022 May 6;6(3):229-33.
2. Conic RR, Damiani G, Frigerio A, Tsai S, Bragazzi NL, Chu TW, Mesinkovska NA, Koyfman SA, Joshi NP, Budd GT, Vidimos A. Incidence and outcomes of cutaneous angiosarcoma: a SEER population-based study. Journal of the American Academy of Dermatology. 2020 Sep 1;83(3):809-16.
3. Ishida Y, Otsuka A, Kabashima K. Cutaneous angiosarcoma: update on biology and latest treatment. Current Opinion in Oncology. 2018 Mar;30(2):107.
4. Painter CA, Jain E, Tomson BN, Dunphy M, Stoddard RE, Thomas BS, Damon AL, Shah S, Kim D, Gómez Tejeda Zañudo J, Hornick JL. The Angiosarcoma Project: enabling genomic and clinical discoveries in a rare cancer through patient-partnered research. Nature medicine. 2020 Feb;26(2):181-7.

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Outcomes of Mohs Micrographic Surgery for Basal Cell Carcinoma of the Distal Third of the Nose

Introduction:

Basal cell carcinoma (BCC) on aesthetically sensitive areas such as the nose is best treated with Mohs micrographic surgery (MMS). It achieves the highest tumour cure rates by ensuring perfect margin control and preserving as much tissue as possible. [1] The Mohs surgeon must choose the surgical repair that will result in the greatest surgical and cosmetic results for the patient once the tumour has been entirely removed. [2]

Many surgeons classify nasal deformities into two groups: those that affect the top two-thirds of the nose and those that affect the lower third. While a primary closure is frequently thought to be the simplest repair, tissue-limited locations such as the supratip, infratip, and alae of the nose make this difficult, if not impossible. Many surgeons would use a local skin flap, full thickness skin graft (FTSG), or leave the defect to repair by secondary intention due to the cartilaginous distal part of the nose's lack of flexibility (SI). Each surgeon has a personal preference, but there is no mechanism to aid in the decision-making process. In this study, we used an objective scar measuring following MMS on the distal third of the nose to compare surgical results of a local skin flap, FTSG, and SI.

Methods:

A retrospective chart assessment of 66 MMS deformities performed by a single surgeon on the distal part of the nose was completed. The Vancouver Scar Scale was used to collect and measure clinical photos of MMS abnormalities and postoperative scars at six months (VSS). The connection between the key predictor variables and VSS was determined using Pearson Chi-square and Fisher's Exact tests. Two physicians evaluated and scored clinical photos of MMS deficiencies and surgical scars at one week and six months (Figure 1) using the VSS.

Results:

Of the 66 patients retained, 52 had a low VSS (77.61%), 11 had a medium VSS (16.42%), and three had a high VSS (3 %). (4.48 %). Thirty-two patients with low VSS were treated with a local flap (76.92%), nine with FTSG (17.31%), and three with SI (5.77 %). Two of the 11 patients with medium VSS received a local flap (18.15%), nine received FTSG (81.82%), and zero received SI. Zero of the three patients with a high VSS had a local flap or SI, but all three had FTSG (100 %). Bivariate analysis revealed that the type of repair used

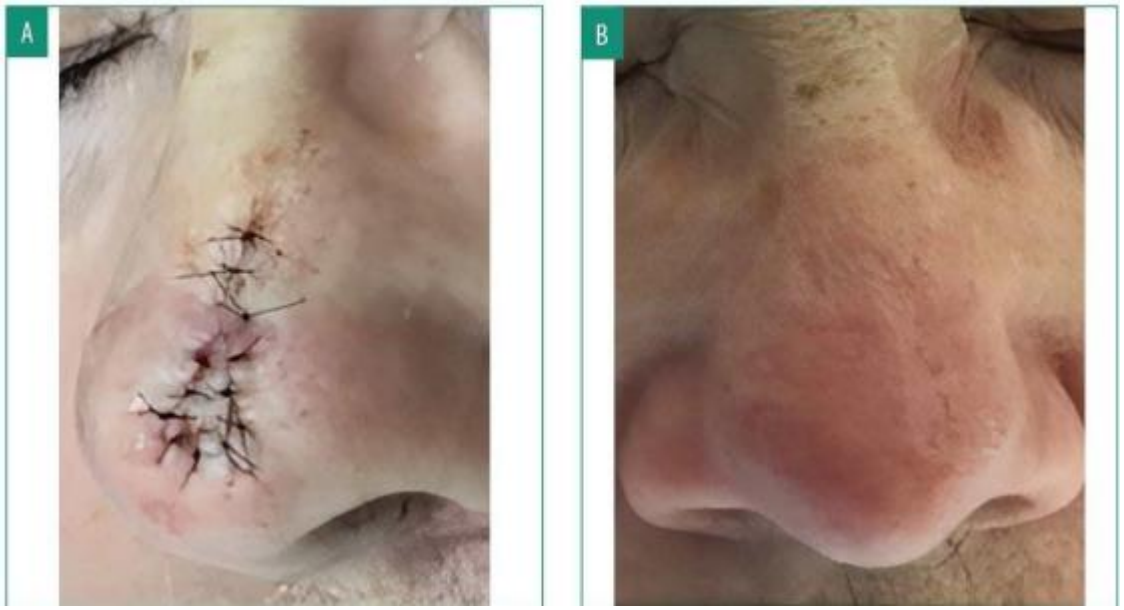

Figure 1: A patient who underwent local skin flap imaged immediately after repair (A) and at 6 months follow up (B)

was linked to VSS at six months, with patients who received a local skin flap having better outcomes.

Discussion:

BCC is the most prevalent type of cutaneous cancer, and it affects mostly White people in sun-exposed areas like the face. This study mirrored established epidemiologic trends of BCC histological subtypes, with nodular being the most frequent (48 % in present study). Infiltrative was the second most prevalent subtype in our cohort (23.38 %). While infiltrative BCC patterns may occasionally result in deeper final flaws, the relationship between defect depth and VSS reported above was not statistically significant. Up to 40% of BCCs might have multiple histological subtypes; in this analysis, 16.88 % of the patients had mixed histology of BCC. Finally, 11.69 % of the participants in this study showed a superficial pattern. [3]

The VSS of all three patients who were treated by SI was determined to be low (100%); none had a medium or high VSS. Two of the flaws were on the alar crease and one was on the supratip, according to the patient concerns of these three patients who cured via SI. This is in line with current surgical protocols, which allow for SI in concave areas like the alar crease. It's also worth mentioning that none of the three individuals had full-thickness abnormalities; just the dermis and subcutaneous tissue were affected. This supports the idea that SI should only be used for superficial defects in the distal portion of the nose, and that full-thickness

surgical defects should be treated with a surgical closure. While reduced VSS was attained in individuals who received SI, it is also important to note the cohort's small sample size. Other studies have found similar modest numbers of individuals who underwent SI. [4] While reduced VSS was attained in individuals who received SI, it is also important to note the cohort's small sample size. Other studies have found similar modest numbers of individuals who underwent SI.

Conclusion:

These data suggest that, as compared to FTSG or SI, local skin flaps may result in a reduced VSS at six months, thereby providing superior surgical outcomes in the treatment of BCC on the distal part of the nose.

According to the results, local skin flaps may result in a reduced VSS at six months when compared to FTSG or SI, suggesting that they may provide better surgical outcomes in the treatment of BCC on the distal part of the nose.

Reference:

1. Cameron MC, Lee E, Hibler BP, Barker CA, Mori S, Cordova M, Nehal KS, Rossi AM. Basal cell carcinoma: Epidemiology; pathophysiology; clinical and histological subtypes; and disease associations. Journal of the American Academy of Dermatology. 2019 Feb 1;80(2):303-17.
2. Liu KY, Silvestri B, Marquez J, Huston TL. Secondary intention healing after mohs surgical excision as an alternative to surgical repair: evaluation of wound characteristics and esthetic outcomes. Annals of Plastic Surgery. 2020 Jul 1;85(S1):S28-32.
3. Cameron MC, Lee E, Hibler BP, Giordano CN, Barker CA, Mori S, Cordova M, Nehal KS, Rossi AM. Basal cell carcinoma: Contemporary approaches to diagnosis, treatment, and prevention. Journal of the American Academy of Dermatology. 2019 Feb 1;80(2):321-39.
4. Hill D, Kim K, Mansouri B, Sager L, Housewright C. Quantity and characteristics of flap or graft repairs for skin cancer on the nose or ears: a comparison between Mohs micrographic surgery and plastic surgery. Cutis. 2019 May 1;103(5):284-7.

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Lichen Amyloidosis on the Scalp: A Case Report

Introduction:

PCA (primary cutaneous amyloidosis) is a common skin disorder marked by amyloid deposition in the dermis without systemic involvement. [1] PCA has several subtypes, including lichen amyloidosis (LA), macular amyloidosis (MA), and nodular amyloidosis (rarely) (NA). LA is defined by the appearance of many pruritic, hyperkeratotic, lichenoid papules on the pretibial areas, as well as the arms and thighs on rare occasions. Scalp involvement, on the other hand, has been recorded infrequently. [2] A patient with LA lesions that confined the scalp skin was given in this study.

Case Report:

For one year, a 64-year-old Chinese man had several dark-brown papules on his head. The lesions were asymptomatic at first, but as time went on, they grew in quantity. He had been diagnosed with androgenetic alopecia (AGA) for a decade and had received no treatment other than a one-month course of topical 5 percent minoxidil. The patient was otherwise in good health and had no family history of any kind. Over the crown and vertex area of the scalp, physical examination revealed distinct 1- to 3-mm flat-topped reddish papules (Figure 1A). Dermoscopy revealed a whitish scar-like centre surrounded by brownish spots or brown fine streaks extending from the centre, set against a background of classic AGA hair and scalp changes (Figure 1B).

Deposition of pink amorphous material in the papillary dermis was found histologically, along with scattered melanophages in the enlarged dermal papillae (Figure 1C). Congo red staining was used to highlight the deposits (Figure 1D). He was finally diagnosed with LA, and after two months of treatment with clobetasol propionate/all-trans retinoic acid combination ointment, he saw a considerable improvement in the look of his lesions with no side effects.

Discussion:

The extensor surfaces of the extremities are the most typical site of LA. Involvement of the scalp is uncommonly described in the literature. A patient with papules extending from the left frontal hairline to the forehead was described in a study. A patient with LA covering the middle area of the anterior scalp and lower limbs was documented in another investigation, and both patients complained of itching. A case of NA was documented in a female patient with severe, ulcerated alopecic nodules on the vertex of the head, according to a study.

Because of AGA, the patient in this trial denied itching and taking any routine medicine or therapy other than topical minoxidil. The lesions were thought to be caused by long-term sun exposure of the scalp due to severe alopecia, according to the study. One of the underlying reasons of amyloidosis cutis dyschromica, an atypical clinical variation of PCA, has been suggested to be sun exposure. Lichen planopilaris, seborrheic dermatitis, verruca plana, and epidermodysplasia verruciformis are among the clinical differential diagnoses in this case.

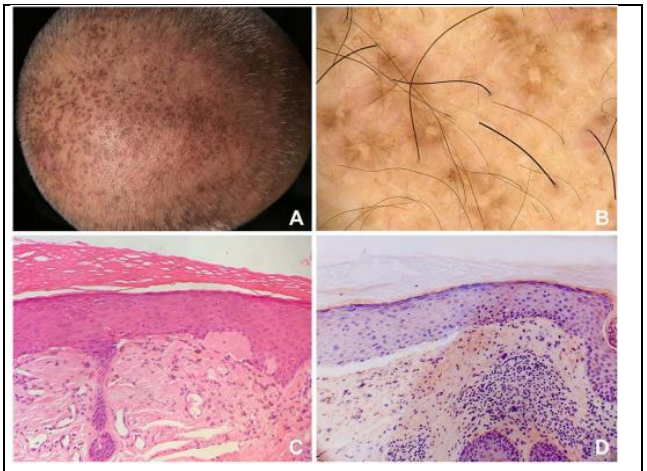

Figure 1 Multiple hyperpigmented maculopapules on the scalp (A). Dermoscopic images showed whitish scar-like centre surrounded by brown streaks (B) (30×). Hyperkeratosis, deposits of amyloid in the dermal papillae and melanophages within the papillary dermis (C) (H&E, 100×). Staining of the eosinophilic material in the papillary dermis (D) (Congo red, 100×).

Dermoscopy may be useful in confirming clinical suspicions of PCA prior to skin biopsy. LA's features are still being researched. Current thinking holds that LA has white central hubs or whitish scar-like centres (corresponding to amyloid deposits or hyperkeratosis with acanthosis in pathology) surrounded by grey brown pigmentation, primarily in the form of dots, globules, or peppering (corresponding to pigment incontinence and basal hyperpigmentation), which is referred to as a "two-zone pattern."

Patients with PCA have been treated with a variety of modalities, including topical steroids, calcineurin inhibitors, systemic antihistamines, retinoids, and various laser therapies. 1 However, there is no cure as of yet. Emerging biological agents and small molecule agents, such as dupilumab, IL-31, or JAK inhibitors, could be potential PCA therapies, particularly for generalised type.

Conclusion:

Primary cutaneous amyloidosis is a relatively common skin disorder characterised by extracellular deposition of amyloid materials caused by protein degradation in the skin. Aside from typical clinical manifestations, some forms of PCA have an unusual distribution, such as lichenoid papules on the scalp in this case. The study highlighted dermatologists' awareness of atypical morphology or location of PCA, and it suggests that dermoscopy could be a useful tool in the disease's diagnosis.

Lichen amyloidosis (LA) is a type of primary cutaneous amyloidosis that manifests as discrete, lichenoid papules with itching on the extensor surfaces of the extremities. The involvement of the scalp is rarely reported in the literature. A case of LA over the crown and vertex areas of the scalp was reported in this study. The lesions improved significantly after 2 months of topical clobetasol propionate/all-trans retinoic acid compound ointment treatment.

Reference:

1. Hamie L, Haddad I, Nasser N, Kurban M, Abbas O. Primary localized cutaneous amyloidosis of keratinocyte origin: an update with emphasis on atypical clinical variants. American Journal of Clinical Dermatology. 2021 Sep;22(5):667-80.
2. Kim Y, Ioffreda MD, Chung CG. Lichen amyloidosis of the scalp and forehead. Dermatology Online Journal. 2017;23(11).
3. Bellinato F, Rosina P, Sina S, Girolomoni G. Primary nodular localized cutaneous amyloidosis of the scalp associated with systemic lupus erythematosus. Turkish Journal of Rheumatology. 2022;37(1):145-7.
4. Behera B, Kumari R, Mohan Thappa D, Gochhait D, Hanuman Srinivas B, Ayyanar P. Dermoscopic features of primary cutaneous amyloidosis in skin of colour: A retrospective analysis of 48 patients from South India. Australasian Journal of Dermatology. 2021 Aug;62(3):370-4.
5. Chen J, Yang B. Tofacitinib for the treatment of primary cutaneous amyloidosis: a case report. Dermatologic Therapy. 2022 Apr;35(4):e15312.