ĐIỂM BÁO DA LIỀU THÁNG 3/2023



TS.BS Nguyễn Trọng Hào Ngày 10/3/2023

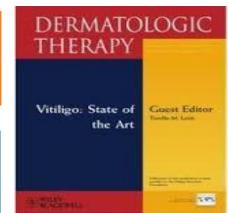


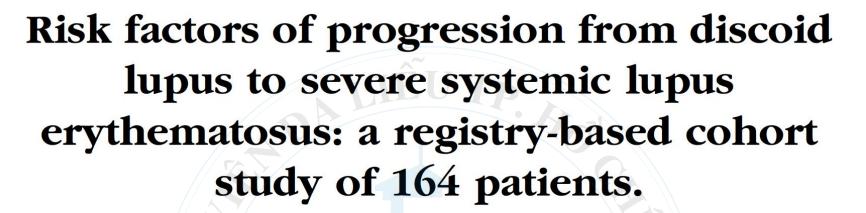
Dermatologic Clinics



British Journal of Dermatology









Lisa Fredeau, MD, Delphine S. Courvoisier, PhD, Raphael Ait Mehdi, MD, Saskia Ingen-Housz-Oro, MD, Emmanuel Mahe, MD, Nathalie Costedoat-Chalumeau, MD, PhD, Laurent Arnaud, MD, PhD, Camille Francès, MD, PhD, Alexis Mathian, MD, Marie Jachiet, MD, Zahir Amoura, MD, PhD, Jean David Bouaziz, MD, PhD, and François Chasset, MD, PhD, for the EMSED study group

Background: No study has assessed the risk factors of progression from discoid lupus erythematosus (DLE) to severe systemic lupus erythematosus (sSLE) (defined as requiring hospitalization and specific treatment).

Objective: To identify the risks factors of and generate a predicting score for progression to sSLE among patients with isolated DLE or associated with systemic lupus erythematosus with mild biological abnormalities.

Table IV. Generation of a **s**core associated with progression from discoid lupus erythematosus to severe systemic lupus erythematosus

| | Model 1* | | Model 2 [†] | | |
|------------------------------------|----------------|---------|---|---------|--------|
| Features | OR (95% CI) | P value | OR (95% CI) | P value | Points |
| Sociodemographic features | | | | | |
| Age at diagnosis of DLE < 25 y | 4.4 (1.5-13.4) | .0061 | 2.8 (1.1-7.0) | .0243 | 1 |
| Female sex | 4.1 (0.4-42.2) | .1822 | | | |
| Phototype V-VI | 4.0 (1.2-13.4) | .0189 | 2.7 (1.1-7.0) | .0364 | 1 |
| Clinical features | | | | | |
| Generalized DLE | 1.3 (0.4-4.2) | .6492 | \- | - | |
| Arthralgias | 1.5 (0.4-5.1) | .5183 | - | - | |
| Biological features at baseline | | | | - | |
| (presence) | | | | | |
| ANA titers ≥ 1:320 | 7.4 (1.2-46.3) | .0188 | 15 (3.3-67.3) | <.0001 | 5 |
| Anti-dsDNA | 2.5 (0.6-10.4) | .1987 | M | | |
| Anti-SSA | 2.0 (0.7-6.1) | .1999 | | - | |
| Anti-Sm | 3.0 (0.8-11.0) | .0990 | | - | |
| Anemia | 1.9 (1.2-22.4) | .6027 | | - | |
| Lymphopenia | 1.2 (0.4-3.6) | .7596 | - () | = | |
| DLE/SLE ACR/EULAR classification | | | | | |
| SLE according ACR 2019 at baseline | 2.3 (0.3-16.1) | .3837 | FIR - | - | |
| 8 | | | N' // | | |

ACR, American College of Rheumatology; ANA, antinuclear antibody; DLE, discoid lupus erythematosus; EULAR, European Alliance of Associations for Rheumatology; OR, odds ratio; SLE, systemic lupus erythematosus.

Fredeau L, et al. J Am Acad Dermatol. 2023 Mar;88(3):551-559

^{*}Model 1 includes all variables selected from the literature review (see Supplementary Table I).

[†]Model 2 includes variables selected by backward elimination.

Hindawi Dermatologic Therapy Volume 2023, Article ID 3427026, 6 pages https://doi.org/10.1155/2023/3427026



Research Article

Topical Calcipotriol for the Treatment of Cutaneous Warts: An Assessor-Blind Randomized Placebo-Controlled Trial

Armaghan Kazeminejad , ¹ Arezoo Bodaghi , ² Zohreh Hajheydari , ¹ Mahmood Moosazadeh , ³ Mohammadreza Rafati , ⁴ and Nasim Gholizadeh ¹

¹Department of Dermatology, Faculty of Medicine, Mazandaran University of Medical Sciences, Sari, Iran

²Student Research Committee, Faculty of Medicine, Mazandaran University of Medical Sciences, Sari, Iran

³Gastrointestinal Cancer Research Center, Non-Communicable Diseases Institute, Mazandaran University of Medical Sciences, Sari, Iran

⁴Department of Clinical Pharmacy, Pharmaceutical Sciences Research Center, Faculty of Pharmacy, Mazandaran University of Medical Sciences, Sari, Iran

TABLE 2: Comparison of the number and size of lesions between groups.

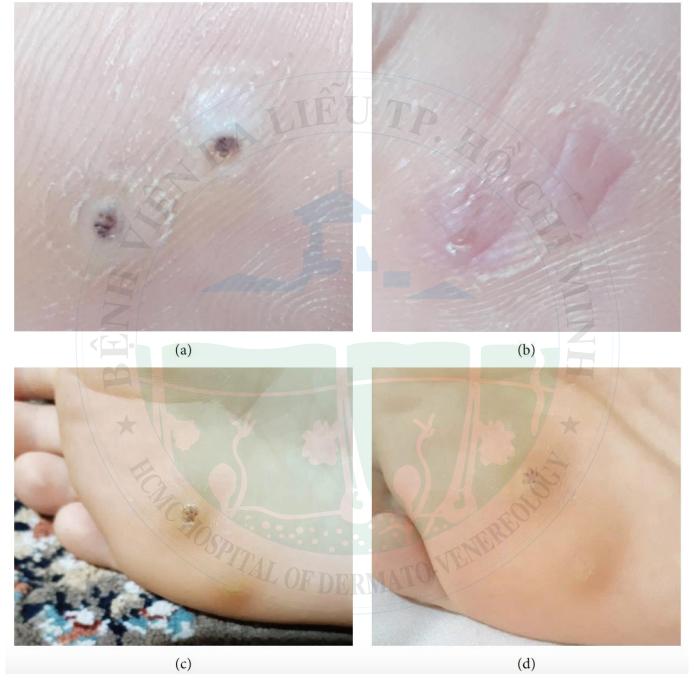
| Variables | Calcipotriol (n = 28) | Control $(n = 28)$ | P value* |
|---|-----------------------|--------------------|----------|
| Number of lesions, mean (SD) | TIELLTA | | |
| Before intervention | 5.54 (5.09) | 5.11 (3.75) | 0.927 |
| 1 st month | 2.89 (5.09) | 4.50 (3.92) | 0.009 |
| 2 nd month | 1.61 (3.79) | 3.96 (3.97) | 0.001 |
| 4 th month | 0.68 (3.02) | 3.72 (3.85) | < 0.001 |
| Size of lesions (cm ²), mean (SD) | | | |
| Before intervention | 0.44 (0.19) | 0.45 (0.15) | 0.699 |
| 1 st month | 0.22 (0.21) | 0.41 (0.19) | 0.001 |
| 2 nd month | 0.13 (0.19) | 0.37 (0.22) | < 0.001 |
| 4 th month | 0.06 (0.18) | 0.37 (0.22) | < 0.001 |

Abbreviations: N, number; SD, standard deviation. *Analyzed by the independent t-test.

Table 3: Comparison of adverse events and response to treatment after 4 months between groups.

| Variables | Calcipotriol $(n = 28)$ | Control $(n = 28)$ | P value* |
|--------------------------------|-------------------------|--------------------|----------|
| Response to treatment, N (%) | 24 (85.7) | 4 (16.0) | < 0.001 |
| Adverse events, N (%) | 4 (14.3) | 1 (3.6) | 0.352 |

Abbreviations: N, number. *Analyzed by Fisher's exact test.



Armaghan Kazeminejad, et al, *Dermatologic Therapy*, vol. 2023, Article ID 3427026, 6 pages, 2023. https://doi.org/10.1155/2023/3427026

Research

JAMA Dermatology | Original Investigation

Efficacy of Methotrexate Alone vs Methotrexate Plus Low-Dose Prednisone in Patients With Alopecia Areata Totalis or Universalis A 2-Step Double-Blind Randomized Clinical Trial

Pascal Joly, MD, PhD; Audrey Lafon, MD; Estelle Houivet, MS; Nathalie Donnadieu, MD;
Marie-Aleth Richard, MD, PhD; Alain Dupuy, MD, PhD; Emmanuel Delaporte, MD, PhD; Philippe Bernard, MD, PhD;
Laurent Machet, MD, PhD; Antonella Tosti, MD, PhD; Veronique Del Marmol, MD, PhD; Ramon Grimalt, MD, PhD;
Pierre A. de Viragh, MD, PhD; Jacques Benichou, MD, PhD; Olivier Chosidow, MD, PhD; Philippe Assouly, MD;
Pascal Reygagne, MD

IMPORTANCE Poor therapeutic results have been reported in patients with alopecia areata totalis (AT) or universalis (AU), the most severe and disabling types of alopecia areata (AA). Methotrexate, an inexpensive treatment, might be effective in AU and AT.

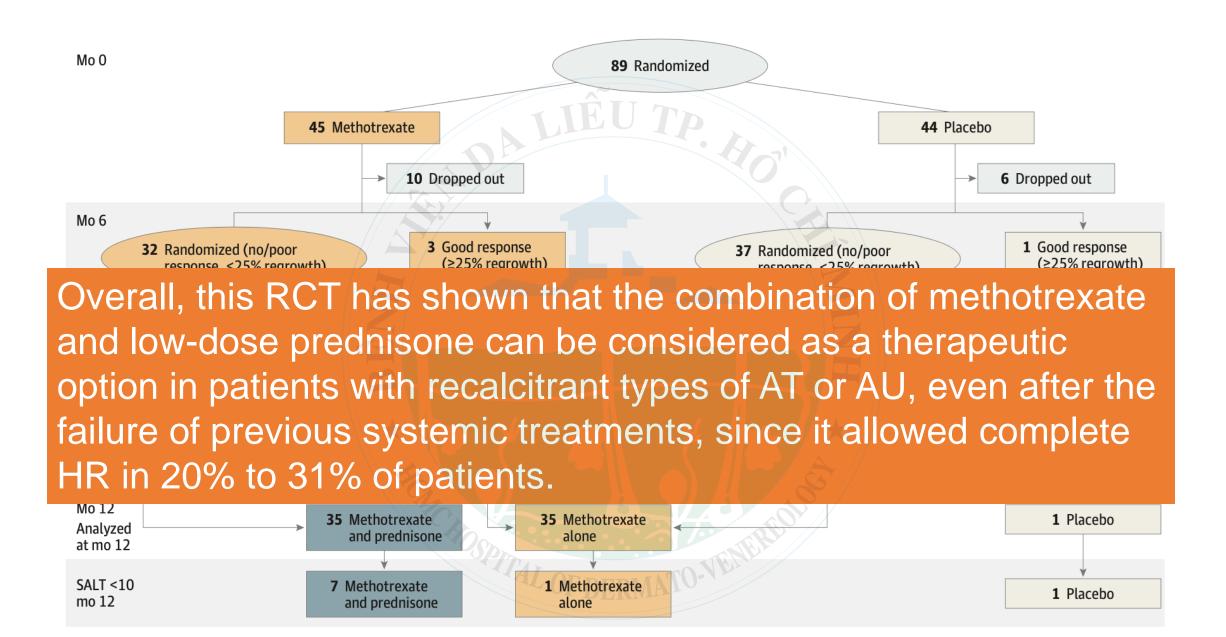
OBJECTIVE To evaluate the efficacy and tolerance of methotrexate alone or combined with low-dose prednisone in patients with chronic and recalcitrant AT and AU.

DESIGN, SETTING, AND PARTICIPANTS This academic, multicenter, double-blind, randomized clinical trial was conducted at 8 dermatology departments at university hospitals between March 2014 and December 2016 and included adult patients with AT or AU evolving for more than 6 months despite previous topical and systemic treatments. Data analysis was performed from October 2018 to June 2019.

+ Supplemental content







Simple techniques for circumferential cryoanesthesia



Mohammed I. AlJasser, MBBS, FRCPC

Key words: analgesia; anesthesia; circumferential; cooling; cotton; cryoanesthesia; freezing; ice; ring anesthesia; sponge.

SURGICAL CHALLENGE

Cooling is an effective method for anesthesia (cryoanesthesia) in a wide range of procedures in dermatology, including botulinum toxin injections for palmar hyperhidrosis. Ice can be applied directly to the skin then removed for the delivery of a relatively painless injection. However, having effective cryoanesthesia without the need to remove the ice instrument might be more practical and effective. Circumferential cryoanesthesia can be used to achieve this goal. This has been described with the use of a glove filled with water and allowed to freeze. Circumferential cryoanesthesia is then achieved by injecting between the glove fingers. However, the full glove might be quite bulky to handle and might not practically provide full circumferential cryoanesthesia.

THE SOLUTION

Cotton has been described as a method to hold water and is then frozen for use in dental anesthesia. However, this was not used to provide circumferential anesthesia. We describe 2 simple methods using sponge or cotton. A small hole is created in the middle of a sponge (Fig 1). The sponge is then soaked in water and allowed to freeze. It is important to place a plastic sheet beneath the sponge in the freezer to hold the absorbed water in place. Sometimes a thin layer of ice develops in the hole, which can be easily broken. The frozen sponge can then be used for circumferential anesthesia (Fig 1). The smaller the created hole is, the more effective the anesthesia will be. Cotton can be used in a similar manner, as shown in Fig 2.





AlJasser MI. J Am Acad Dermatol. 2023 Mar;88(3):e119-e121

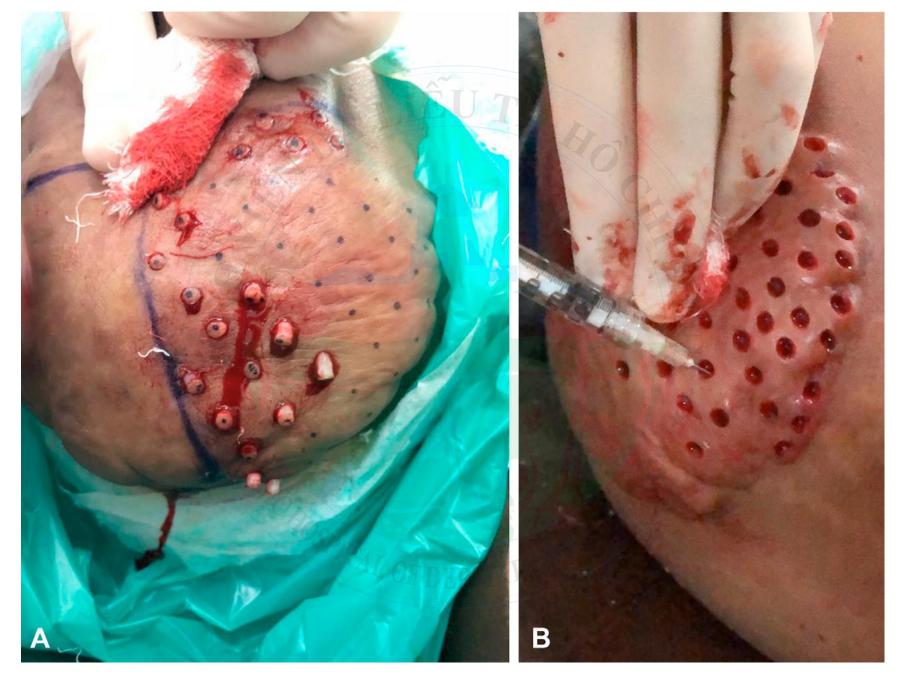


Fractionated devolumizing keloid tissue: The 'pop' method: A novel technique to facilitate administration of intralesional corticosteroid in difficult keloids

Rachita Dhurat, MBBS, MD, Sanober B. Daruwalla, MBBS, and Aseem Sharma, MBBS, MD, DNB *Mumbai, India*

SURGICAL CHALLENGE

Intralesional steroid is the first-line accepted treatment for keloid. It has been a challenge for most clinicians to inject triamcinolone acetonide into large compact keloids. Cryotherapy has been tried to soften such keloids. However, cryotherapy gives undesirable side effects such as ulceration and secondary infection. Surgical excision of keloid tissue can trigger the formation of a similar or even larger keloid. We propose the use of a simple biopsy punch, which is available in the everyday dermatology outpatient department, to achieve effective reduction of keloidal tissue and facilitate easy administration of intralesional agents.



Dhurat R, Daruwalla SB, Sharma A. J Am Acad Dermatol. 2023 Feb;88(2):e75-e77.



Dhurat R, Daruwalla SB, Sharma A. J Am Acad Dermatol. 2023 Feb;88(2):e75-e77.

A potassium hydroxide fountain pen for precise application over molluscum contagiosum lesions

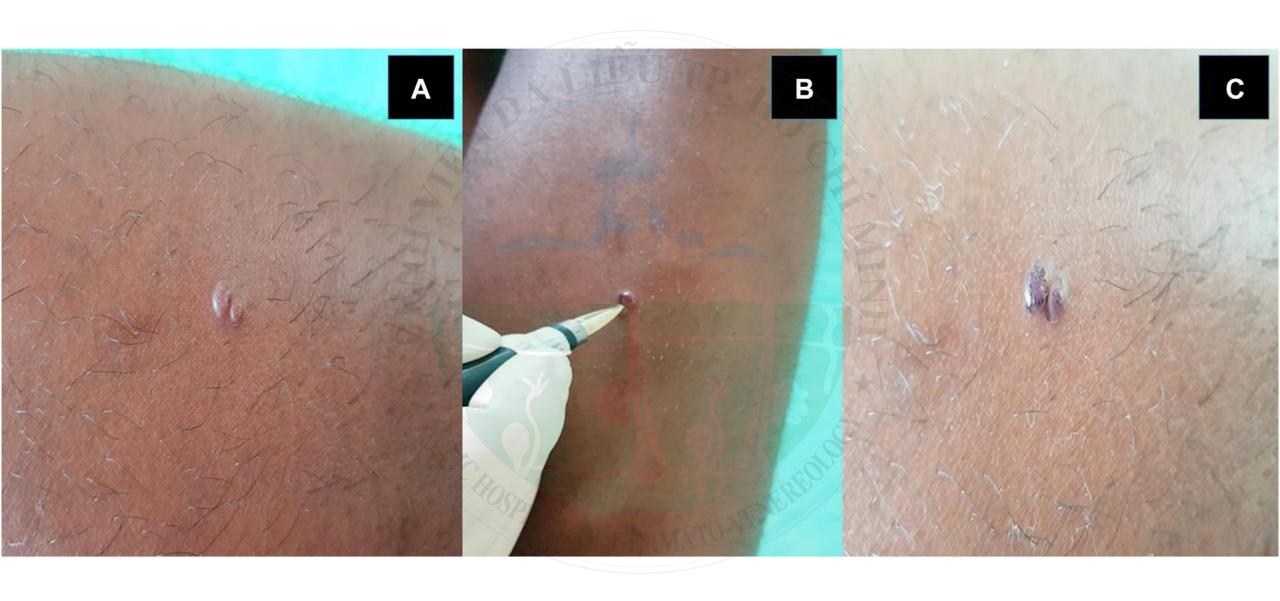


Sandip Agrawal, MD, Aseem Sharma, MD, Maryam Motiwala, MD, and Rachita Dhurat, MD *Mumbai, India*

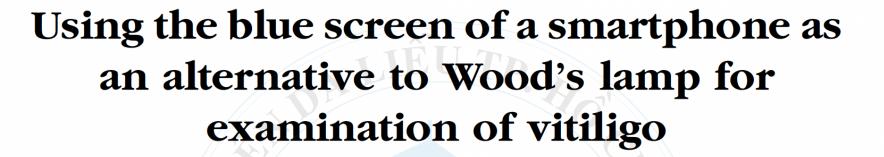
Key words: fountain pen; irritant contact dermatitis; methylene blue; molluscum contagiosum; potassium hydroxide; toothpick.

THERAPEUTIC CHALLENGE

Potassium hydroxide (KOH), as a 10% solution, is one of the most popular treatment modalities for molluscum contagiosum. It is administered as an office-based therapy or as a home-based once-daily regimen to the patient's guardian. A common untoward effect is that spillage of KOH onto healthy skin can cause irritant contact dermatitis or postinflammatory hyperpigmentation, or both. Hence, the advice given to the parent or guardian is to apply medication just over the dome of the papular lesion, taking due care to prevent trickling and dripping. Traditionally, a toothpick or cotton bud is used to apply KOH over such lesions. However, limiting the application to the lesions is often challenging.



Agrawal Set al. J Am Acad Dermatol. 2023 Jan;88(1):e7-e8.



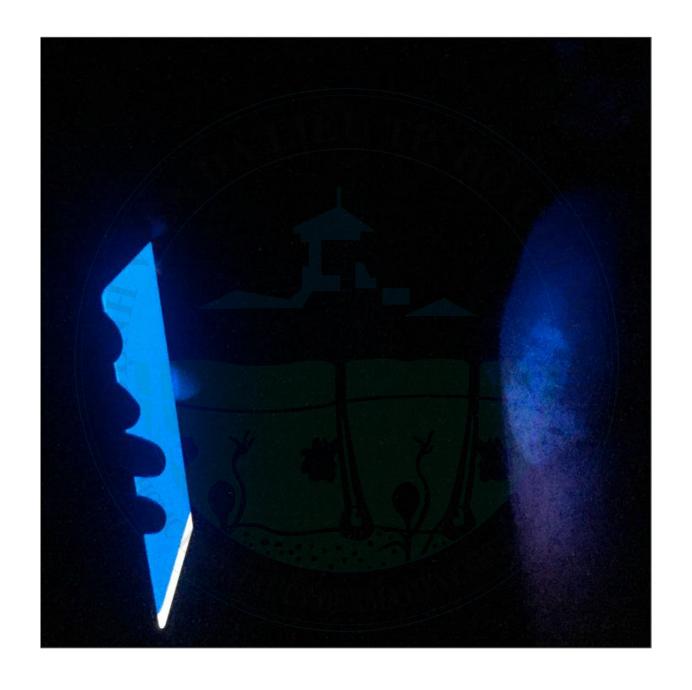


Sandip Agrawal, MD, Aseem Sharma, MD, Rachita Dhurat, MD, and Kiran Chahal, MD

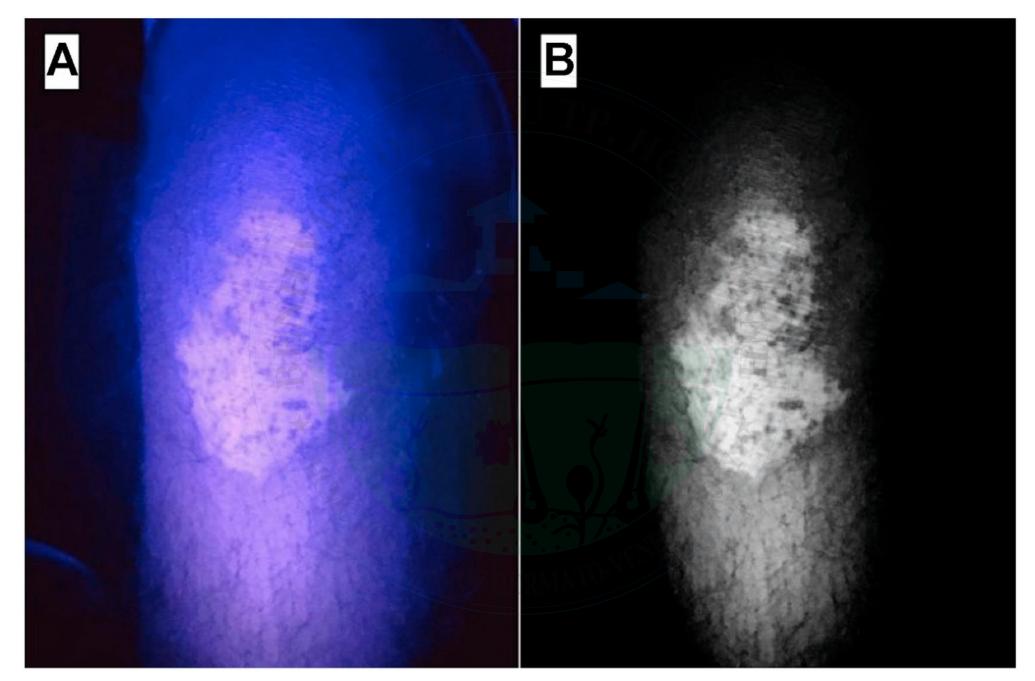
Key words: accentuation; clinical examination; grayscale modification; smartphone; violet light; vitiligo; Wood's lamp.

CLINICAL CHALLENGE

The Wood's lamp is an instrument that is commonly used as a bedside investigative modality. Vitiligo is a common, autoimmune disorder characterized by depigmentation of the skin and mucosal surfaces. When the radiation (wavelength, 320-400 nm) from a Wood's lamp falls on a vitiliginous lesion, the lack of epidermal melanin facilitates visible autofluorescence of dermal collagen, thereby accentuating the lesion. This accentuation is used to differentiate vitiligo from other disorders with hypopigmentation and depigmentation. In resource-poor settings, the Wood's lamp is often not available, and this may hamper vitiligo examination. We describe herein use of the blue screen of a smartphone as a readily available alternative to Wood's lamp.



Agrawal S, et al. J Am Acad Dermatol. 2023 Jan;88(1):e5-e6



Agrawal S, et al. J Am Acad Dermatol. 2023 Jan;88(1):e5-e6

CONTINUING MEDICAL EDUCATION

Disorders of hyperpigmentation. Part I. Pathogenesis and clinical features of common pigmentary disorders



Rebecca F. Wang, MD, Dayoung Ko, MD, Ben J. Friedman, MD, Henry W. Lim, MD, and Tasneem F. Mohammad, MD

Detroit, Michigan

Learning objectives

After completing this learning activity, participants should be able to classify select disorders of hyperpigmentation as epidermal, dermal, or mixed hyperpigmentation; recognize differences in etiology and presentation of drug-induced hyperpigmentation; describe key features of mucosal and nail hyperpigmentation; and recognize and compare the common clinical and histologic features of hyperpigmentation.

Disclosures

Editors

The editors involved with this CME activity and all content validation/peer reviewers of the journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Planners

Jeremy Brauer, MD, FAAD, is a consultant for Cutera, Inc. and Solta Medical and receives honoraria. Matthew Zirwas, MD, FAAD, is a consultant and investigator for Galderma Global and receives honoraria and grants/research funding.

Reviewers

Kara Melissa Tiangco Torres-Culala is a speaker for Galderma and receives honoraria. George Hruza, MD, MBA, FAAD, is a stockholder for Teva and does not receive compensation.





Dayoung Ko, MD, Rebecca F. Wang, MD, David Ozog, MD, Henry W. Lim, MD, and
Tasneem F. Mohammad, MD

Detroit, Michigan

Learning objectives

After completing this learning objective, the reader will be able to better discuss this aspect of the literature.

Disclosures

Editors

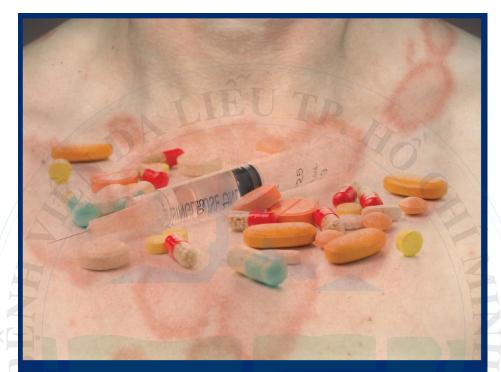
The editors involved with this CME activity and all content validation/peer reviewers of the journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Planners

Jeremy Brauer, MD, FAAD, is a consultant for Cutera, Inc. and Solta Medical and receives honoraria. Matthew Zirwas, MD, FAAD, is a consultant and investigator for Galderma Global and receives honoraria and grants/research funding.

Reviewers

Kara Melissa Tiangco Torres-Culala is a speaker for Galderma and receives honoraria. George Hruza, MD, MBA, FAAD, is a stockholder for Teva and does not receive compensation.



Handbook of Systemic Drug Treatment in Dermatology

Third Edition

Edited by

Sarah H. Wakelin Howard I. Maibach Clive B. Archer



Contents

| | ix |
|---------------|--|
| | eviationsx |
| LIST OF AUDIO | eviations |
| | |
| Chapter 1 | Acitretin1 |
| | Peter C.M. van de Kerkhof |
| | |
| Chapter 2 | Acne Antibiotics |
| | Alexander Nast, Ricardo N. Werner and Natalie R. Attard |
| Chanter 3 | Alitretinoin 13 |
| Chapter 5 | N. Farah Mahmood and Graham A. Johnston |
| | N. Faran Manmood and Granam A. Johnston |
| Chapter 4 | Antiandrogens19 |
| | Shirin Zaheri and Iaisha Ali |
| | |
| Chapter 5 | Antibiotics Commonly Used for Skin Infections |
| | Hui Min Liew, Victoria J. Hogarth and Roderick J. Hay |
| Chapter 6 | Antifungals |
| Chapter 0 | |
| | Rachael Morris-Jones |
| Chapter 7 | Antimalarials |
| | Thomas Tull and Mark Goodfield |
| | |
| Chapter 8 | Antivirals for Herpesviruses67 |
| | Hannah Cookson |
| Cl | Annual Control of the |
| Cnapter 9 | Apremilast |
| | Gayathri K. Perera |
| Chapter 10 | Azathioprine |
| • | Sarah H. Wakelin and Simon Meggitt |
| | 00 |
| Chapter 11 | Azole Antihelminths |
| | Mahreen Ameen |
| | |

| Chapter 12 | Bexarotene 88 |
|------------|--|
| | Julia J. Scarisbrick |
| | |
| Chapter 13 | Ciclosporin |
| | Murtaza Khan and John Berth-Jones |
| Chapter 14 | Colchicine |
| | Esther A. Hullah and Michael P. Escudier |
| | |
| Chapter 15 | Corticosteroids |
| | Clive B. Archer |
| Chapter 16 | Cyclophosphamide |
| | Matthew Scorer, Olga Golberg and Karen Harman |
| | |
| Chapter 17 | Dapsone |
| | Charles Archer and Antonia Lloyd-Lavery |
| Chapter 18 | Dimethyl Fumarate |
| Chapter 16 | Oonagh Molloy and Brian Kirby |
| | Conagn money and Brian Kirby |
| Chapter 19 | Interleukin-4/13 Inhibitors |
| | Alison Sears and Andrew Pink |
| Chapter 20 | Interleukin (IL)-12/23 and IL-23 Inhibitors |
| Chapter 20 | Charles H. Earnshaw and Richard B. Warren |
| | Charles H. Earnsnaw and Richard B. warren |
| Chapter 21 | Interleukin-17 Inhibitors |
| | Leena Chularojanamontri and Christopher E.M. Griffiths |
| Chartan 22 | Isotretinoin 151 |
| Chapter 22 | |
| | Alison M. Layton |
| Chapter 23 | Ivermectin |
| | Jonathan Kentley and L. Claire Fuller |
| | |
| Chapter 24 | Intravenous Immunoglobulin |
| | Derrick Phillips |
| Chapter 25 | Janus Kinase (JAK) Inhibitors |
| | Courtney E. Heron, Lindsay C. Strowd and Steven R. Feldman |

| Chapter 26 | Malignant Melanoma Drug Treatments | 177 |
|------------|--|-------|
| | Charlotte L. Edwards and Louise A. Fearfield | |
| Chapter 27 | Methotrexate | 195 |
| | Richard Woolf and Catherine Smith | |
| Chapter 28 | Mycophenolate Mofetil | 203 |
| | Joey E. Lai-Cheong and Jane Setterfield | |
| Chapter 29 | Omalizumab | 208 |
| | Chris Rutkowski, Annette Wagner and Clive Grattan | |
| Chapter 30 | Potassium Iodide | 212 |
| | Genevieve Osborne | |
| Chapter 31 | Propranolol | 215 |
| • | Susannah Baron | |
| Chapter 32 | Psoralens | 220 |
| • | Hiva Fassihi | |
| Chapter 33 | Rituximab | 228 |
| | Richard W. Groves | |
| Chapter 34 | Sulfapyridine and Sulfamethoxypyridazine | 233 |
| • | Ellie Rashidghamat | |
| Chapter 35 | Thalidomide | 239 |
| • | Elaine Agius and Robert P.E. Sarkany | |
| Chapter 36 | Tumour Necrosis Factor Antagonists | 245 |
| | Leena Chularojanamontri and Christopher E.M. Griffiths | |
| Chanter 37 | Vismodegib | 253 - |
| Chapter 57 | Faisal R. Ali and John T. Lear | TE. |
| Chanter 38 | Systemic Therapy in Children and Young People | 257 |
| Chapter 50 | Susannah Baron | 25 / |
| Chanter 20 | Systemic Thereny and Vidney Disasce | 266 |
| Chapter 39 | Systemic Therapy and Kidney Disease | 200 |

| Chapter 40 | Systemic Therapy and Liver Disease | 0 |
|------------|---|---|
| | Maria Bashyam and Shahid A. Khan | |
| Chapter 41 | Systemic Therapy in Older People | 9 |
| | Sarita Singh, Rosanna Fox and Manuraj Singh | |
| Index | | 4 |

14 Colchicine

Esther A. Hullah and Michael P. Escudier

CLASSIFICATION AND MODE OF ACTION

Colchicine is a naturally occuring alkaloid, obtained from plants of the lily family, including Colchicum autumnale, which has been used for medicinal purposes since the time of ancient Greece. It occurs as pale to greenish yellow crystals, is irritating to skin lipid soluble and oxidizes rapidly on light exposure, so it must be stored in the dark.

Following oral administration, colchicine is rapidly absorbed in the jejunum and ileum, and it undergoes significant hepatic first-pass metabolism. While there is wide individual variability in bioavailability, primarily due to differing expression of protein targets, including CYP34A and P-glycoprotein, peak plasma levels are generally achieved after 30–120 minutes and within leucocytes at around 48 hours.

Colchicine is primarily metabolized in the liver, by the cytochrome P450 enzyme CYP3A4, via demethylation before hepatobiliary excretion in the stool. The remaining 10–20% is excreted unchanged in the urine, such that renal impairment may prolong the half-life and increase the risk of toxicity.

The cellular effects of colchicine, including its anti-inflammatory action, are primarily accounted for by its inhibition of microtubule polymerisation and the resultant effects on cell division and migration, signal transduction, gene expressions and cellular transport. Other mechanisms which may play a role include inhibition of cyclooxygenases COX-1 and COX-2.

Colchicine is seen at particularly high concentrations in leucocytes and impairs neutrophil function, macrophage activation and mast cell degranulation.

INDICATIONS AND DERMATOLOGICAL USES

Colchicine is licensed only for use in gout and familial Mediterranean fever (FMF), with all other uses being off-licence.

Gout and pseudogout

The inflammation in gout is mediated by neutrophil and macrophage activation, leucocyte adhesion molecules, inflammasome activation and IL-1 β production, all of which are inhibited by colchicine.

Colchicine is used in the management of acute gout and also as a prophylactic agent against acute flares when initiating allopurinol in patients with chronic gout. It is also recommended for pseudogout or calcium pyrophosphate crystal arthritis prophylaxis and for acute episodes.

FMF

This condition is associated with a mutation in the gene that encodes the protein pyrin, which regulates inflammasome activation, resulting in increased IL-1 β production and inflammation resulting in fever, arthritis and serositis.

Colchicine is the primary treatment for FMF and has good long-term efficacy and safety and proven safety in children, during pregnancy and with nursing patients.

At daily doses of 1.0–2.0 mg, it reduces the frequency of FMF attacks and prevents the development of amyloidosis and subsequent chronic renal failure. It can also induce remission of proteinuria and nephrotic syndrome in patients with established amyloid nephropathy.

Additional indications

The range of potential uses of colchicine has broadened significantly, particularly within dermatology and oral medicine. However, in many cases the current evidence base is limited to case series or case reports.

The following are the exceptions, having been the subject of controlled trials.

- Behçet's Disease: Colchicine significantly reduces the frequency of oral and genital ulceration, pseudofolliculitis and erythema nodosum.
- Chronic Urticaria: Over half of patients treated for a mean period of 7 months achieved a
 partial or complete response.
- Epidermolysis Bullosa Acquisita: Colchicine is an effective monotherapy and acts through its inhibition of secretion of anti-type VII collagen antibody, with improvement in dysphagial symptoms, mucosal erosions, oesophageal stenosis and cutaneous erosions
- Leucocytoclastic Vasculitis: While several case reports support the efficacy of colchicine
 in this group of conditions, a prospective randomized controlled trial using 0.5 mg per day
 did not confirm this.
- Recurrent Aphthous Stomatitis: Colchicine at a dose of 0.5–1.5 mg per day is often effective in reducing the severity (symptoms, size and durations of lesions) as well as the frequency of attacks over a period of 3 months.
- Sweet's Syndrome (Acute Febrile Neutrophilic Dermatosis): Over half of patients show regression of lesions at a dose of 1.0–2.0 mg per day with the remainder responding with the addition of prednisolone.

FORMULATION/PRESENTATION

Oral administration is the most common route of administration. Intravenous delivery, while quicker-acting, is associated with a significantly increased risk of toxicity. In contrast topical use is less well evidenced in the literature.

The starting dose is 0.5 mg daily, increasing to 1–1.5 mg in divided (0.5 mg) doses, as tolerated. The dose tolerated by the patient is usually dependent on gastrointestinal (GI) side effects.

The usual maintenance dose is 0.5–2.0 mg, daily although dosage adjustment is required in patients with moderate-to-severe renal impairment or hepatobiliary dysfunction. Tolerance is improved by delivering the drug in divided doses, while long-term studies have confirmed its safety in chronic use.

SPECIAL POINT

Colchicine has a narrow therapeutic window and a significant variation in the dose required to cause morbidity or mortality, and as little as 6–7 mg has caused death. Toxicity typically manifests as GI symptoms, with the later development of widespread organ dysfunction including renal failure, arrhythmias and heart failure.

As mentioned above, its hepatobiliary and renal excretion means that impairment of either pathway can result in its accumulation and toxicity. Similarly, colchicine overdose can arise from drug interactions, e.g. antibiotics, antifungals, calcium channel blockers, immunosuppressants and statins. Acute toxicity manifests as cholera-like symptoms and signs of dehydration, electrolyte disturbance, metabolic acidosis, renal failure and shock. Convulsions, muscle paralysis, neuropathy and respiratory distress are common.

BASELINE INVESTIGATIONS AND CONSIDERATIONS

- Full blood count (FBC) (complete blood count [CBC]).
- · Urea, electrolytes and creatinine.
- · Liver function tests (LFTs).

MONITORING

- FBC (CBC) and differential white cell count every month.
- LFTs and renal indices every 3 months.

CONTRAINDICATIONS

- Haematological disease.
- Severe renal impairment or haemodialysis (colchicine cannot be removed by dialysis or exchange transfusion).

CAUTIONS

Care should be exercised in patients who:

- Are older or debilitated as they may be especially susceptible to cumulative toxicity, which leads to GI, renal, hepatic, cardiac or haematological complications.
- Have gastrointestinal hepatic or cardiac disease who are at increased risk of developing toxicity.
- Have renal impairment or are concurrently using nephrotoxic drugs as they are at a greater risk of toxicity.
- Have chronic kidney disease i.e. stage 5 with a glomerular filtration rate (GFR) <15 mL/min.

IMPORTANT DRUG INTERACTIONS

- Macrolide antibiotics (erythromycin, clarithromycin and others) increase the risk of colchicine toxicity, due to interactions with the cytochrome P450 (CYP450) 3A4 microsomal enzyme system.
- Ciclosporin (cyclosporine) concentrations are increased by colchicine, with an increased risk of nephrotoxicity and neuromuscular adverse effects.
- Vitamin B12 absorption may be impaired by colchicine, resulting in megaloblastic anaemia.
- Statins and fibrates may cause acute myopathy when given with colchicine. Patients should be advised to report any muscular pain or weakness.
- Other potential interactions include drugs metabolized by CYP3A4, including azole antifungals, antiviral drugs and cardiac medication.

ADVERSE EFFECTS AND THEIR MANAGEMENT

Colchicine is generally well-tolerated. The most common unwanted effects are:

- Gastrointestinal: Nausea, vomiting and diarrhoea, which may worsen with increasing dosage, and occur in 5–10% of patients. Less commonly elevation of liver transaminases can also occur as malabsorption syndrome, affecting B12, fat, protein, actively transported sugars and electrolytes. Other gastrointestinal effects include paralytic ileus.
- Bone marrow suppression: Agranulocytosis, thrombocytopenia and aplastic anaemia can
 occur after prolonged treatment. Administration of granulocyte colony stimulating factor
 (G-CSF) should be considered in such cases.
- Myopathy and neuropathy: This occurs especially in patients with renal impairment. The
 myopathy is proximal, with elevated serum creatinine phosphokinase, and the neuropathy
 is axonal. Myopathy recovers on withdrawal of colchicine, but neurological recovery may
 be slow.
- Dermatological: These include urticaria; rarely, Stevens

 –Johnson syndrome, toxic epidermal necrolysis and alopecia universalis; and very rarely, porphyria cutanea tarda.

There have been isolated reports of bladder spasms, renal damage and haematuria, and hypothyroidism.

USE IN SPECIAL SITUATIONS

PREGNANCY

The use of colchicine in pregnancy is controversial as a result of the potential risks and should only be considered if the potential benefits to the mother outweigh the possible risk to the fetus, e.g. FMF.

A systematic review and meta-analysis of four papers addressing the use of colchicine throughout pregnancy, for FMF, did not reveal an increased incidence of miscarriage or major fetal malformations. In fact, the incidence of miscarriage was significantly lower in patients who took colchicine compared with those who did not. There was also no significant difference in birth weight or gestational age between those who did and did not take colchicine. However, when not limited to FMF, colchicine use was associated with a significantly lower birth weight and gestational age compared with a control group including, healthy patients who did not take colchicine.

LACTATION

While colchicine is excreted into breast milk, there are no reported adverse effects in breastfed infants and it is therefore considered compatible with breastfeeding.

CHILDREN

Safe use of colchicine in children has been reported in a number of case reports, e.g. as an adjuvant treatment with systemic corticosteroids in children with linear IgA disease.

ESSENTIAL PATIENT INFORMATION

Patients should be advised:

- · To reduce dosage of colchicine if weakness, anorexia, nausea, vomiting or diarrhoea occurs.
- · Of the dangers of overdosage.
- That as colchicine can adversely affect the fetus, pregnancy should be avoided during treatment.



NEWS & PERSPECTIVE

DRUGS & DISEASES

CME & EDUCATION

ACADEMY

VIDEO

DECISION POINT

News > Quick Take

Closing the Toilet Lid Before Flushing Is Important

Medscape Staff February 14, 2023











Laser lights expose the otherwise invisible aerosol spray that is sent into the air by flushing a toilet with the lid up. The lasers show how pathogens from human waste can spread in public restrooms and can potentially expose people to contagious diseases, according to the results of tests conducted by a group of civil, environmental, and architectural engineers from the University of Colorado Boulder.



Recommendations

H1N1 Influenza A (Swine Flu) Alert Center Resource Center



Prevention and Control of Seasonal Influenza With Vaccines: Recommendations

of the Advisory Committee on Immunization Practices — United States, 2022–23 Influenza Season

2.5 CME / CE / ABIM MOC Credits



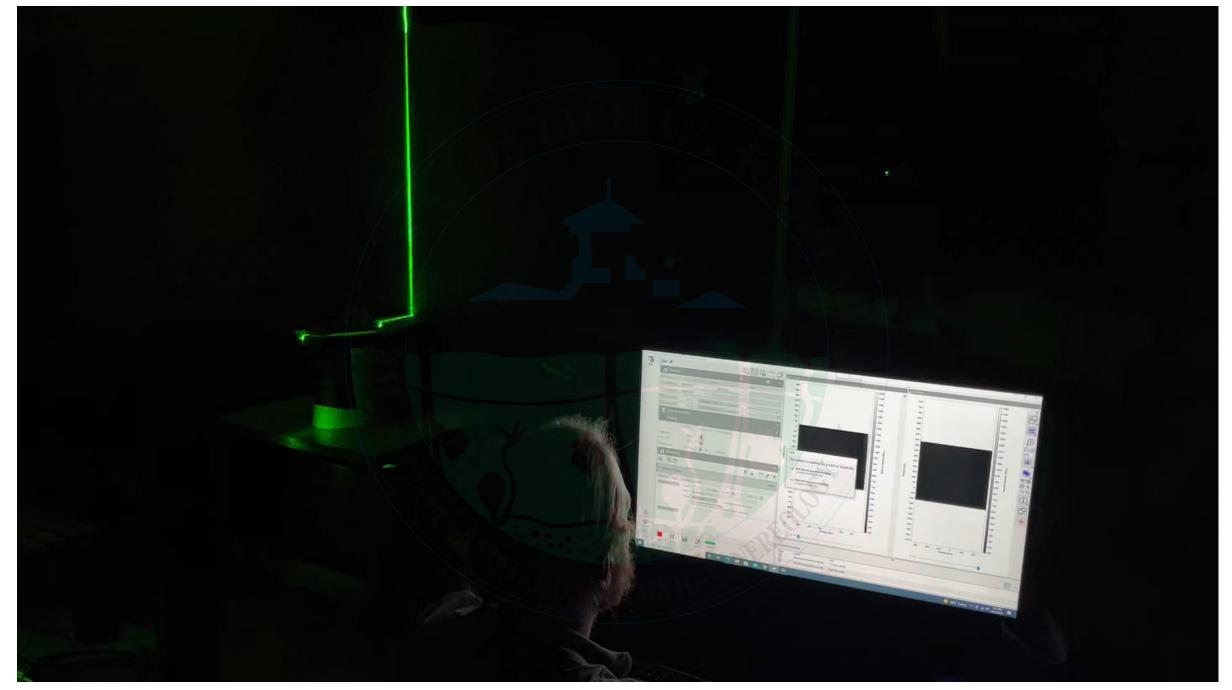
NUDGE-FLU: Electronic 'Nudges' Boost Flu Shot Uptake in Seniors



https://theconversation.com/toilets-spew-invisible-aerosol-plumes-with-every-flush-heres-the-proof-captured-by-high-powered-lasers-195717



https://theconversation.com/toilets-spew-invisible-aerosol-plumes-with-every-flush-heres-the-proof-captured-by-high-powered-lasers-195717



https://theconversation.com/toilets-spew-invisible-aerosol-plumes-with-every-flush-heres-the-proof-captured-by-high-powered-lasers-195717

Summary

- Risk factors of progression from discoid lupus to severe systemic lupus erythematosus: a registry-based cohort study of 164 patients
- Topical Calcipotriol for the Treatment of Cutaneous Warts: An Assessor-Blind Randomized Placebo-Controlled Trial
- 3. Efficacy of Methotrexate Alone vs Methotrexate Plus Low-Dose Prednisone in Patients With Alopecia Areata Totalis or Universalis A 2-Step Double-Blind Randomized Clinical Trial
- 4. Simple techniques for circumferential cryoanesthesia
- 5. Fractionated devolumizing keloid tissue: The 'pop' method: A novel technique to facilitate administration of intralesional corticosteroid in difficult keloids
- 6. A potassium hydroxide fountain pen for precise application over molluscum contagiosum lesions
- Using the blue screen of a smartphone as an alternative to Wood's lamp for examination of vitiligo
- 8. Disorders of hyperpigmentation. Part I, II.
- 9. Book review: Handbook of Systemic Drug Treatment in Dermatology (3rd edition, 2023)
- 10. Closing the Toilet Lid Before Flushing Is Important

