DPU

DEPARTMENT OF DERMATOLOGY

1.	Bullous Pemphigoid: A Case Series	Dr. Vidhi Malu
2.	Non-invasive Dermatological Modalities for Clinical Examination	Dr. Shubhangi Gupta
3.	Fractional Micro Needling Radiofrequency Treatment of Acne Scars	Dr. Nishtha Mishra
4.	SIRT1, Vitiligo and Skin Cancer	Dr. Anushka Agarwal

DPU

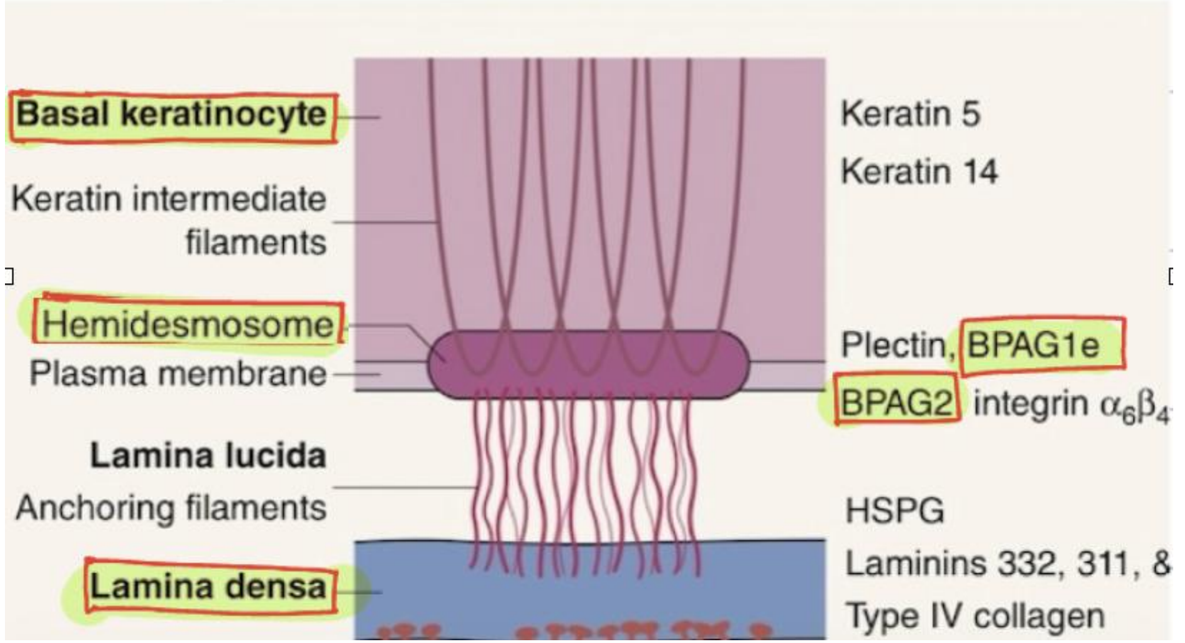
Bullous Pemphigoid: A Case Series

Dr Vidhi Malu

Introduction

- BP is the most common autoimmune subepidermal blistering disease, typically affecting the extremely aged.
- However, there is lot of heterogeneity in its:-
  - clinical presentation,
  - variants,
  - location,
  - age of onset and
  - concurrent diseases

- Till 1953, all blistering diseases were grouped under Pemphigus (Gk:“pemphix”- blister) , when Lever first distinguished bullous pemphigoid from pemphigus—blisters being subepidermal in the former unlike intraepidermal in the latter.



- A decade later, Jordon and Beutner identified the hemidesmosomal antigens (BPAg1 and BPAg2).

- Hemidesmosomes are multi-protein complexes that connect the basal keratinocytes to the lamina densa.

Case 1

42 Y/ M

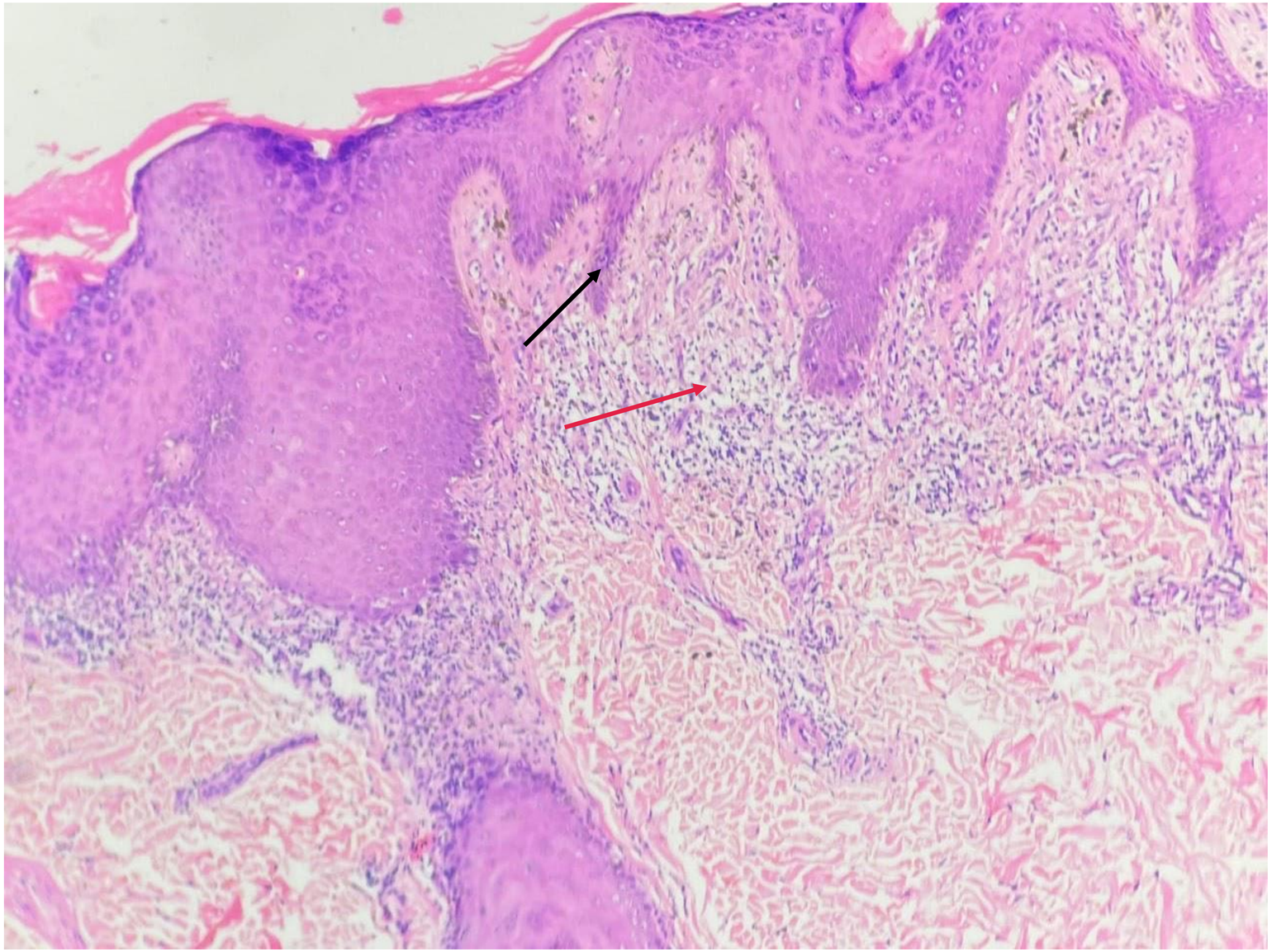
2014 → 2019 → 2024

- Pruritic papular eruptions on limbs
- LP diagnosed clinically
- By a private practitioner
- Rx- oral and topical corticosteroids
- Partial relief of symptoms

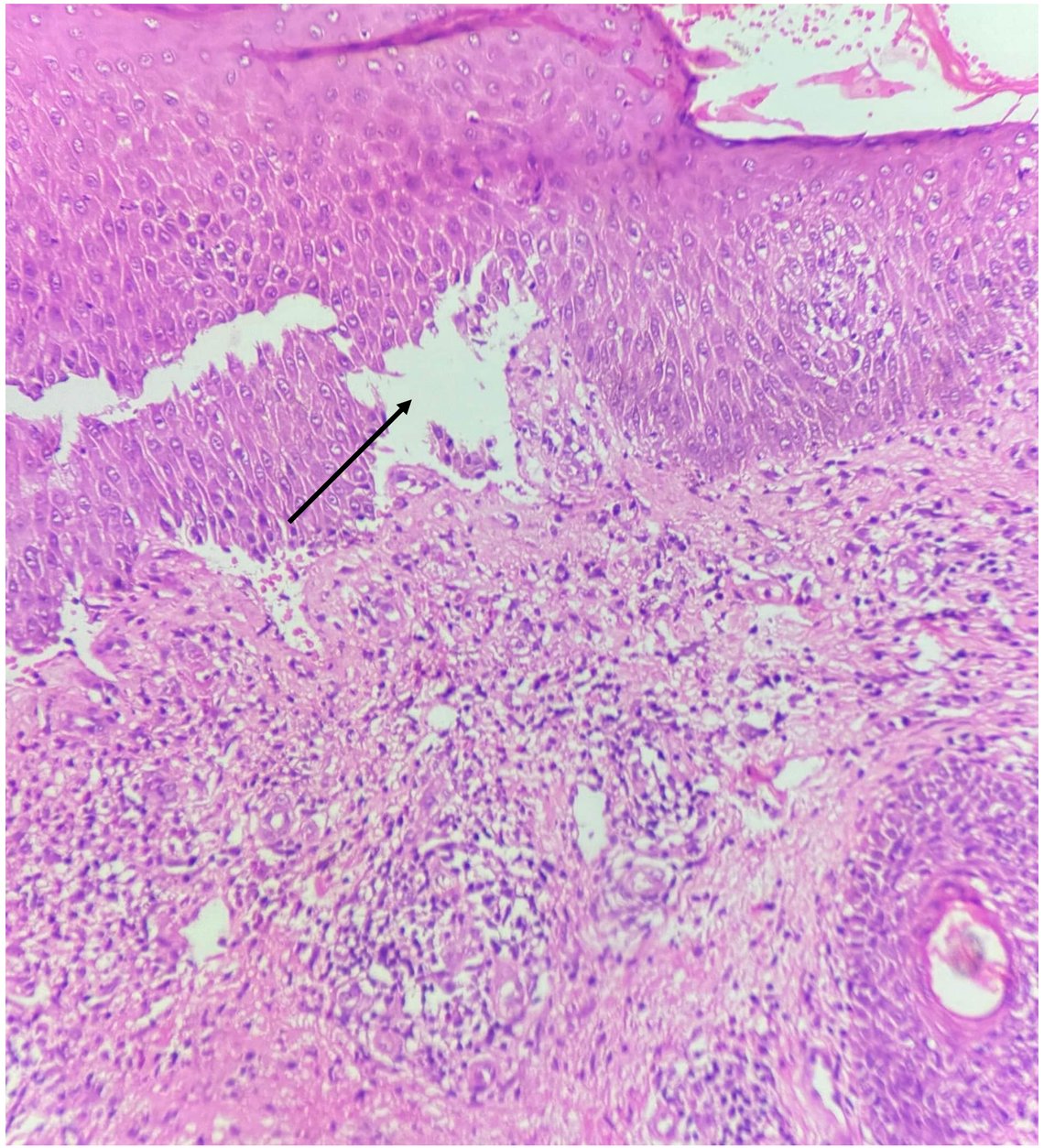
- Oral ulcers

- Biopsies performed -leg plaque
  - trunk bulla
- Anti BPAg 180 antibodies: 59.97 RU/ml
- Serum IgE : 4361 IU/ml

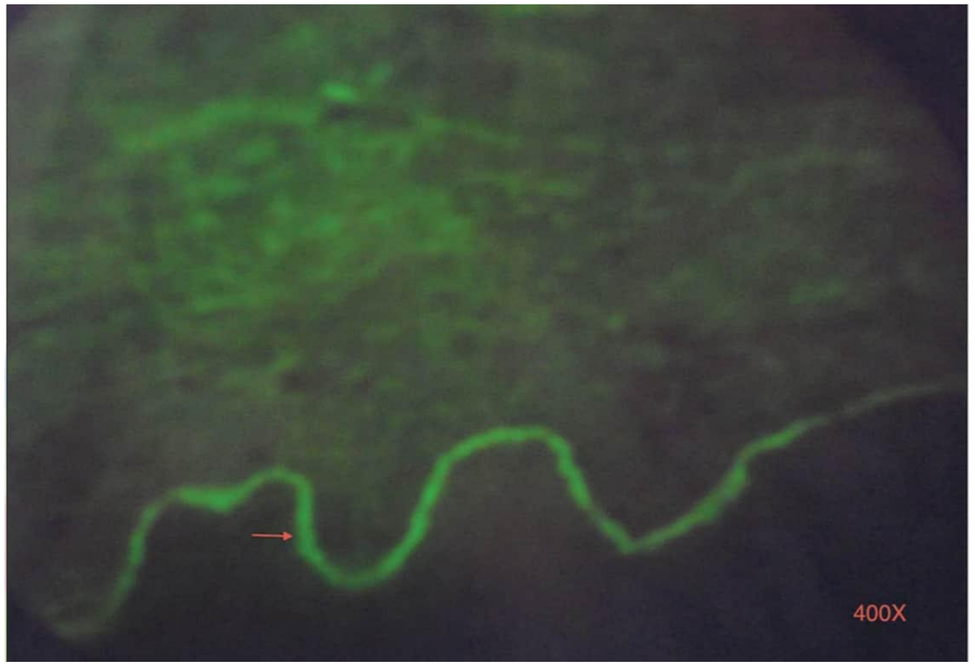
Biopsy - leg plaque - LP



Biopsy from bulla- BP



Direct immunofluorescence



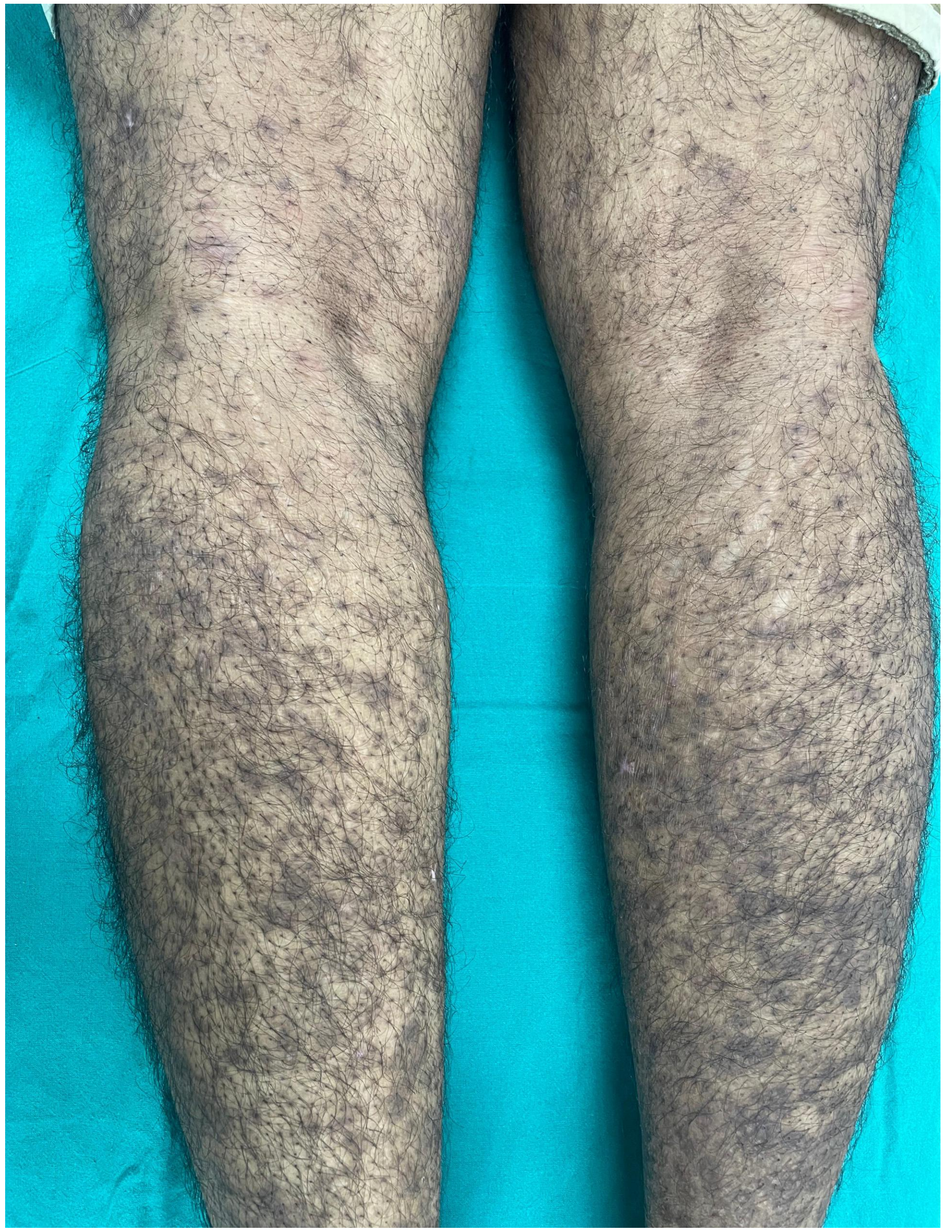
- This case showing concurrence of LP and BP is an example of overlap between the two diseases called Lichen planus pemphigoides.
- Lesions of the two can be independent as in the index case or as bullae over LP papule.
- In LPP, autoantibodies target the BPAg2 antigen, like in BP, but has a younger age of onset
- Pathogenesis: LP damaging basal layer ⟶ exposing antigens ⟶ causing epitope spreading ⟶ autoantibody formation ⟶ subepidermal bullae.

Treatment

- Oral betamethasone pulse (twice weekly)
- Inj omalizumab (monoclonal anti-IgE) 300mg s/c monthly.
- Cap acitretin (retinoid) 25mg OD
- Topical clobetasol 0.05% cream.
- Marked clinical improvement

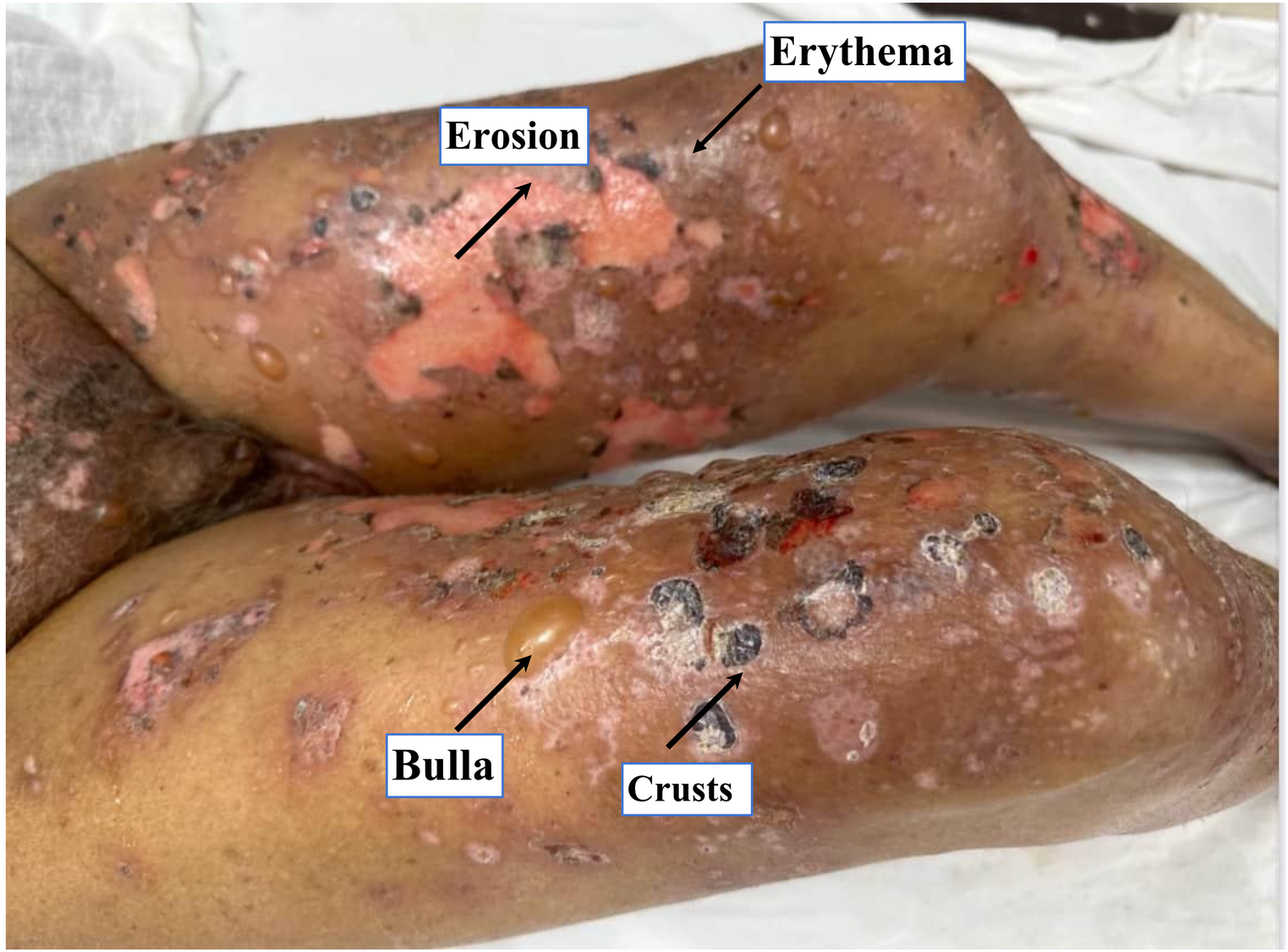
} 3 months

Post treatment



Case 2

81 Y/M



15 Y

HTN

DM

IHD

Mar 2024

Torsemide

Linagliptin

May 2024

- Red, raised itchy papules

Non- bullous prodrome of BP

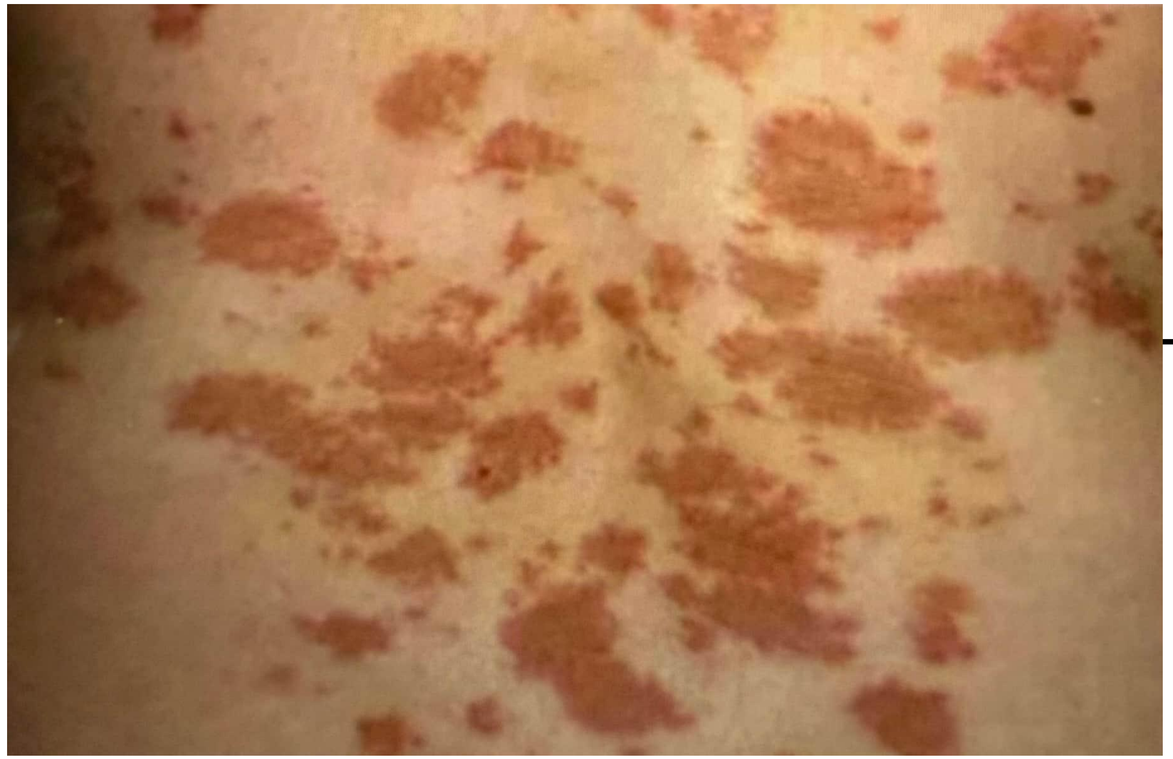
Nov 2024

- Tense blisters
- Erosions
- Crusts
- Clinidipine

and insulin introduced

Dec 2024

Significant improvement



Skin biopsy and DIF s/o BP

Rx on injectable and topical corticosteroids

Post treatment


12:54


12:54

Drug-induced bullous pemphigoid

- Loop diuretics (e.g, torsemide , furosemide)
- DPP-4 inhibitors (linagliptin , vildagliptin)
- Immune checkpoint inhibitors (nivolumab, pembrolizumab)
- TNF inhibitors
- ACE inhibitors
- Antibiotics (amoxicillin, ciprofloxacin) ,
- NSAIDs

Proposed mechanisms of drug-induced BP

- DPP-4 inhibitors alter the immune response by decreasing CD26 expression on T-cells .
- HLA-DQB1*03:01 allele in Japanese may increase risk for DPP-4i-induced BP.
- Loop diuretics- Alter hemidesmosomal proteins, exposing epitopes that trigger BP

Case 3

42 Y/F

2017 June

2024 Dec

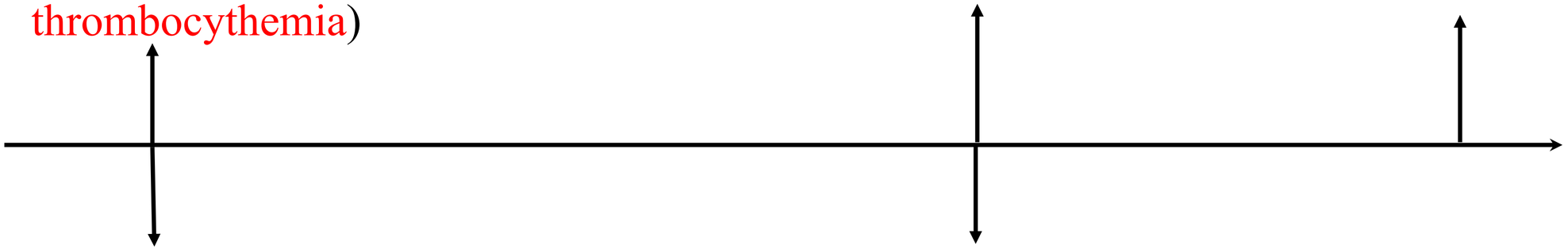
2025 Jan

Primary myeloproliferative neoplasm

(Essential thrombocythemia)

Biopsy- Bullous pemphigoid

Improvement



- Bone marrow aspirate- hypercellular marrow
- PLT - 814000
- Gene mutation study

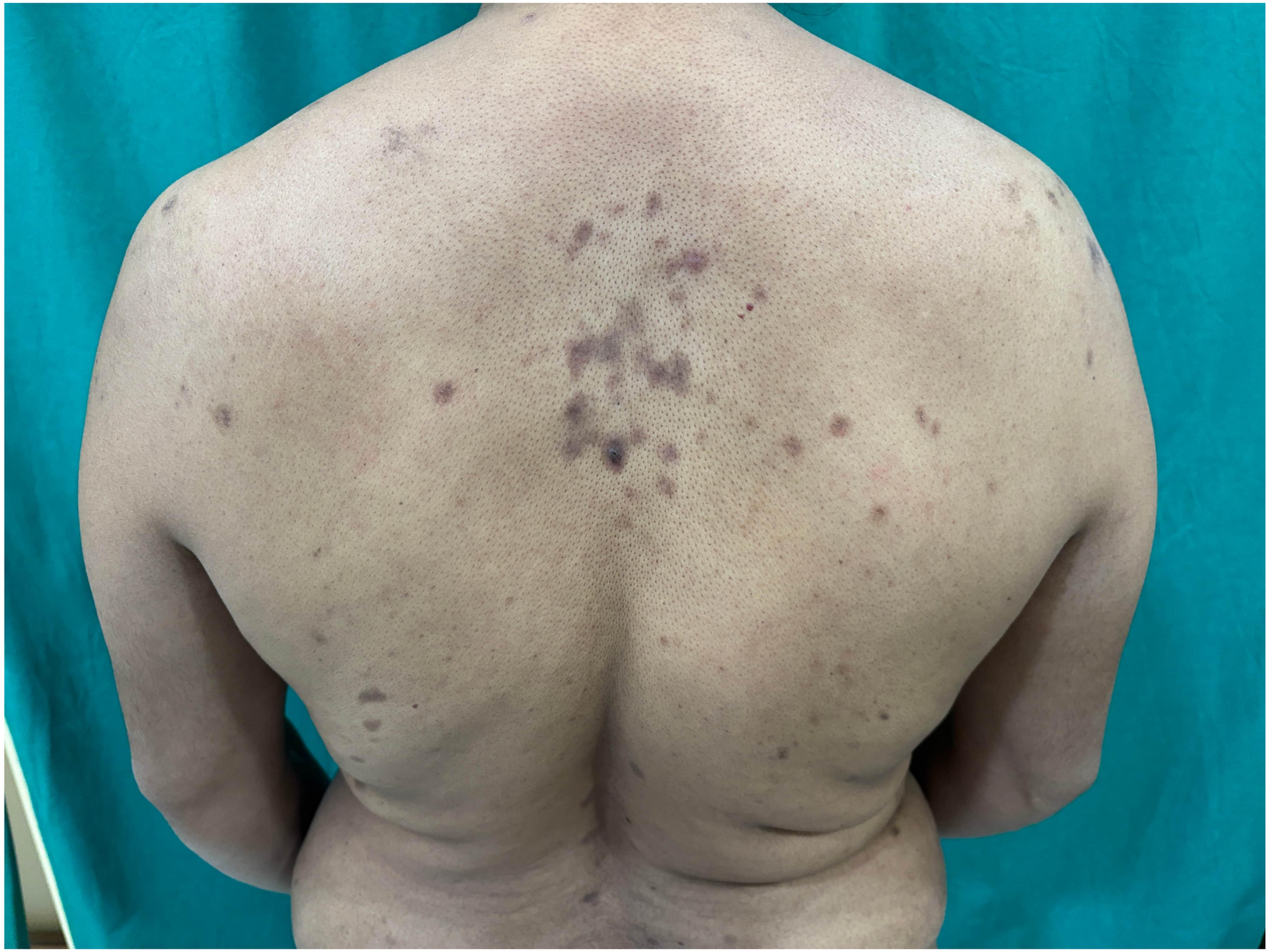
  Jak2V67f +ve

Rx

Systemic corticosteroids

Topical high-potency corticosteroids

Post treatment



BP with essential thrombocythemia (MPN) -a rare association

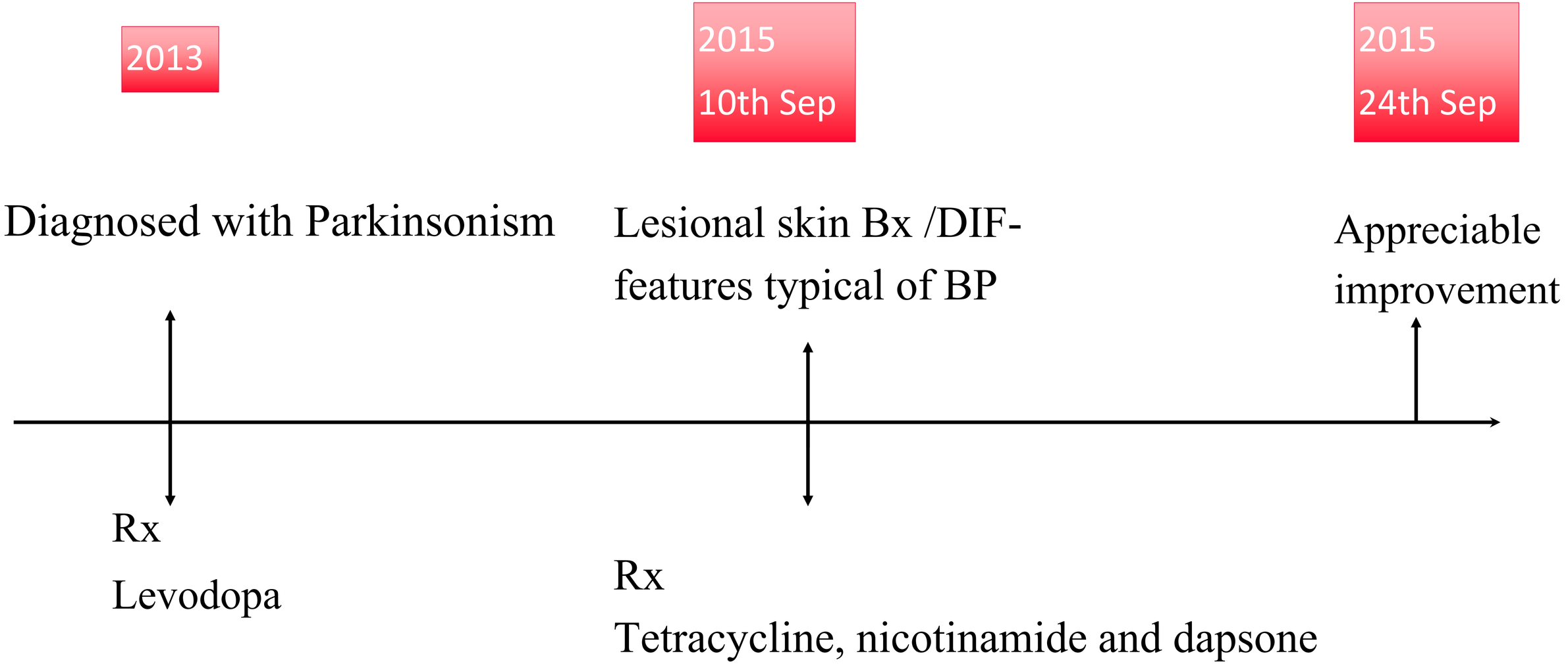
- BP is commonly linked to hematological malignancies:
  - Acute myeloid leukaemia
  - Non- Hodgkin lymphoma
  - Mycosis fungoides
  - Chronic lymphocytic leukemia
  - Multiple myeloma
  - Hypereosinophilic syndrome

Pathophysiology :

- Chronic inflammation and release of cytokines and interleukins may trigger BP.

Case 4

91/F


2013
2015
10th Sep
2015
24th Sep
Diagnosed with Parkinsonism
Lesional skin Bx /DIF-
features typical of BP
Appreciable
improvement
Rx
Levodopa
Rx
Tetracycline, nicotinamide and dapsone

Dyshidrosiform pemphigoid associated with neurological disorders

- Neurological disorders (stroke, dementia, Parkinsonism, epilepsy, multiple sclerosis) expose BPAg1-n, a spliced form of BPAg1-e, that stabilises the sensory neuron cytoskeleton.

  A large UK based study (n=863) of such cases lasting over a year found a 21% prevalence of BP. Autoantibodies generated may cross-react with BPAg1-e, leading to BP.
- The first case of dyshidrosiform pemphigoid caused by Parkinsonism in India was reported in 2016.

Case Report

Dyshidrosiform pemphigoid with Parkinsonism in a nonagenarian Maharashtrian female

Behlim T, Sharma YK, Chaudhari ND, Dash K. Dyshidrosiform pemphigoid with Parkinsonism in a nonagenarian Maharashtrian female. Indian Dermatology Online Journal. 2014 Oct 1;5(4):482-4.

Localised BP

- Sites involved: Pretibial, flexures, genitals, umbilicus, stomal, etc
- Seen in about 5-30% of the patients with BP.

Pretibial

Stomal

Radiation aggravated pemphigoid

Pemphigoid gestationis

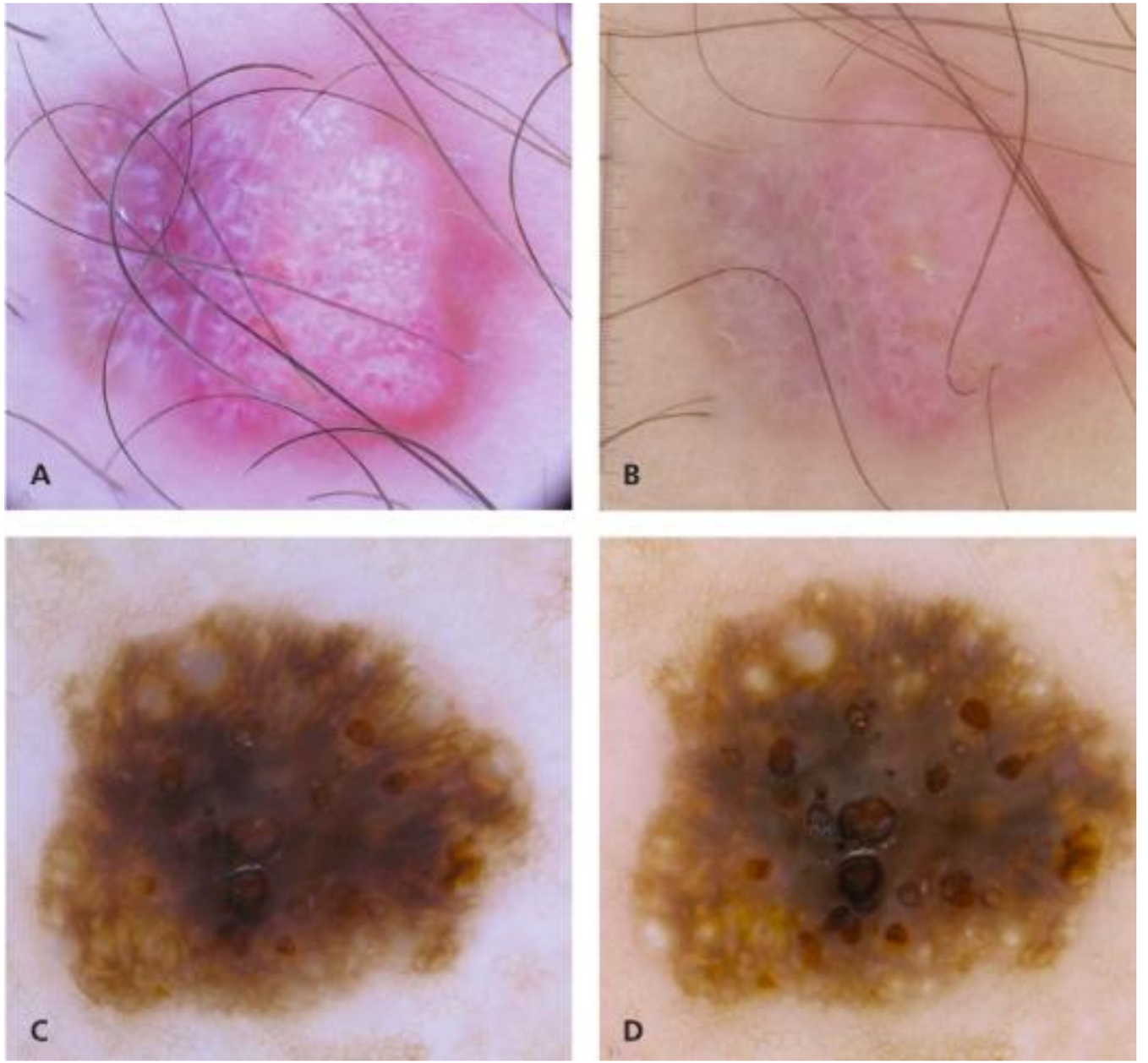
Childhood BP


Thank You

Non-invasive dermatological modalities for clinical examination

-Dr. Shubhangi Gupta

Dermoscopy



What is dermoscopy?

- Technique to visualize structure that cannot be seen with the naked eye
- Applications-
- Trichoscopy
- Mucoscopy
- Nail fold capillaroscopy
- Entomodermatoscopy
- Inflammoscopy

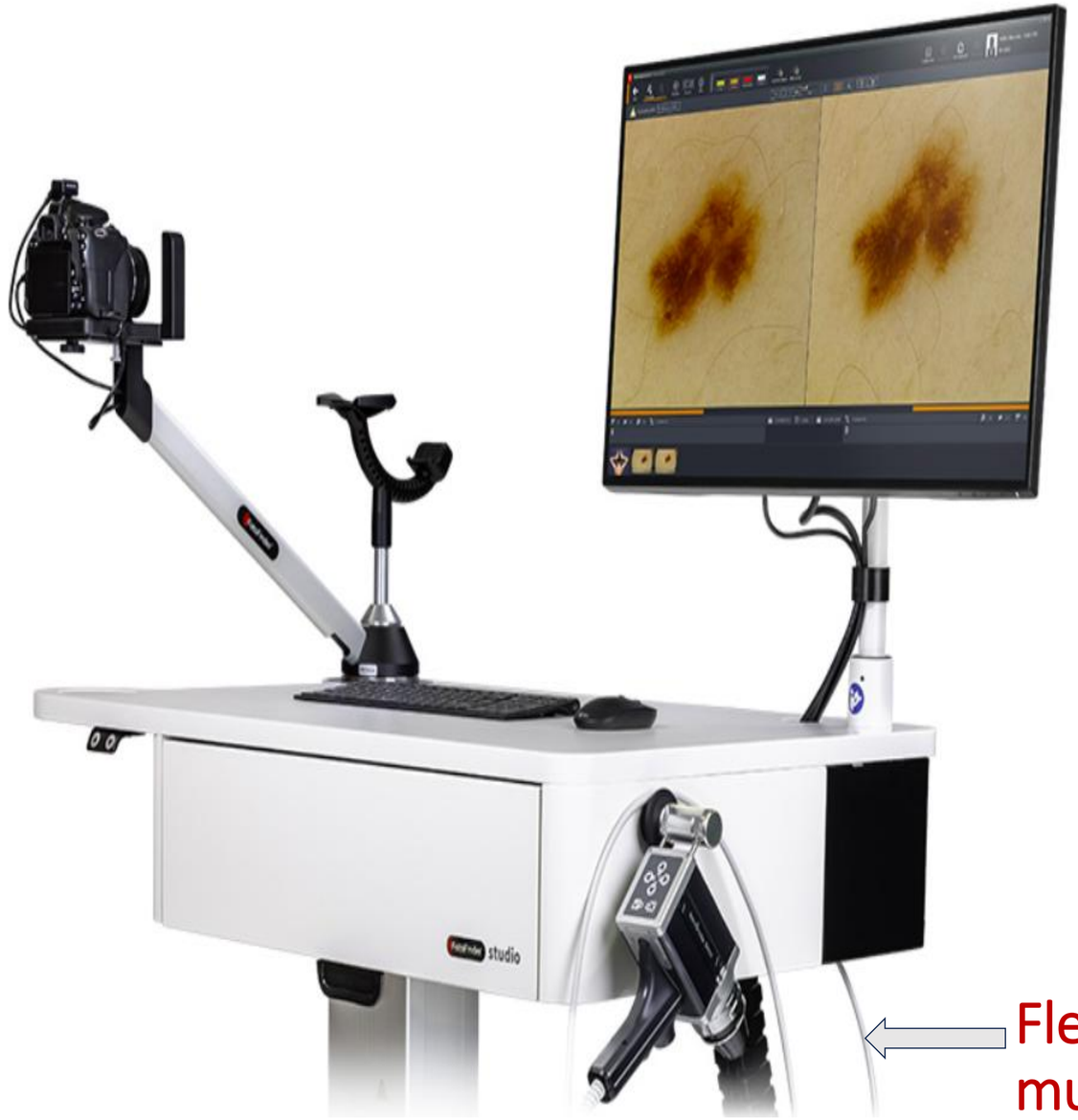
What is the need?

- Bedside non-invasive
- Enhanced diagnostic accuracy
- Reduction in unnecessary biopsies
- Monitoring skin lesions for prognosis and treatment progression
- Useful for pediatric skin lesions

Dermatoscopic tools

Handheld dermatoscope

Video dermatoscope



Integrated AI software

Higher magnification and resolution

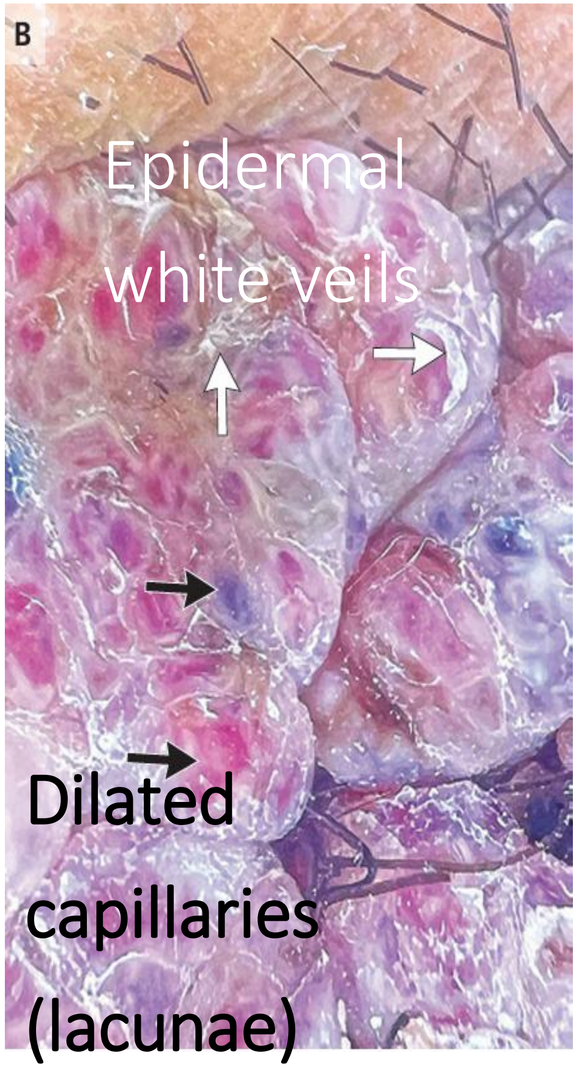
Flexible handle- easy to examine scalp, nails, and mucosal regions

Examples where dermoscopy helps in bedside diagnosis

Infective versus non-infective lesions

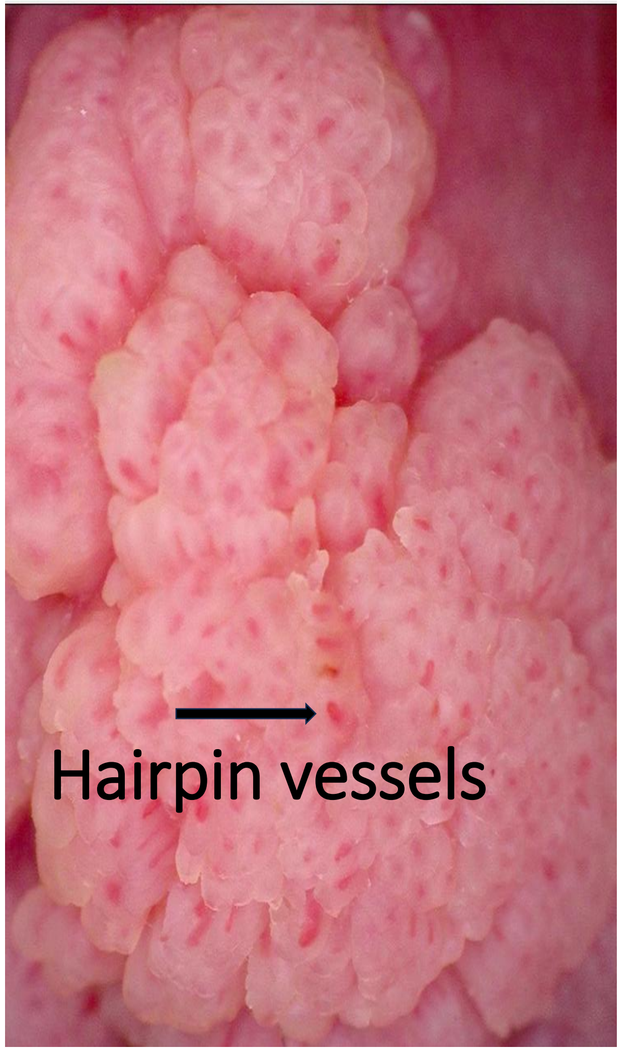
Angiokeratoma of Fordyce versus viral warts

A



1

B

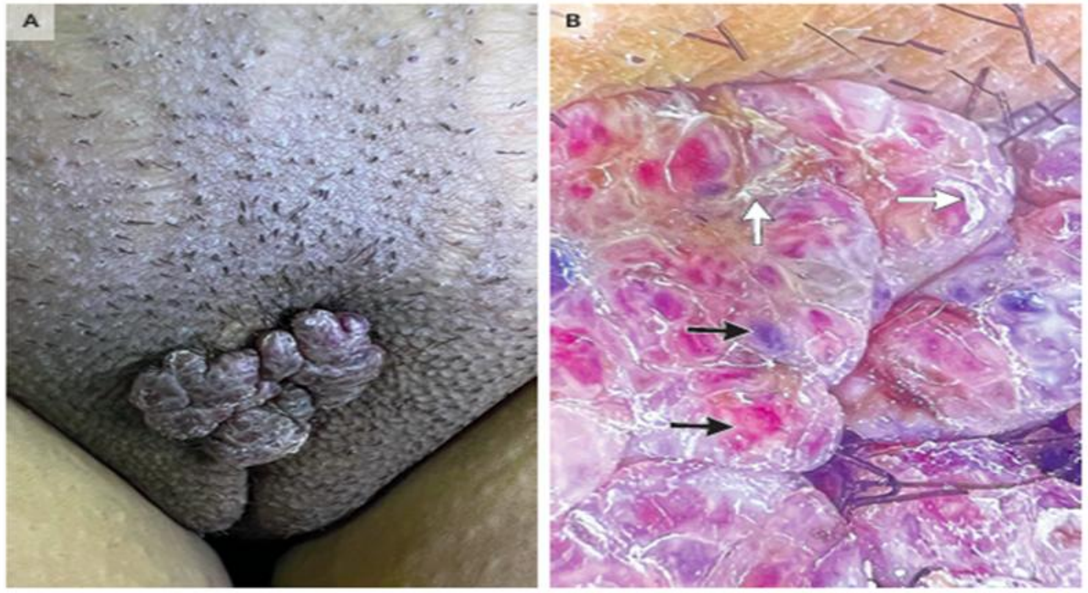


2

IMAGES IN CLINICAL MEDICINE

Stephanie V. Sherman, M.D., *Editor*

Angiokeratoma of Fordyce



A 35-YEAR-OLD WOMAN PRESENTED TO THE DERMATOLOGY CLINIC WITH a 4-year history of itchy, wartlike lesions in her pubic region. The lesions bled when traumatized during shaving of the pubic hair. One year before presentation, cryotherapy had been administered for a presumed diagnosis of genital warts, but the lesions had not abated. Physical examination was notable for hyperpigmented, hyperkeratotic papules forming three plaques over the mons pubis (Panel A). Dermoscopy showed well-demarcated reddish-to-violaceous lacunae, representing dilated capillaries (Panel B, black arrows), and an overlying white veil of epidermal hyperkeratosis (Panel B, white arrows). A diagnosis of angiokeratoma of Fordyce was made. Angiokeratoma of Fordyce is a benign cutaneous vascular lesion characterized by capillary dilatation in the papillary dermis. The lesion is most typically seen on the genitals and is more common in men than in women. The prevalence increases with age. Angiokeratoma of Fordyce may be difficult to distinguish from other genital lesions, such as warts and verrucous hemangiomas. The patient underwent a single session of treatment with a neodymium:yttrium–aluminum–garnet (YAG) laser to reduce the vascularity of the lesions, followed by ablation with an erbium:YAG laser. During telephone follow-up 2 months after the procedure, the patient reported that the lesions had not recurred.

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The NEW ENGLAND JOURNAL of MEDICINE

Scarring versus non-scarring alopecia

Follicular openings on trichoscopy

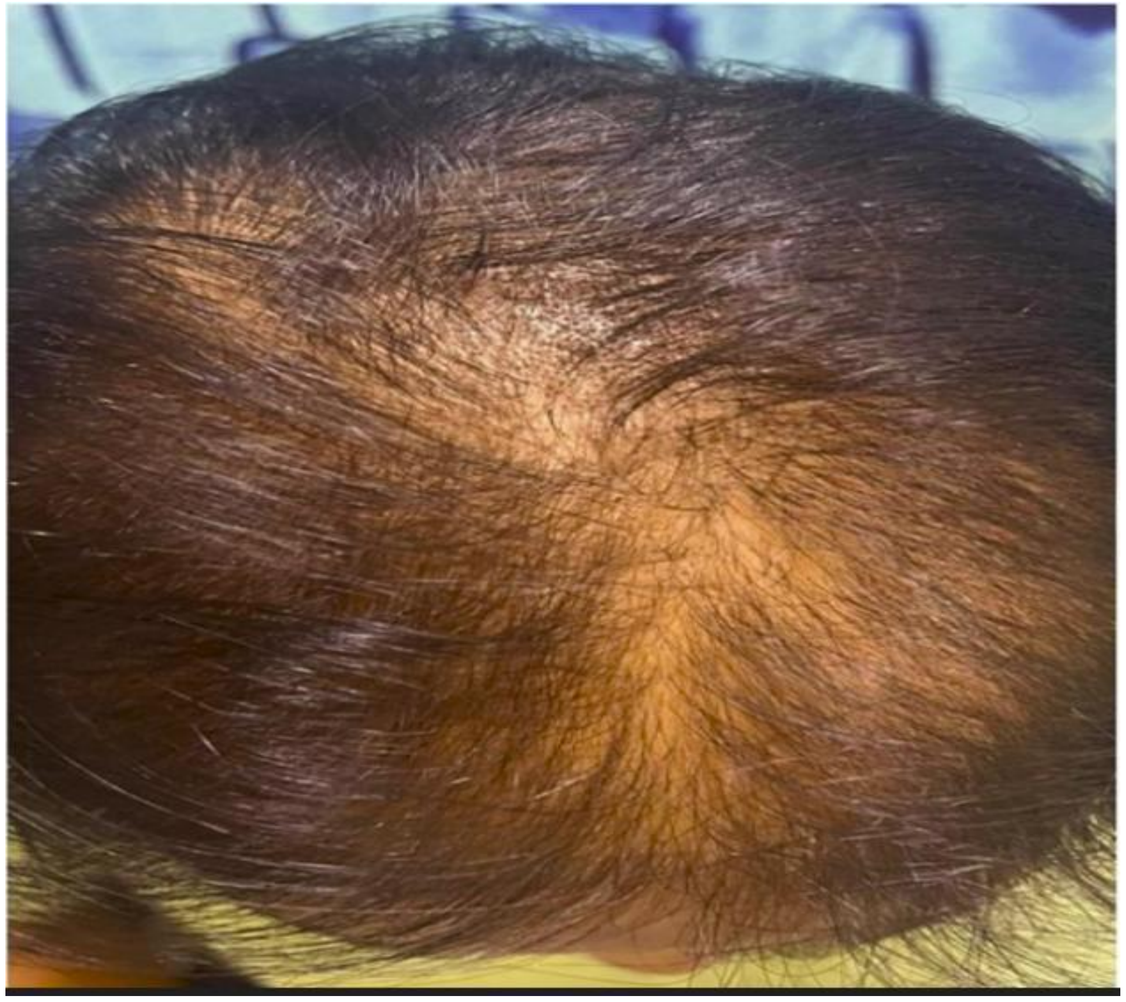
Present

Non- cicatricial alopecia

Absent

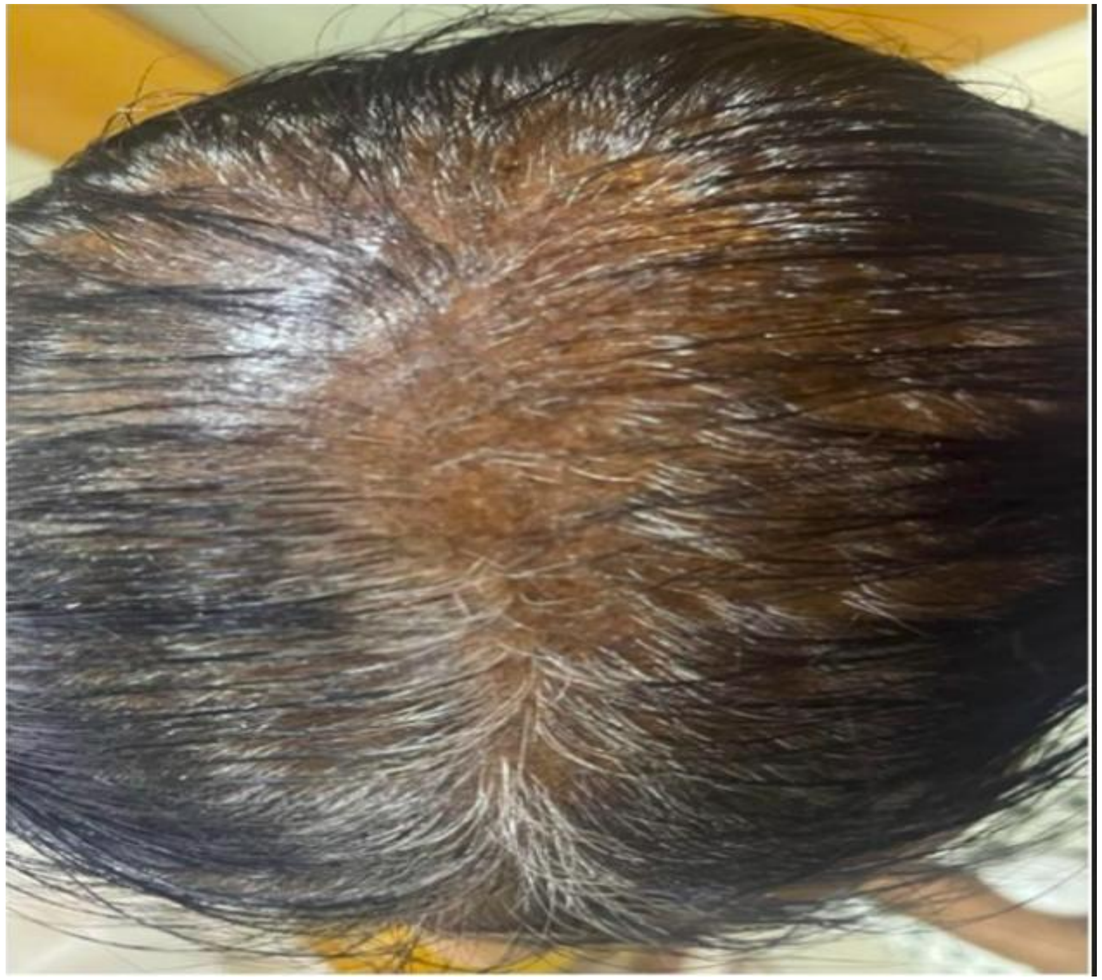
Cicatricial alopecia

Androgenetic alopecia vs lichen planopilaris



Androgenetic alopecia

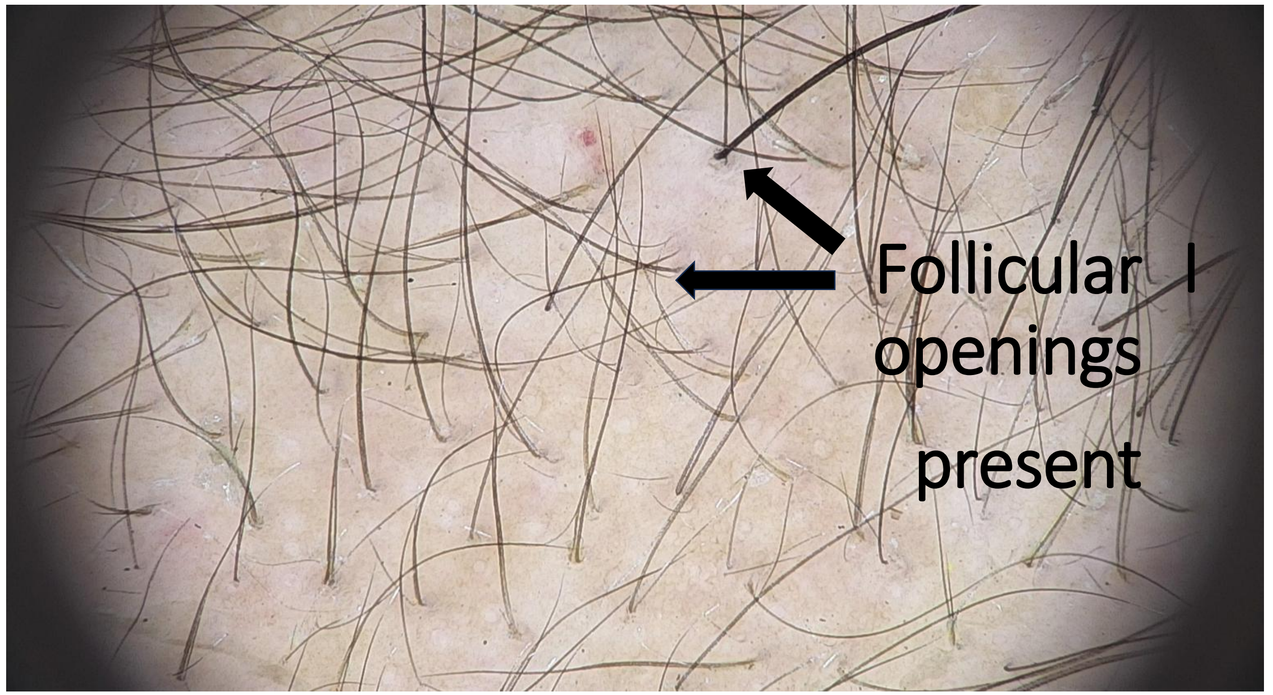
(non- scarring alopecia)



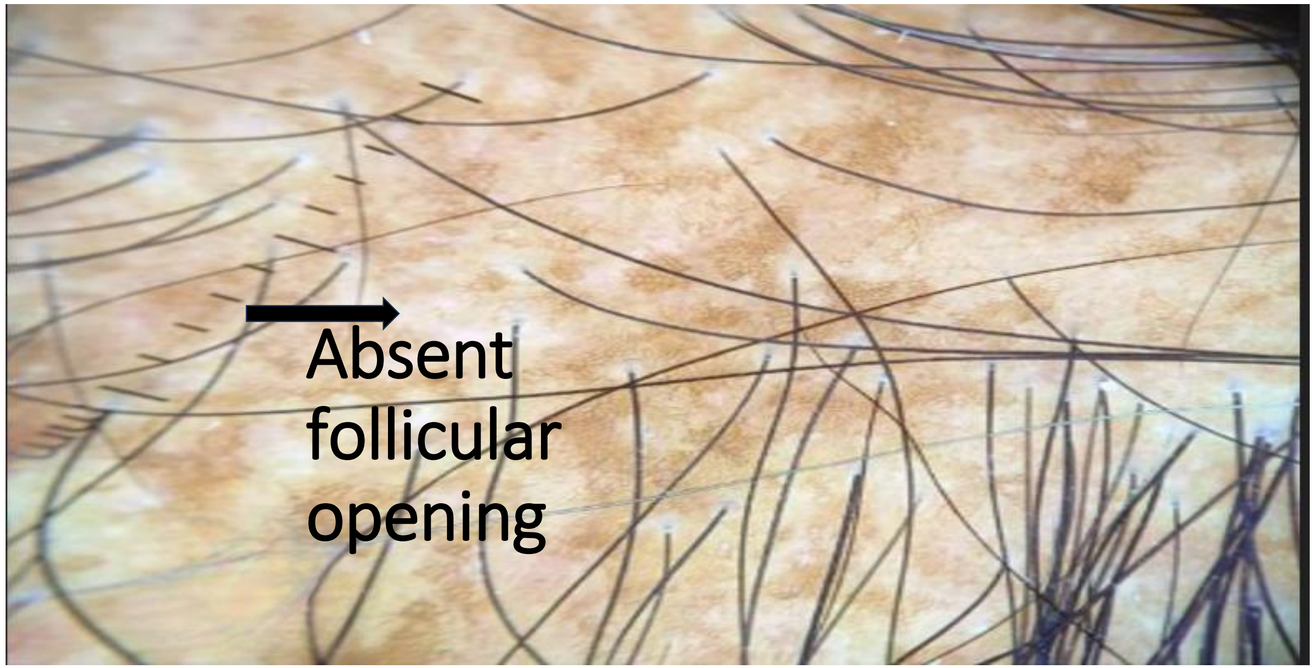
Lichen planopilaris

(scarring alopecia)

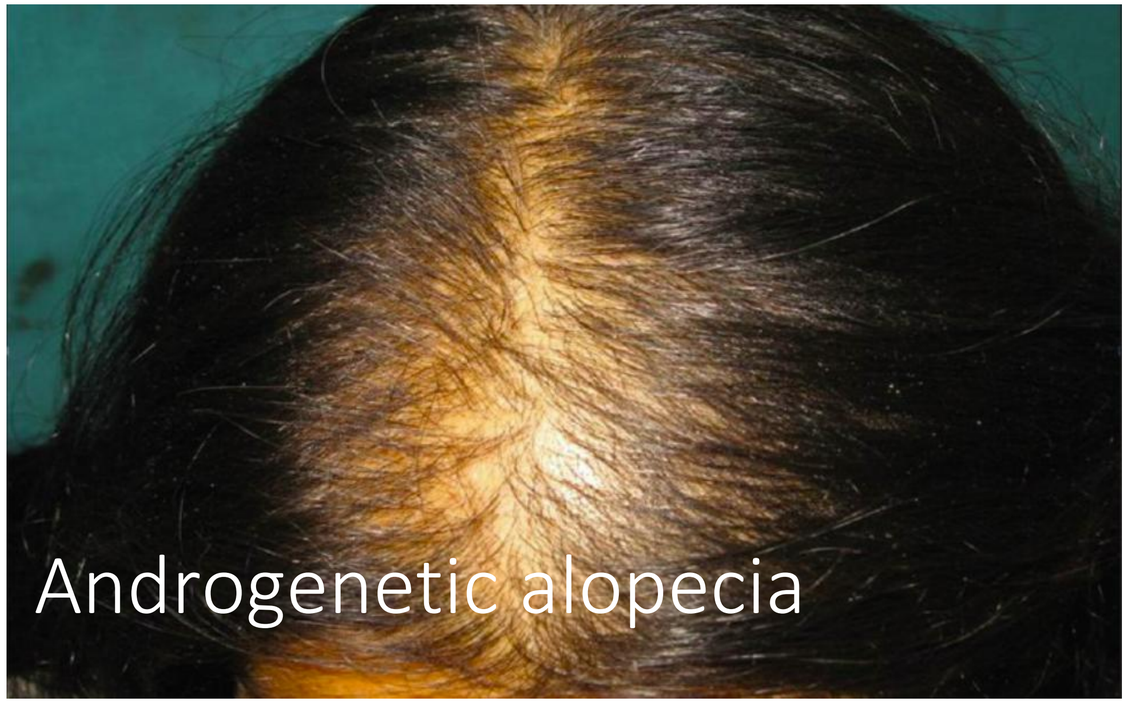

Androgenetic alopecia


Follicular openings present


Lichen planopilaris


Absent follicular opening

Non- scarring alopecia

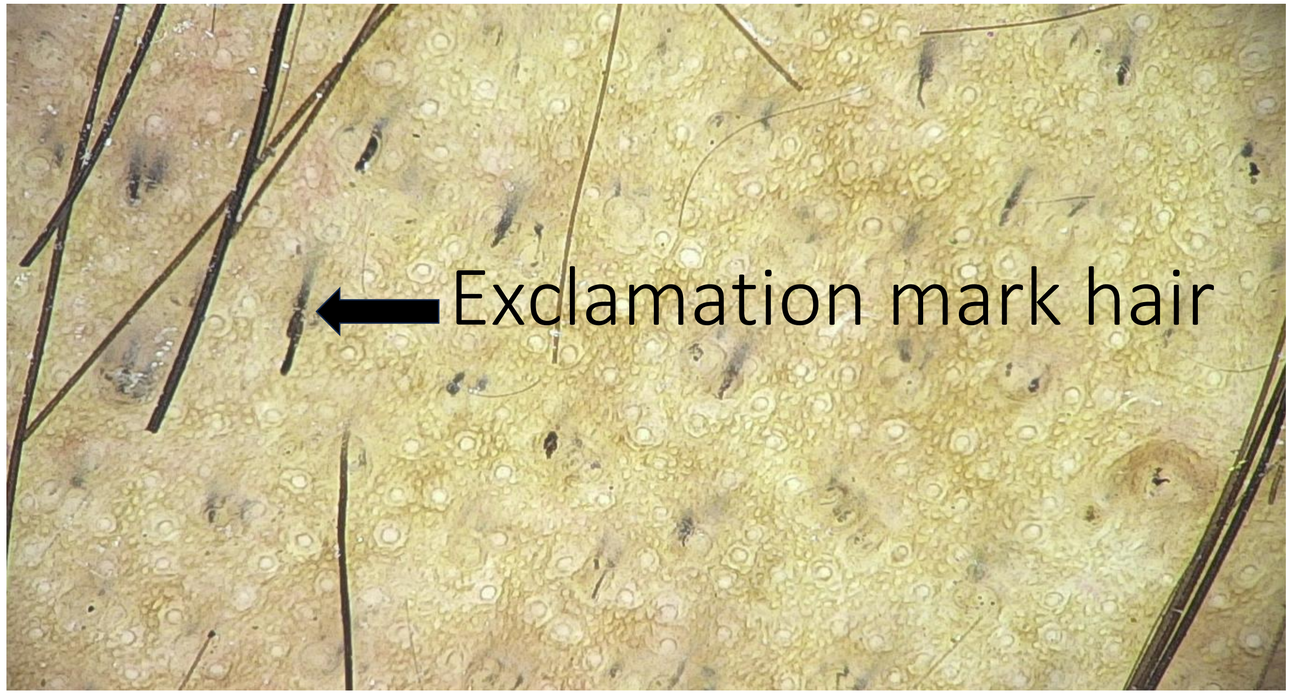

Androgenetic alopecia


Hair diameter
diversity >20%


Telogen effluvium


Hair diameter
diversity <20%

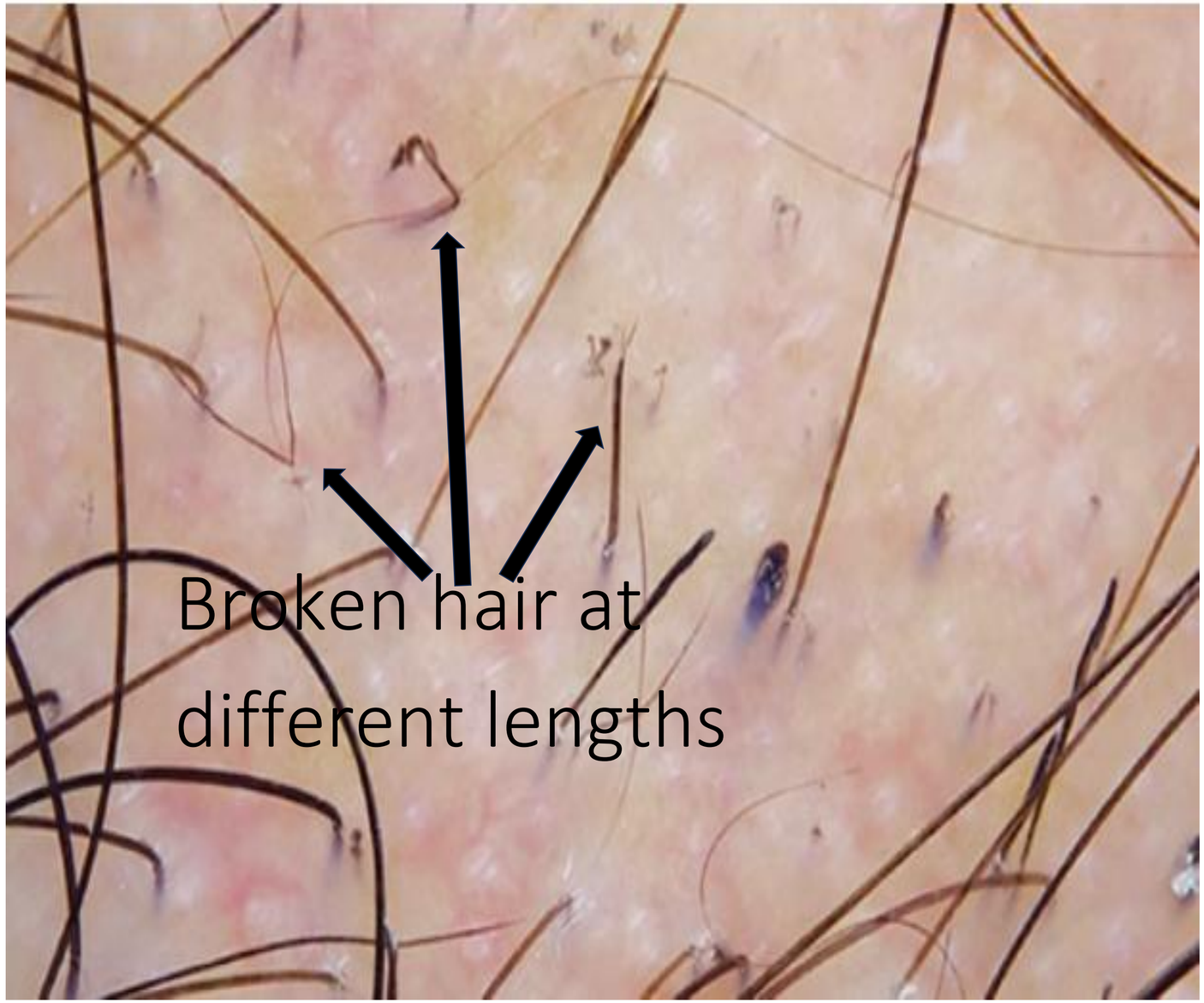

Alopecia areata


Exclamation mark hair


Tinea capitis


Comma hair

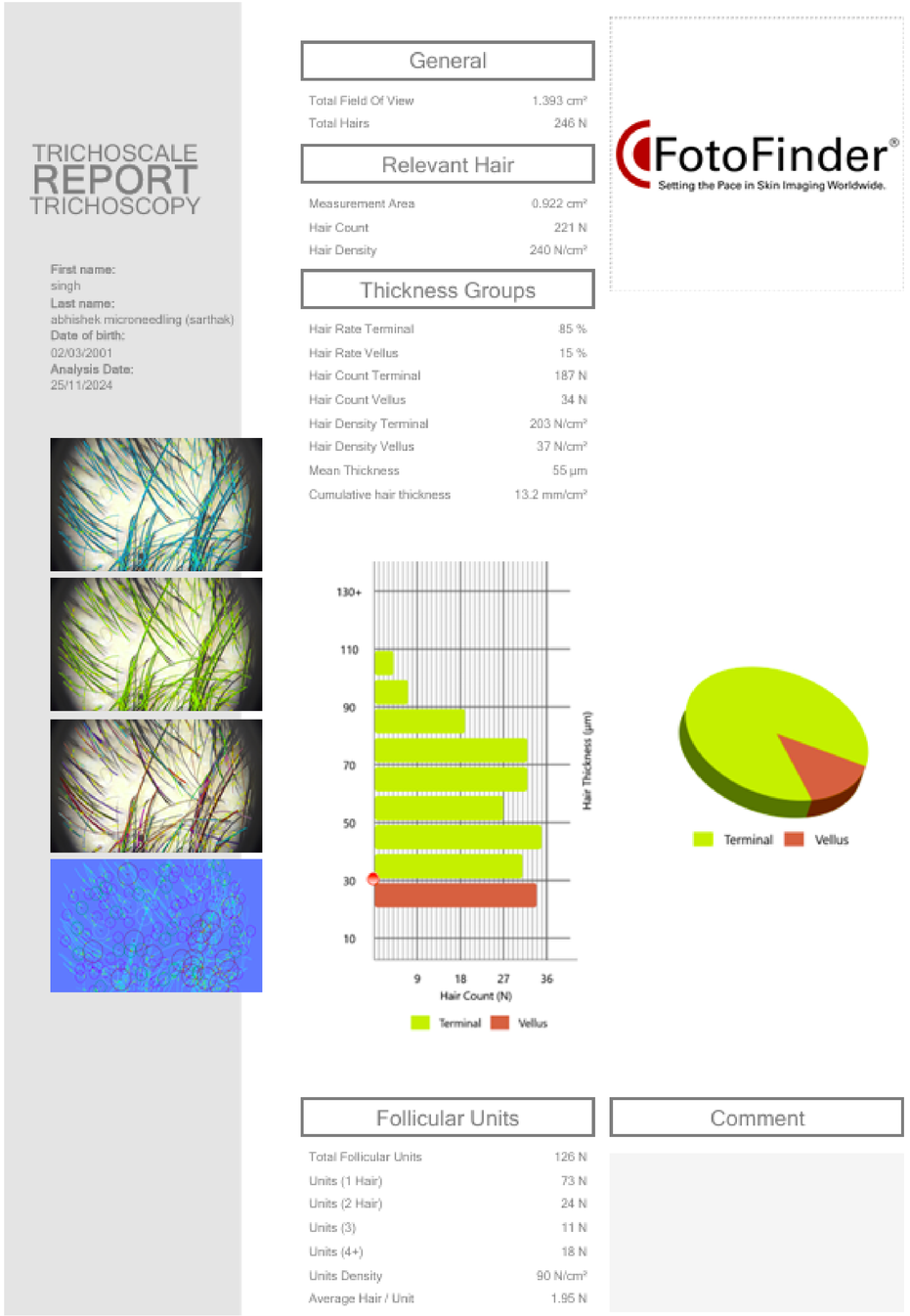
Trichotillomania



Trichoscopy

Trichoscale

- **TrichoScale** is an AI-enabled software tool used in **trichoscopy** for the quantitative analysis of hair and scalp conditions.



FotoFinder Trichoscale DX

TrichoLAB

Back | Fullscreen | Fit | 1:1 | Generate report | Export to .csv | Instant Count

agrawal, seema - 08/03/1996

Patient ID: 7501139664

After regrowth

1 mm

Basic View | Anagen/Telogen | Terminal/Vellus | Single Hair View | Follicular Units

Summary

Hair Density		
Measurement Area	0.922	cm²
Hair Count	0	N
Hair Density	0	N/cm²
Hair Cycle		
Hair Rate Anagen	0	%
Hair Rate Telogen	100	%
Hair Count Anagen	0	N
Hair Count Telogen	0	N
Hair Density Anagen	0	N/cm²
Hair Density Telogen	0	N/cm²
Hair Growth		
Initial Hair Length	0.50	mm
Regrowth Time	2.0	days
Average Growth Rate	0.00	mm/day
Thickness Groups		
Hair Rate Terminal	0	%
Hair Rate Vellus	100	%
Hair Count Terminal	0	N
Hair Count Vellus	0	N
Hair Density Terminal	0	N/cm²
Hair Density Vellus	0	N/cm²
Mean Thickness	0	µm
Cumulative hair thickness	0.0	mm/cm²
Follicular Units		
Total Follicular Units	0	N
Units (1 Hair)	0	N
Units (2 Hair)	0	N
Units (3)	0	N
Units (4+)	0	N
Units Density	0	N/cm²
Average Hair / Unit	0	N

General		
Total Field Of View	1.393	cm²
Total Hairs	0	N

Pie Chart | Length | Thickness Chart

General

Total Field Of View	1.393 cm²
Total Hairs	222 N

Hair Density

Measurement Area	0.922 cm²
Hair Count	193 N
Hair Density	209 N/cm²

Hair Cycle

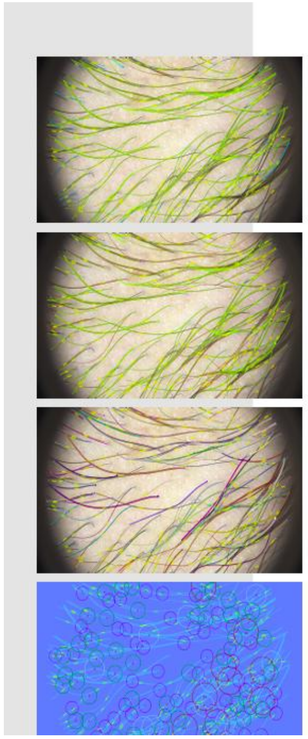
Hair Rate Anagen	96 %
Hair Rate Telogen	4 %
Hair Count Anagen	159 N
Hair Count Telogen	7 N
Hair Density Anagen	201 N/cm²
Hair Density Telogen	8 N/cm²

Thickness Groups

Hair Rate Terminal	52 %
Hair Rate Vellus	48 %
Hair Count Terminal	101 N
Hair Count Vellus	92 N
Hair Density Terminal	110 N/cm²
Hair Density Vellus	99 N/cm²
Mean Thickness	37 µm
Cumulative hair thickness	7.7 mm/cm²

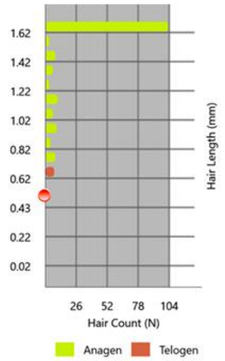
Follicular Units

Total Follicular Units	121 N
Units (1 Hair)	56 N
Units (2 Hair)	43 N
Units (3)	13 N
Units (4+)	9 N
Units Density	87 N/cm²
Average Hair / Unit	1.83 N

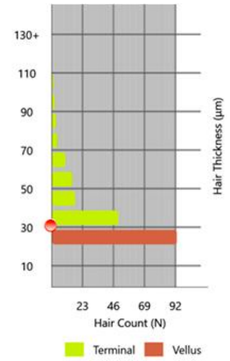


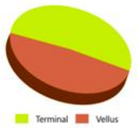
Hair Growth

Initial Hair Length	0.50 mm
Regrowth Time	2.0 days
Average Growth Rate	0.94 mm/day



Mean Thickness	37 µm
Cumulative hair thickness	7.7 mm/cm²



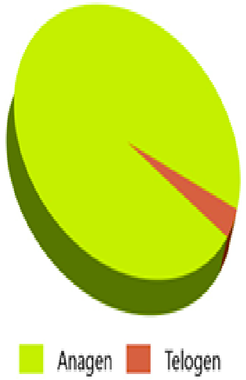


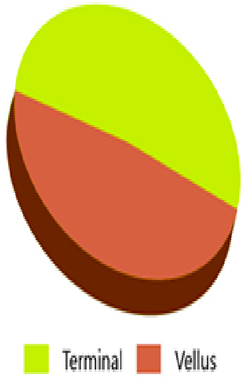
Pre-treatment

Post-treatment

Hair Cycle

Hair Rate Anagen	96 %
Hair Rate Telogen	4 %
Hair Count Anagen	159 N
Hair Count Telogen	7 N
Hair Density Anagen	201 N/cm²
Hair Density Telogen	8 N/cm²

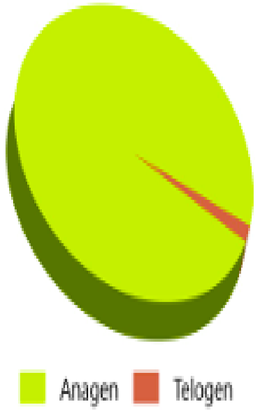


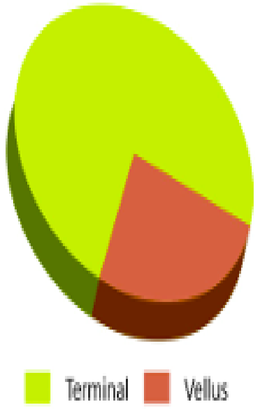


After 3 sessions of HaiRestart

Hair Cycle

Hair Rate Anagen	98 %
Hair Rate Telogen	2 %
Hair Count Anagen	201 N
Hair Count Telogen	5 N
Hair Density Anagen	254 N/cm²
Hair Density Telogen	5 N/cm²





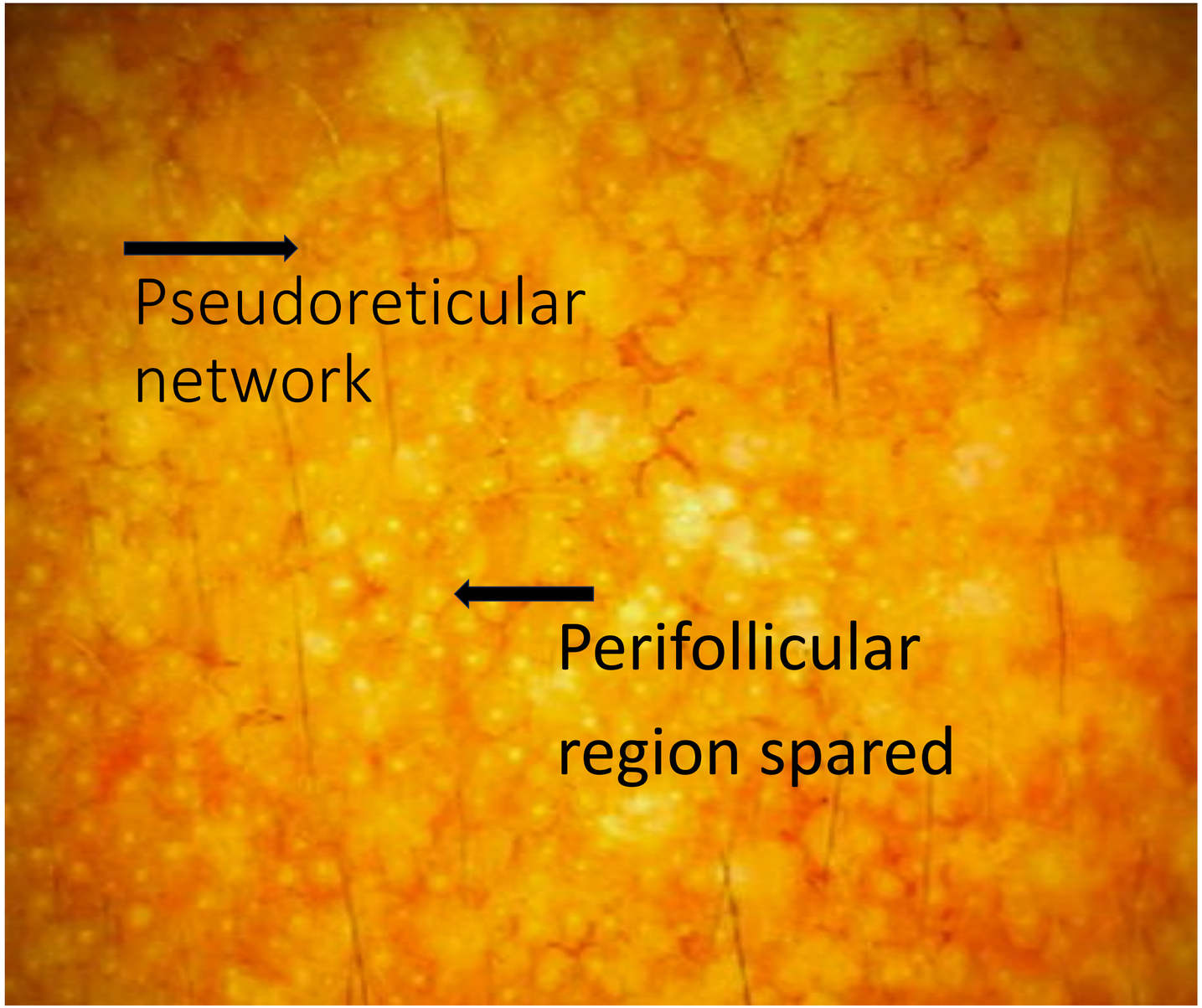
Pigmentary dermatoses

Melasma

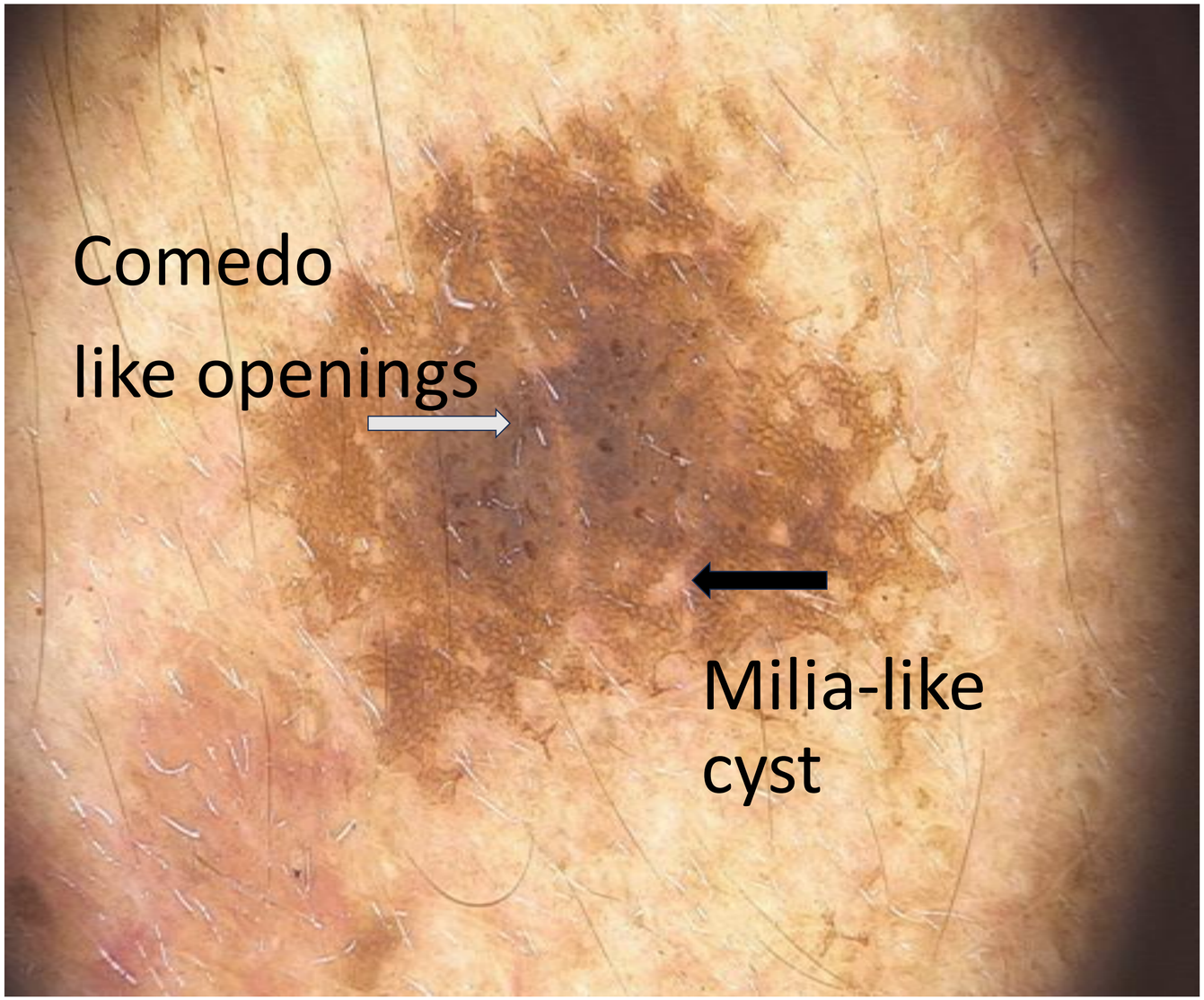
Seborrheic keratoses

Acanthosis nigricans

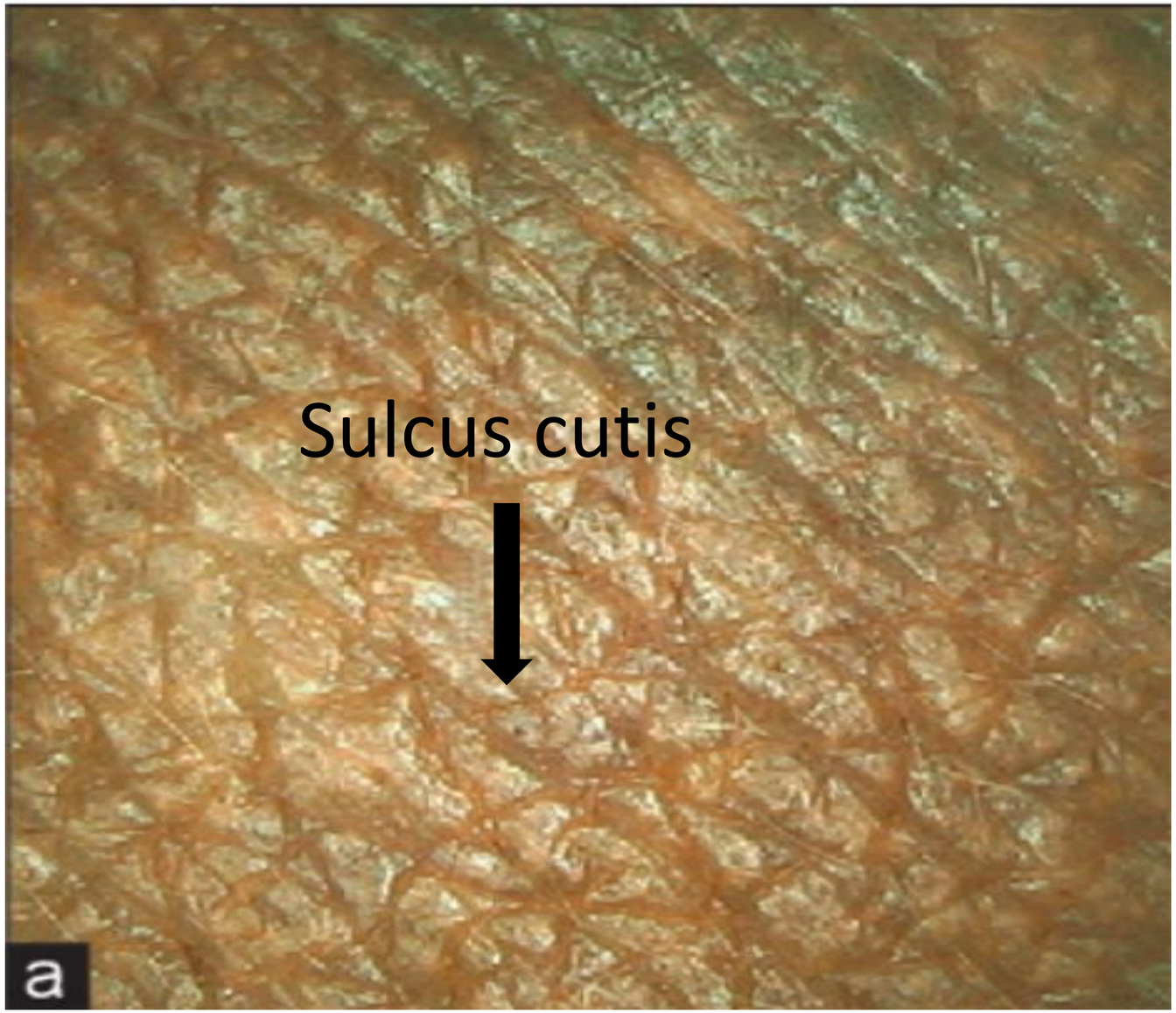
Melasma



Seborrheic keratoses



Acanthosis nigricans

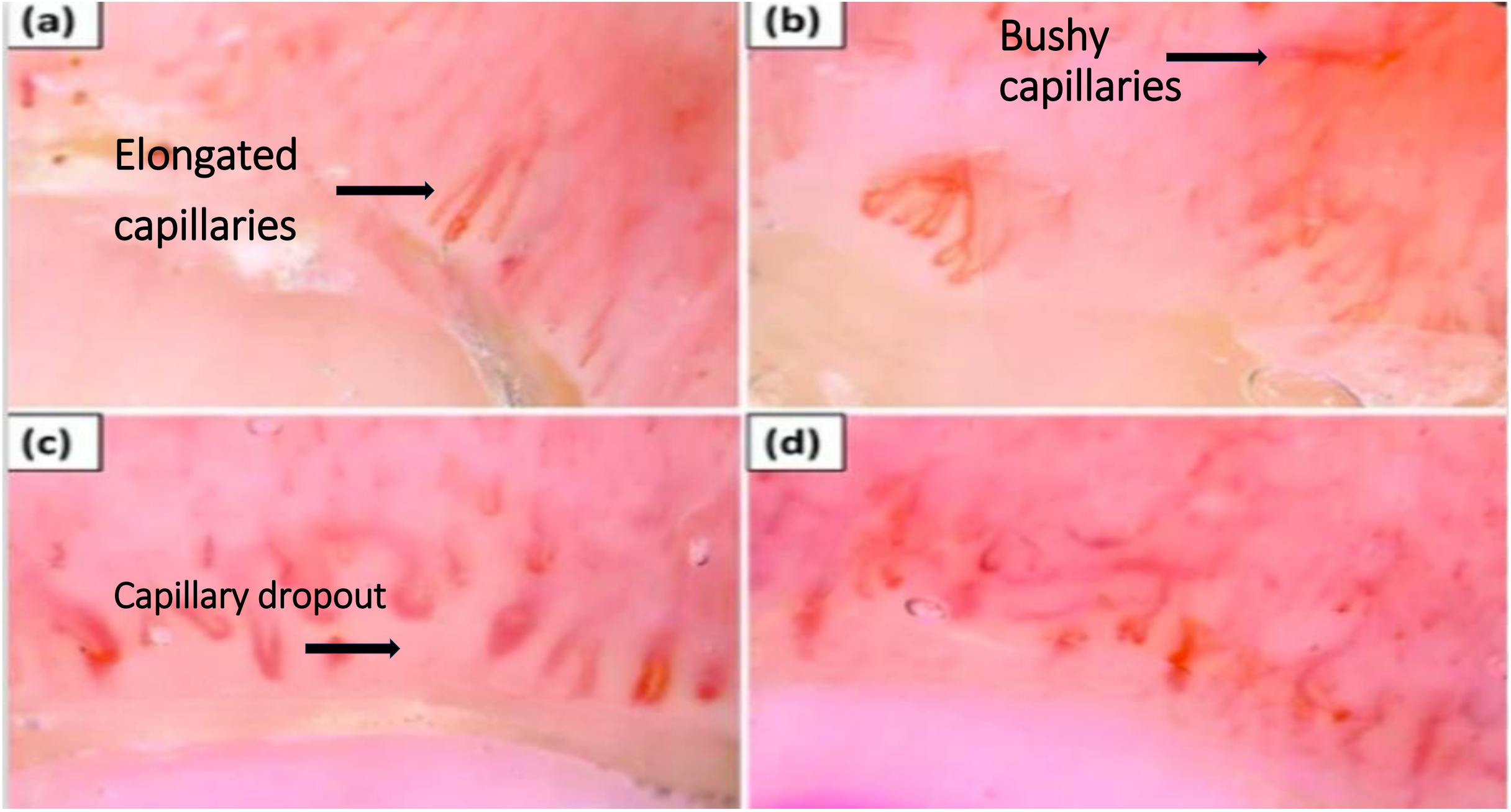


Nail fold capillaroscopy

Dermatomyositis

Heliotrope rash

Gottron's papules with overlying ulcers

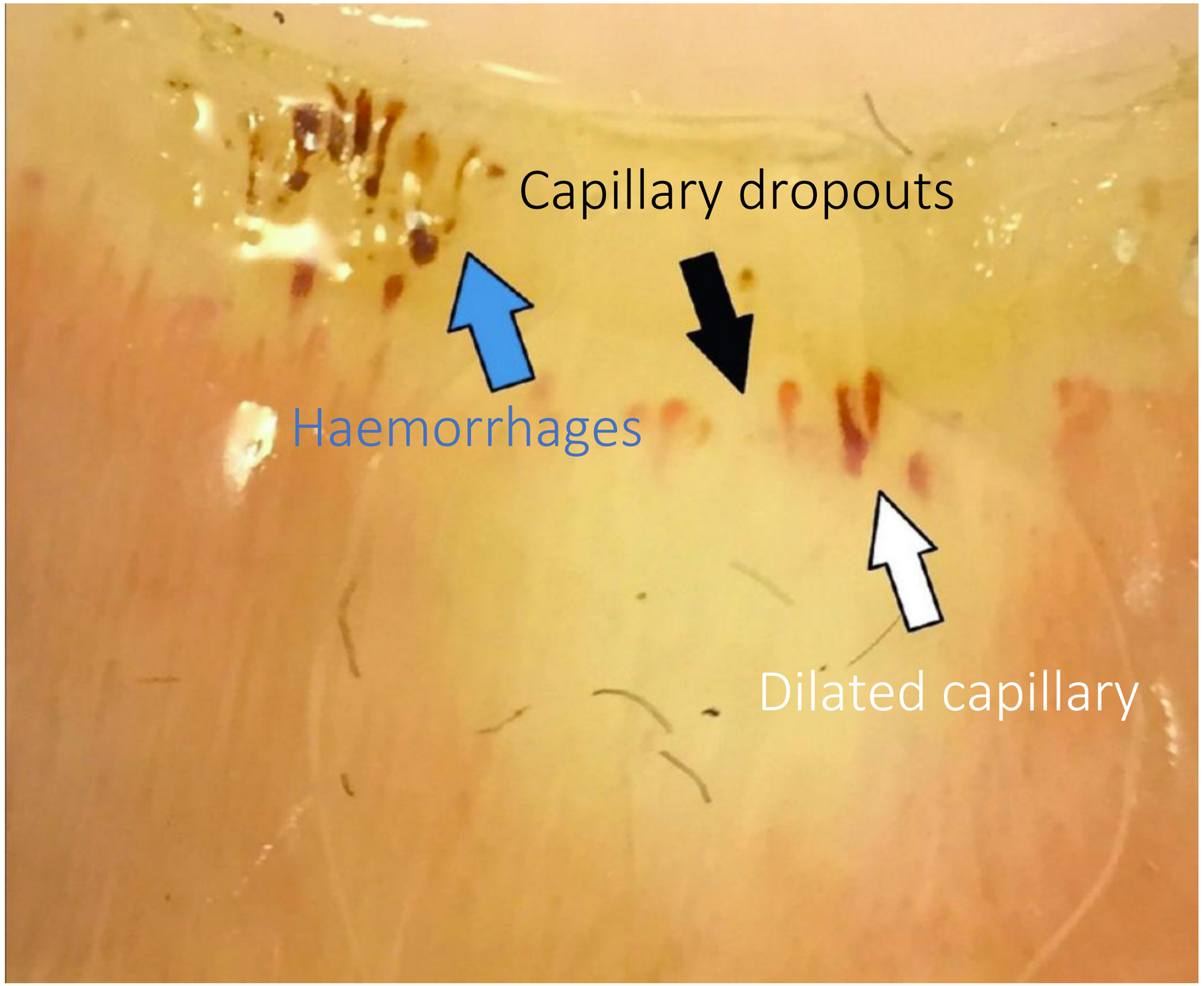

(a)
Elongated capillaries
(b)
Bushy capillaries
(c)
Capillary dropout
(d)

Systemic sclerosis

Mask like face

Salt and pepper pigmentation

Can be used as a **prognostic marker** as it helps in early detection of **Interstitial lung disease (ILD)**.



Conclusion

Dermoscopy stands as a bridge between the visible and the microscopic, transforming mere observation into precision-driven diagnosis.

Together, let us embrace this lens into the unseen, where science meets artistry in the pursuit of better healthcare.

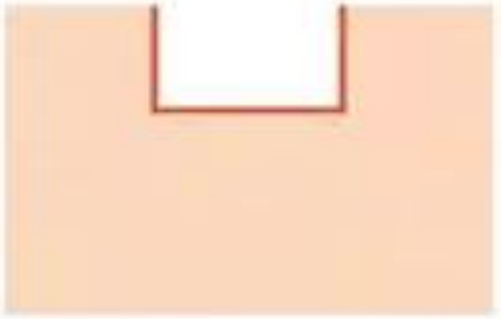

thank you

Fractional microneedling radiofrequency treatment of acne scars

Dr. Nishtha Mishra

Acne scars: a lasting impact

Rolling Scars



Boxed Scars

Icepick Scars

Rolling Scar

Boxcar Scar

Ice Pick Scar

Addressing acne scars: current approaches

Dermaroller

Surgical excision

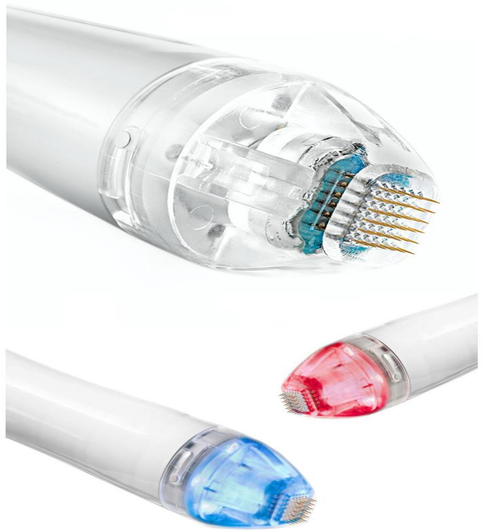
Laser therapy

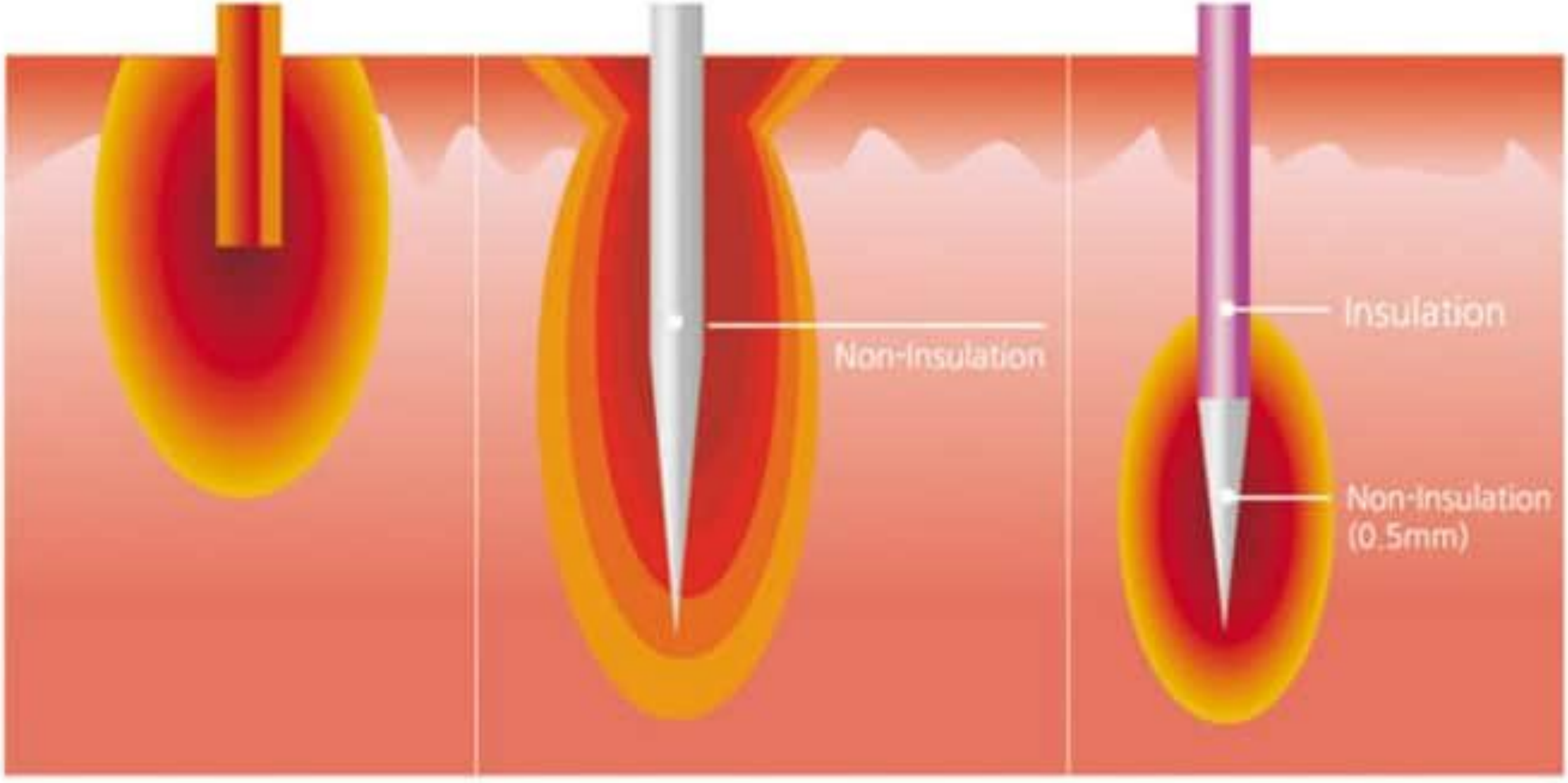
Why do we need a newer modality?

Fractional microneedling radiofrequency

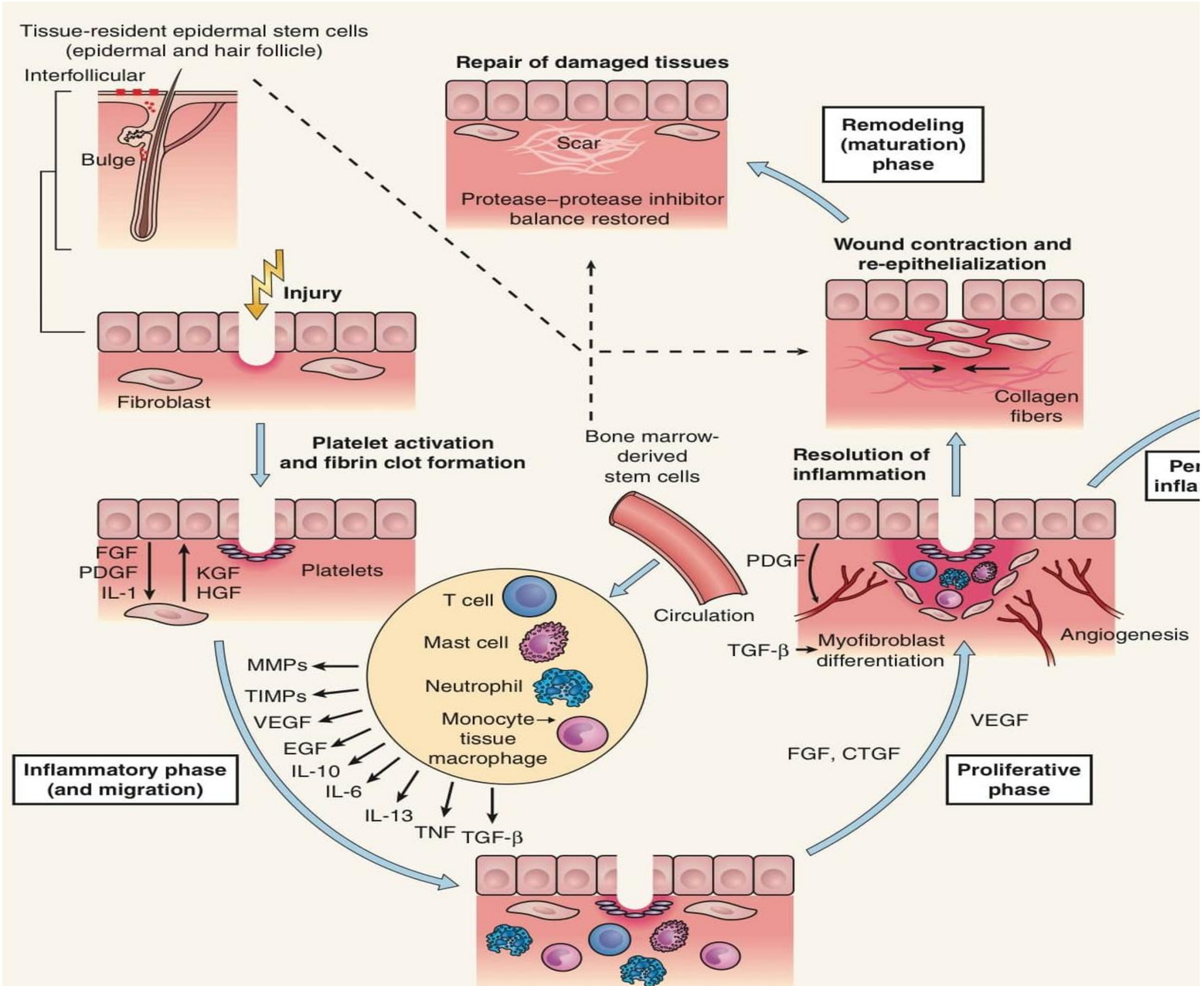
MNRF is a technique combining microneedling & RF energy, creating controlled micro-injuries while delivering RF energy and acts by:

- ↑ Collagen & elastin production
- ↑ Skin remodeling & improved texture
- ↓ Risk of hyperpigmentation

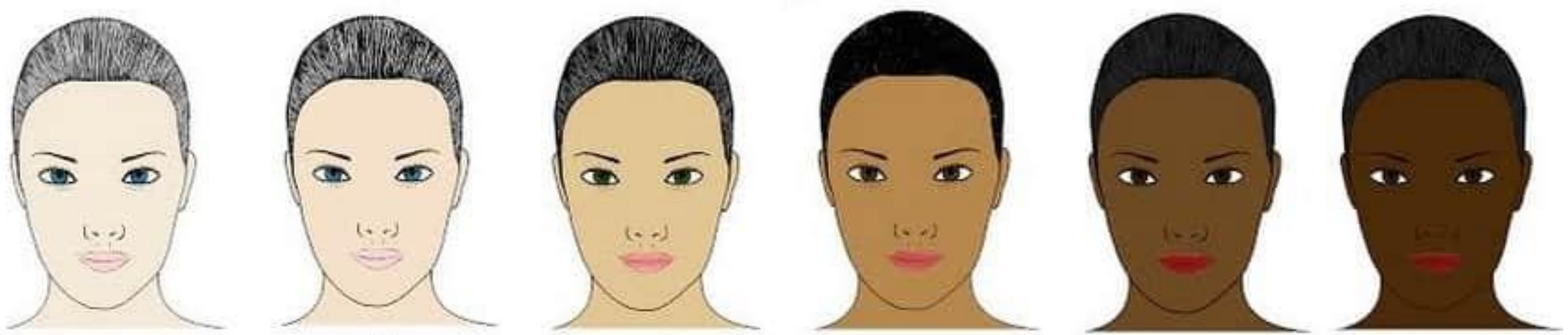



Non-Insulation
Insulation
Non-Insulation
(0.5mm)

Principle of MNRF



Fitzpatrick skin types



Post-inflammatory hyperpigmentation

Study design & patient selection

- **Study type:** Retrospective
- **Sample size:** 55 patients (38 males, 17 females)
- **Study period:** January 2022 – December 2023
- **Approval:** Institutional ethics committee, Dr. D.Y. Patil Medical College
- **Inclusion criteria:**

1. Diagnosed with acne scars
2. Completed **4 MNRF sessions** (1-month intervals)
3. Provided informed consent

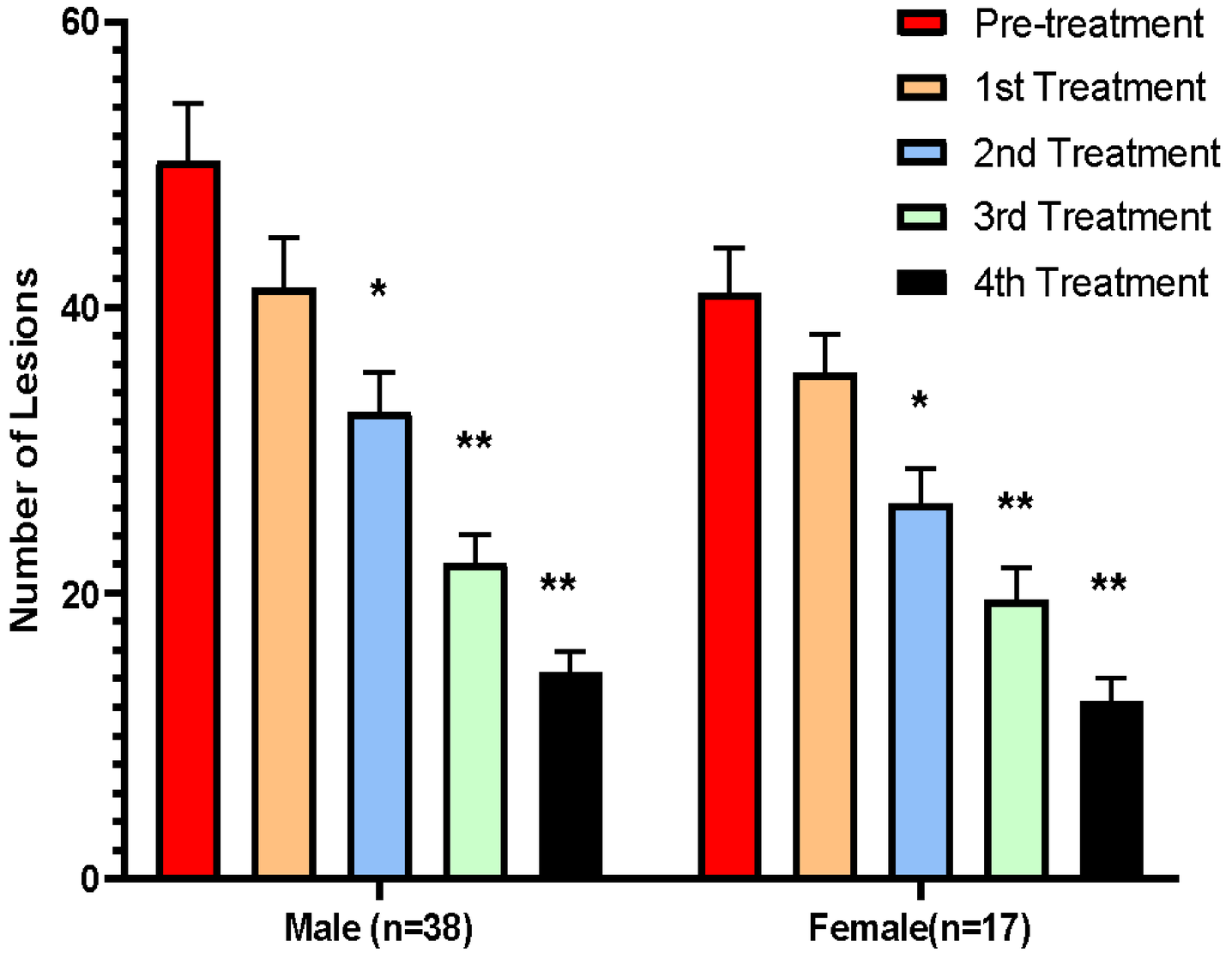
- **Exclusion criteria:**

1. Prior acne scar treatments


(A)
(B)
(C)
(D)

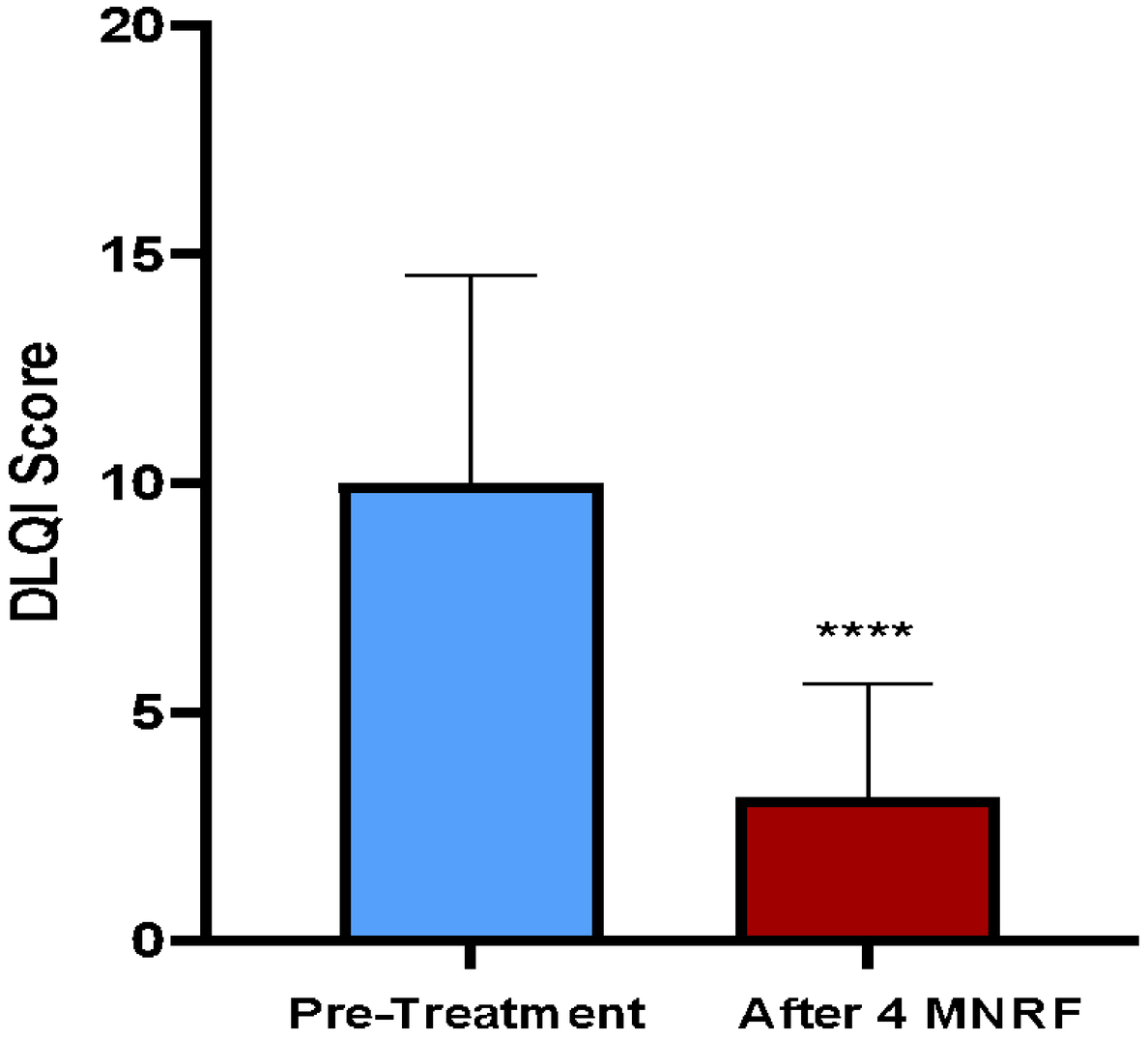

(A)
(B)
(C)
(D)

Reduction in scar count



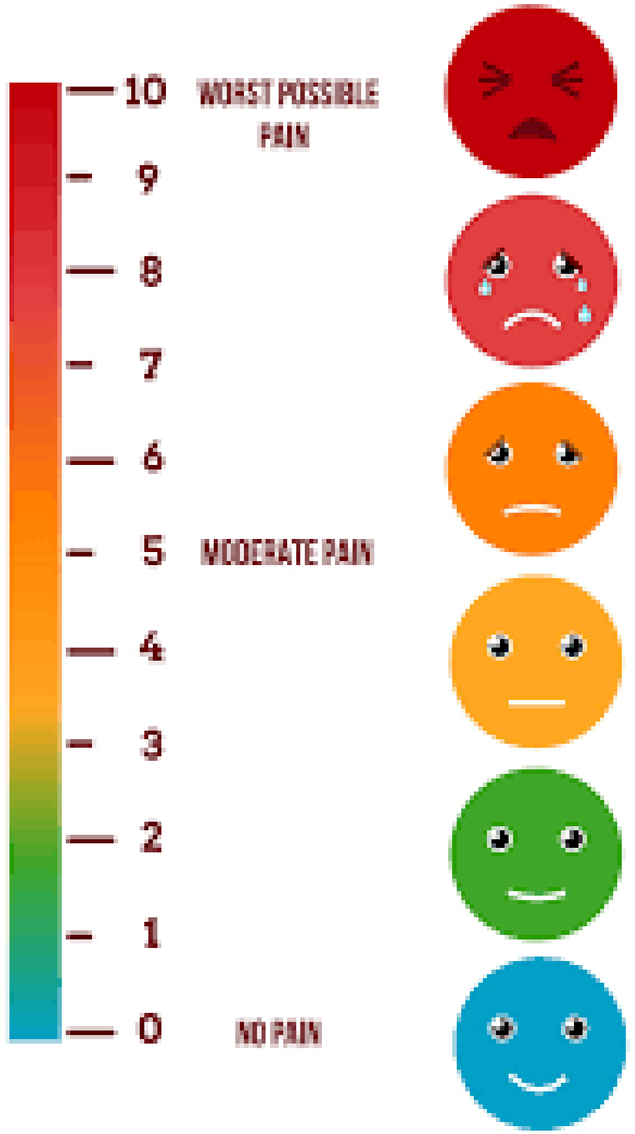
Impact on mean DLQI scores

- Pre-treatment : 10.00
- Post-treatment : 3.147
- Difference is significant ($p < 0.0001$)
- 95% Confidence Interval: -8.608 to -5.098 (significant)



Pain assessment during procedure

- It was done using the VAS (1-10)
- Mean VAS score: 1.54 ± 0.78
- Indicates relatively low pain immediately post-procedure.
- Supports patient compliance and treatment feasibility.



Results

- Significant reduction in acne scar count ($p < 0.0001$).
- Improved DLQI scores indicating patient well-being.
- Minimal pain (VAS = 1.54 ± 0.78) → well-tolerated treatment.


Thank
you

DPU

SIRT1, Vitiligo & Skin Cancer

Dr. Anushka Agarwal

UV rays

01

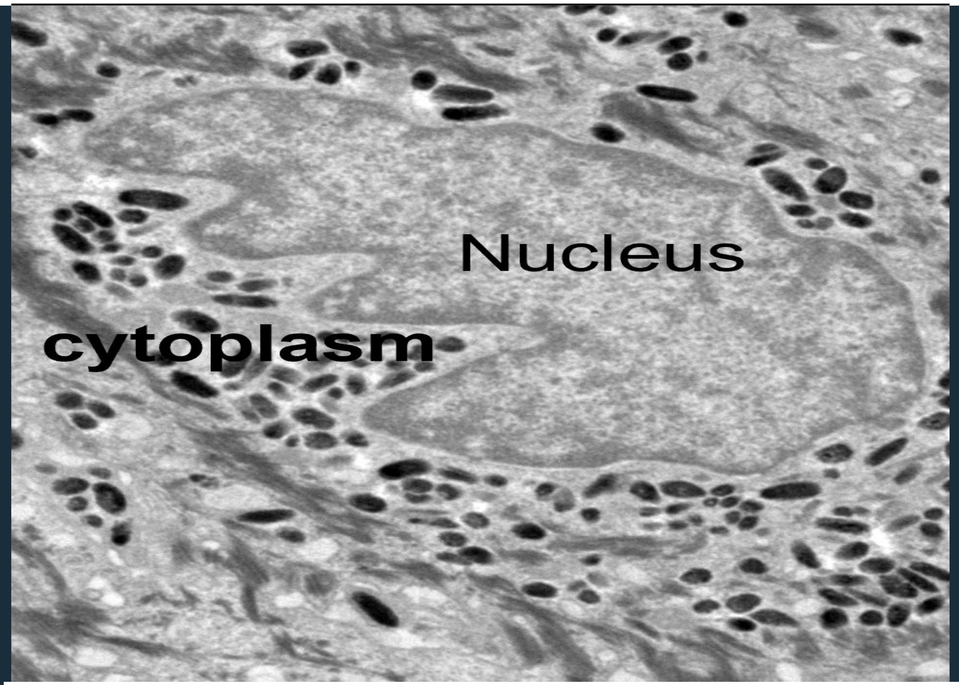
- Skin loses the ability to absorb free radicals.
- These cause damage to DNA, proteins, and other cellular contents and lead to the development of skin cancers.

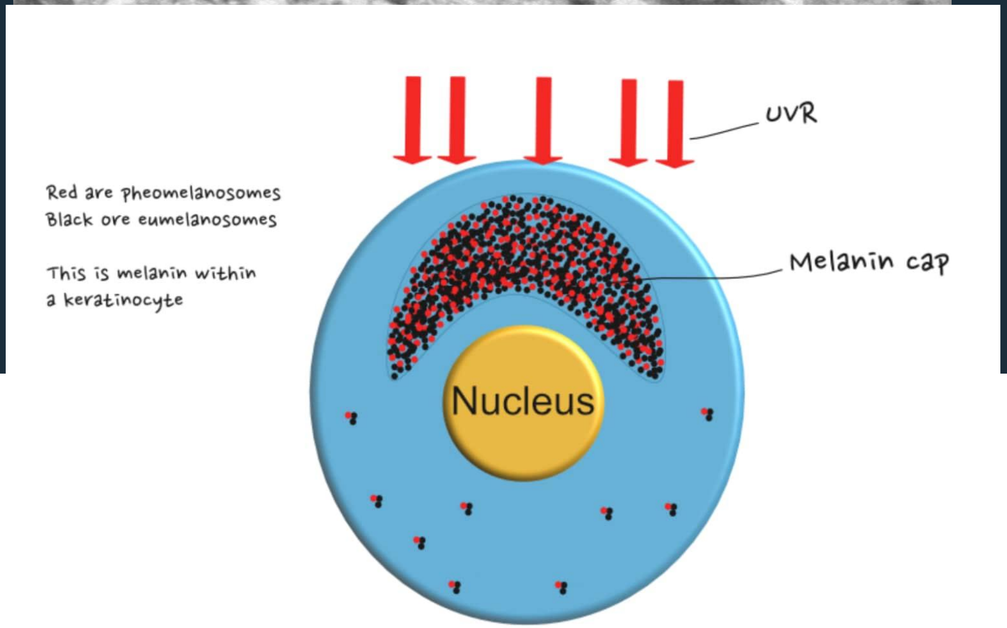
02

- Early effects – redness, delayed tanning, skin thickening, and free radical formation.
- Long-term effects – faster aging, weaker immunity, and skin cancers.

03

- UV-A Radiation: immediate darkening and early aging.
- UV-B Radiation: 1000 times more likely to cause sunburn.
- UV-C Radiation: most dangerous, but filtered by the ozone layer





The Protective effect of

Melanin

- Synthesized within melanosomes and transferred to keratinocytes
- Surrounds the nucleus forming a supranuclear shield
- Acts like a broadband UV absorbant
- Antioxidant
- Free radical scavenger
- SPF : 2 to 4

Skin color X cancer

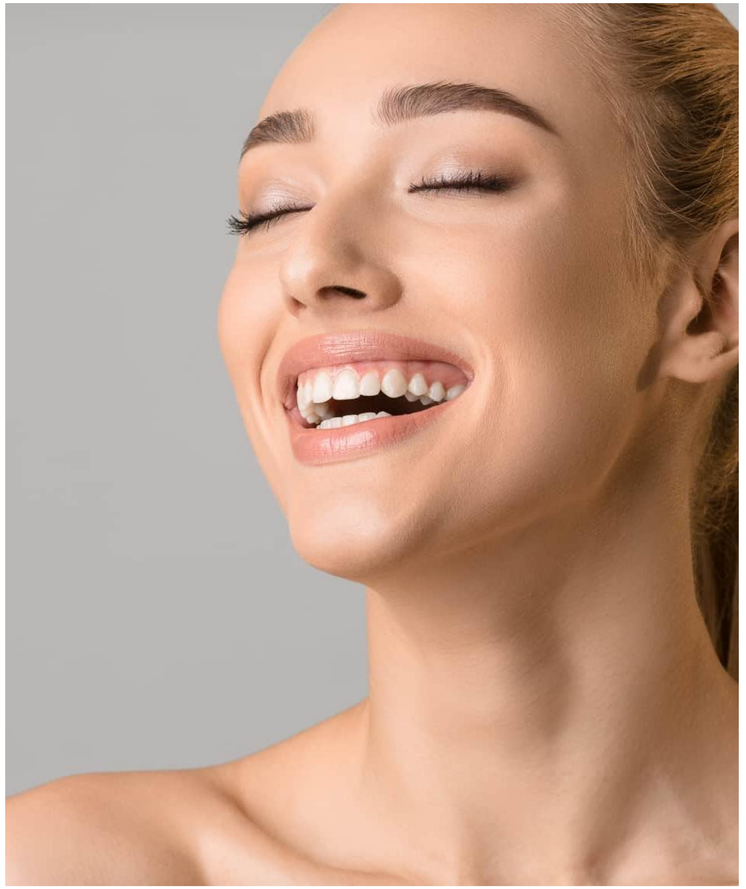
Risk of skin cancer

Highest

Individuals with albinism

No melanin

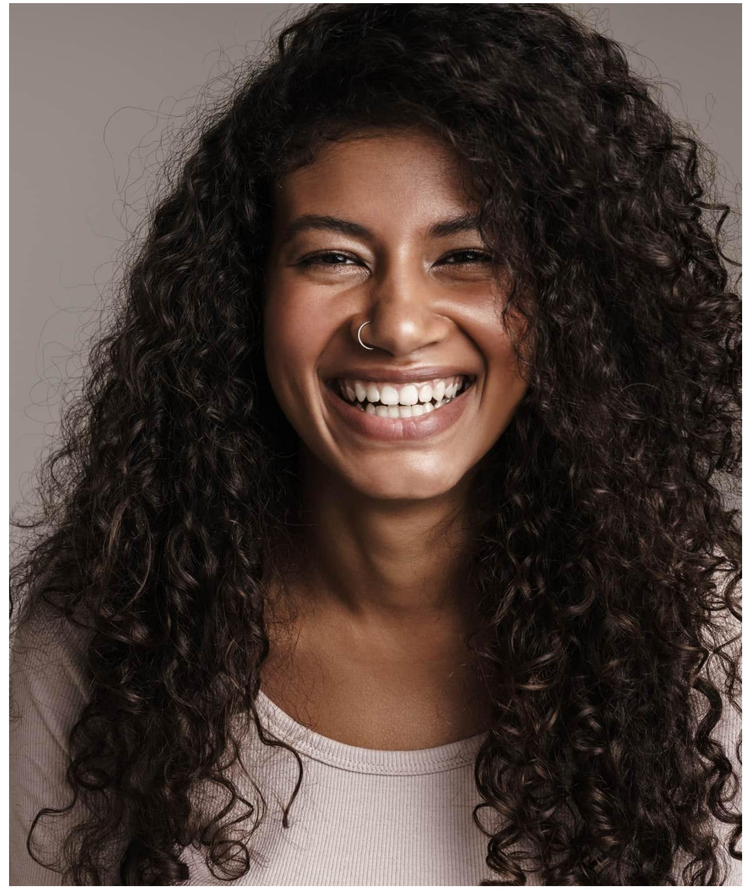
High



Fair skinned individuals

Some melanin

Lowest



Dark skinned individuals

Max melanin

Vitiligo

Patchy depigmenting disorder

- A chronic skin disorder affecting 1-2% of Indians

- Leads to severe psychological stress and a loss of quality of life

- Treatment of choice: UV-B

PARADOX

Surprisingly in Individuals with vitiligo

Decreased melanin

↓

Lower risk of skin cancer

Compared to ethnicity matched individuals

The Question

Why do people with vitiligo have a low risk of developing skin cancer

Study design

Non-lesional biopsies

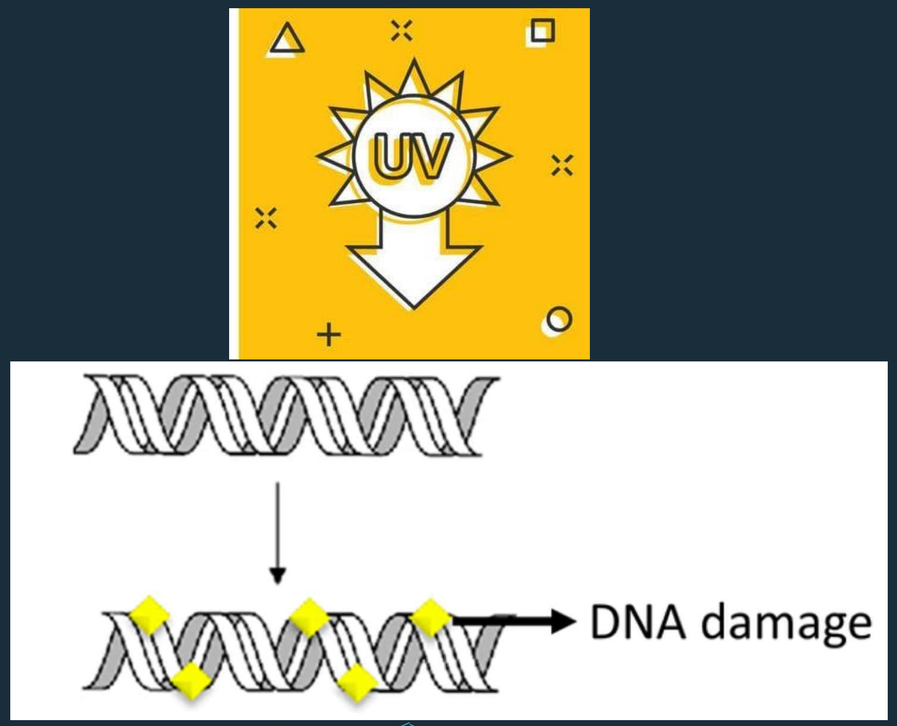
Lesional skin biopsies

Separate epidermis

RNA sequencing

Analyse differentially expressed miRNAs

Detection of UV-B mediated DNA damage in cells

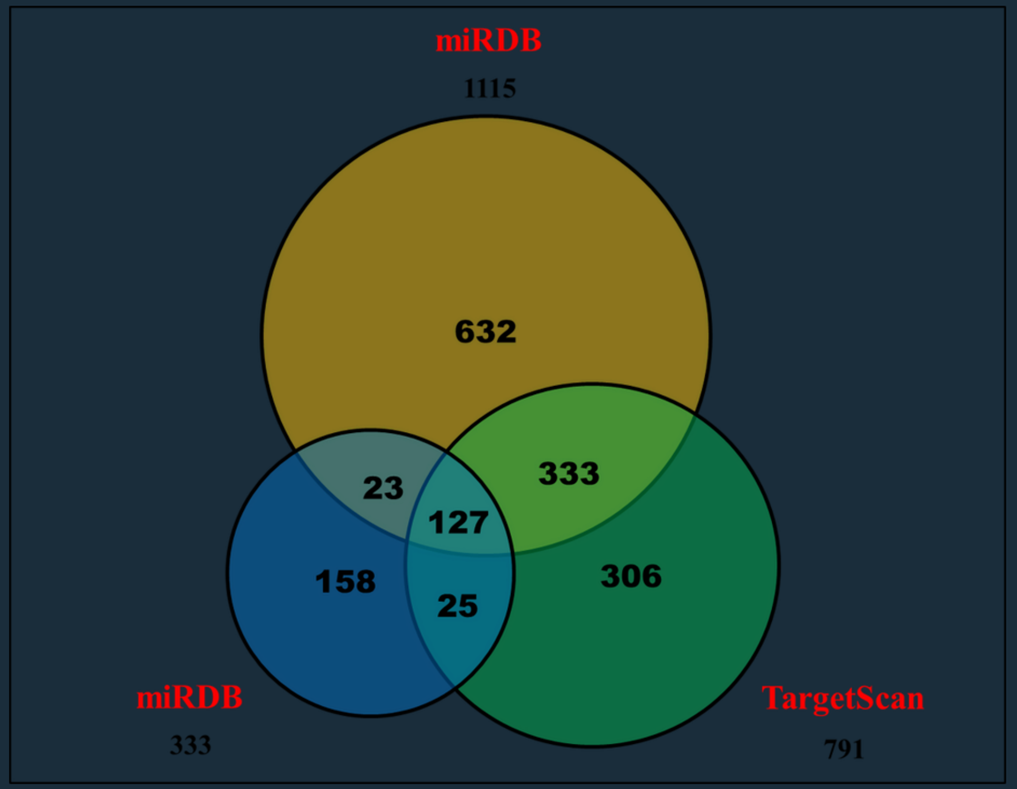


Detected with antibody against γ-H2AX

Detected with antibody against cyclobutane pyrimidine dimers

Bioinformatic tools

Used to predict the potential targets of the most differentially regulated miRNA



Results

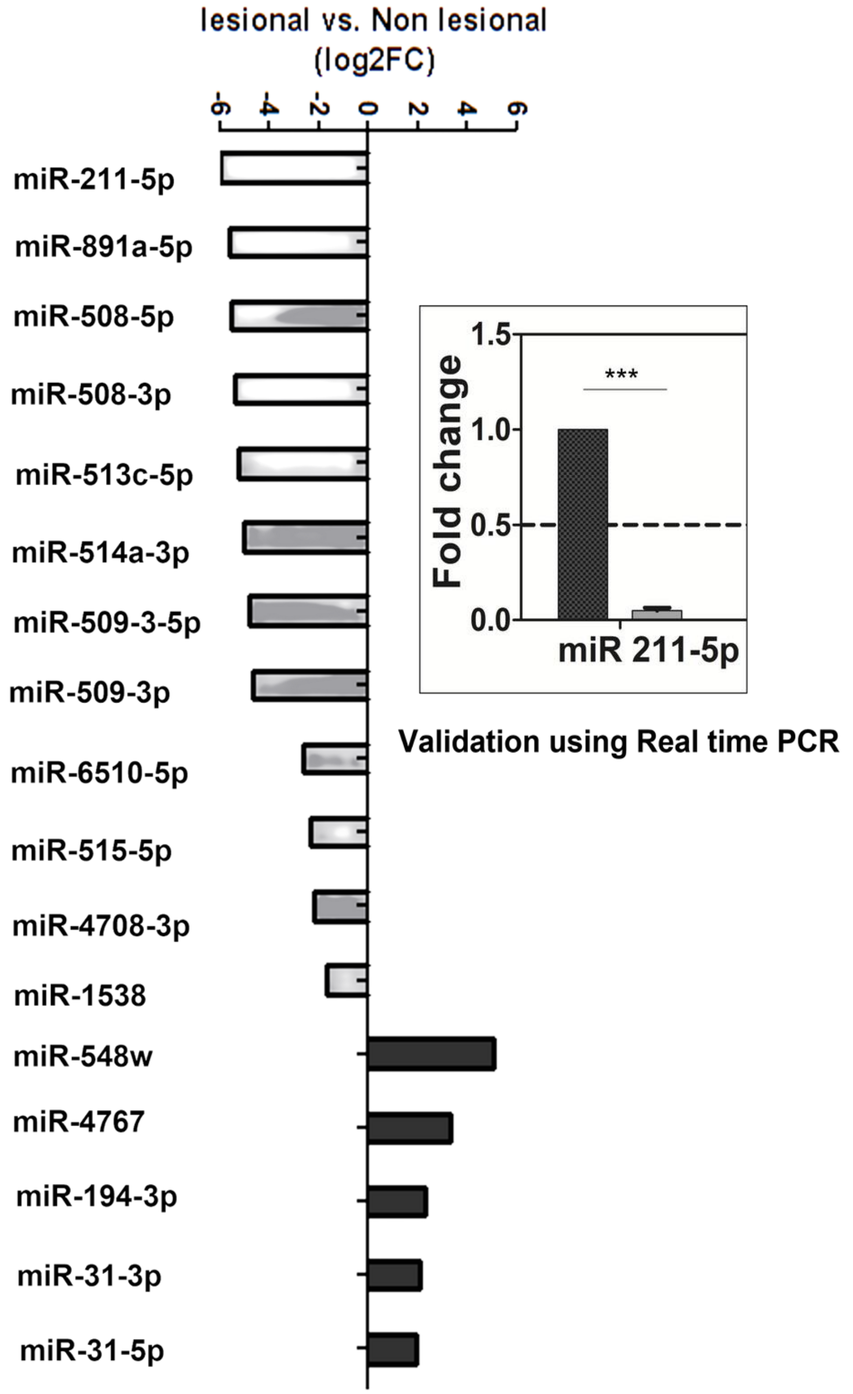
17 differentially regulated miRNAs in vitiligo
5 upregulated, 12 downregulated

miR 211-5p was the most downregulated
Suppresses keratinocyte proliferation and increases UV-mediated DNA damage

Sirtuin 1 is a target of miR-211-5p
Promotes keratinocyte differentiation and protects cells from UV-mediated DNA damage

MALAT1 (Metastasis associated lung adenocarcinoma transcript 1) promotes SIRT1 expression

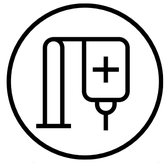
By inhibiting the expression of miR-211.
SIRT1 is the final effector protein



Novel pathway protects amelanotic cells of vitiligo

lncRNA MALAT1 suppresses miR 211 increasing the production of SIRT1 (the final effector protein)

What is SIRT1



NAD-dependent deacetylase

Regulates cell survival

Stress response

Genomic stability

The longevity factor

Controls metabolism

Prevents aging

and cancer

Increased SIRT1 leads to

Decreased γ-H2AX positivity

Decreased CPD formation

Hallmarks of UV related cell damage

Building on this breakthrough

Next gen activators of SIRT1

- Resveratrol - 1g/day to activate SIRT.

Oral sunscreens

- Application of sunscreens all over body - not practical
- Oral activators of SIRT1 - Better Compliance
- Used by wide range of individuals – SLE/ photoaging/ cancer

White armor of vitiligo

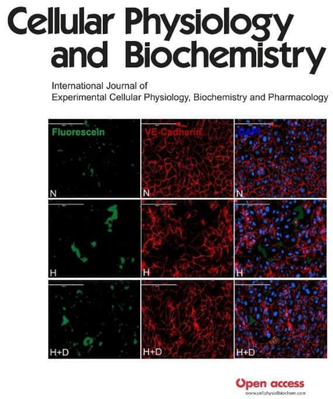
- Our study established that vitiligo patients have a reduced incidence of skin cancer, suggesting a potential protective effect of vitiligo-associated immunity.

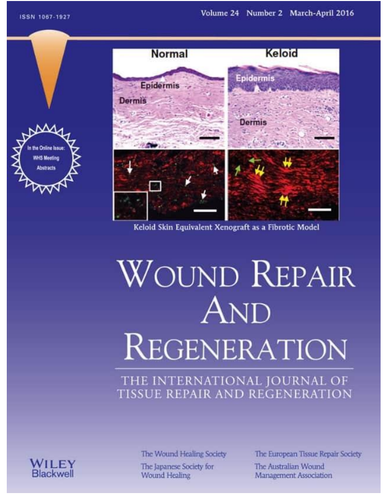
- A large population- based study in the USA of 25,000 vitiligo patients further confirmed lower cancer incidence rates.

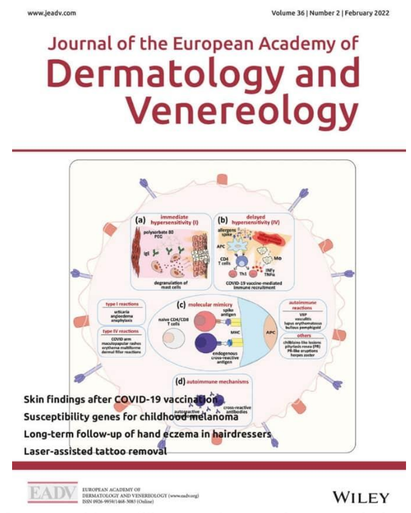
- Extending this concept, a nationwide study in Korea of 1,07,400 vitiligo patients found reduced all-cause and cause- specific mortality in vitiligo patients across various systemic diseases.

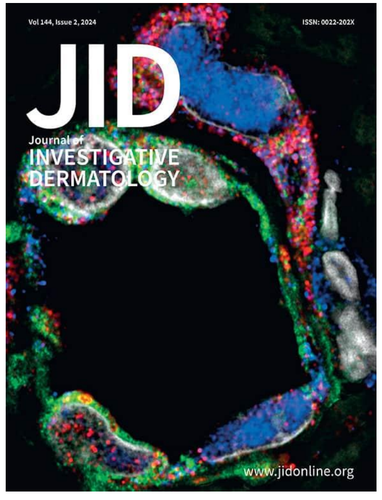
- This indicates that immune mechanisms in vitiligo may offer broader systemic benefits.

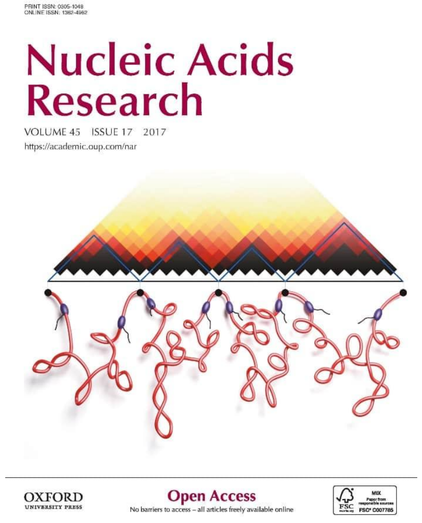
- To prove this concept, we are conducting a unique sibling-based study, comparing vitiligo –affected with their unaffected siblings, to investigate the protective effects of vitiligo beyond skin cancer.











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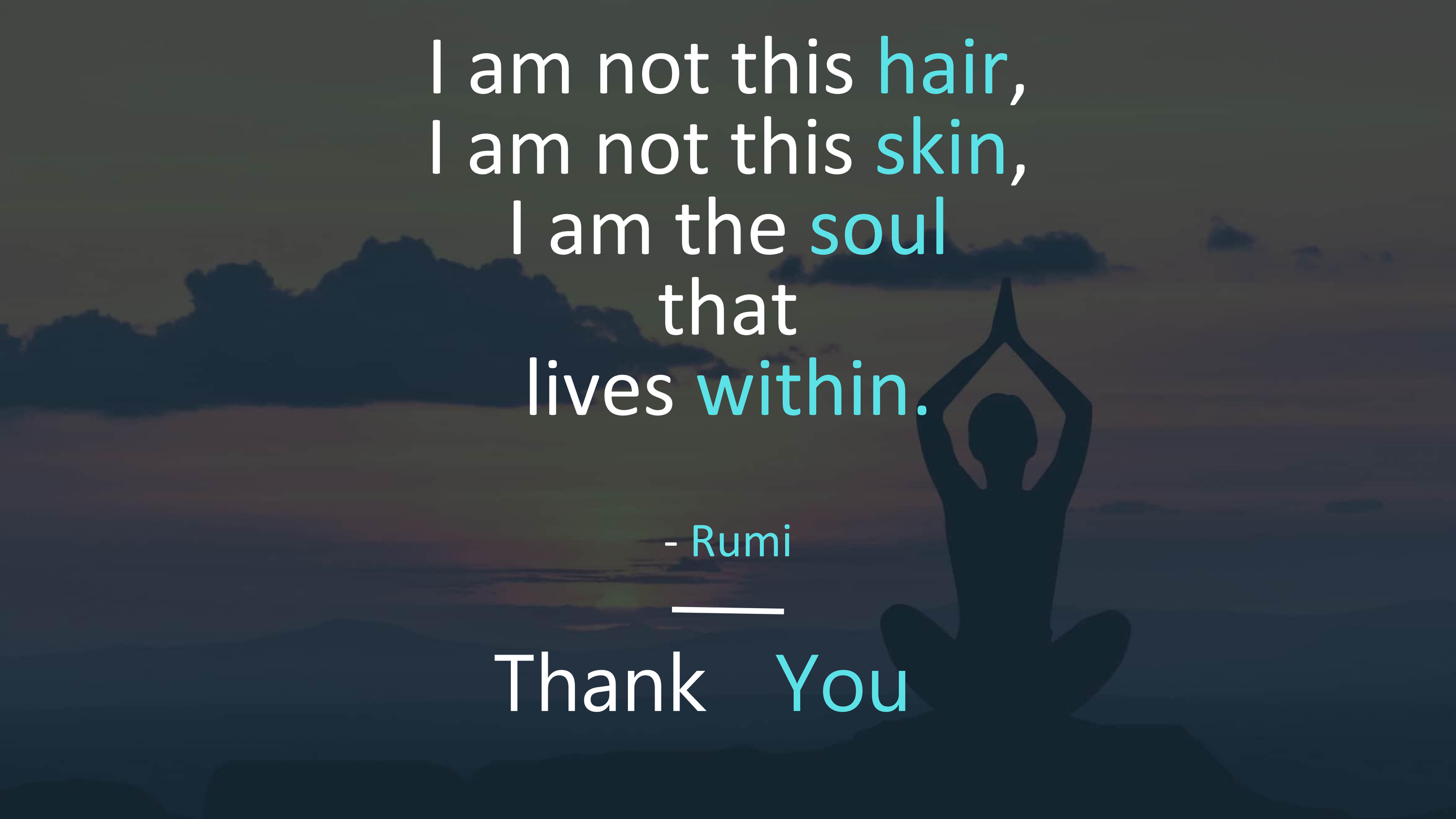
I am not this hair,
I am not this skin,
I am the soul
that
lives within.
- Rumi
Thank You