YUVA DERMA E-BULLETIN

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YUVADERMA
E-Bulletin

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October 2022, Vol. 1, Issue 2

EDITOR'S NICHE

"Any fool can know. The point is to understand."

- Albert Einstein

We know there's no dearth for information available today due to scientific advances. But applying the information acquired into practice requires a deeper understanding of the subject. Here we bring to you the 14th edition of Yuvaderma e-bulletin with a fresh entree of topics, not just limited to the subject but also related to current practices.

Our first article is all about the enticing journey of Dr Balakrishna Ankad in the field of dermoscopy and it's a must read for all the residents aspiring to do well in their careers. We have also taken a special interest in paediatric dermatology in this current edition as it has a case report, approach to the physiological neonatal conditions and spotters as they are often neglected during residency. Keeping up with the ever growing field, we have articles highlighting the need for training in procedural dermatology and soft skills.

Keeping the non-academic interests alive while pursuing their academic endeavours is essential for a fulfilling life. We at Yuvaderma encourage non-academic articles, poems and art to keep this fire alive in an otherwise mundane life. Our non-academic section has poems along with a few art pieces.

I would like to thank Dr. Ravi Rathod and Dr. P Jayanth for giving me this opportunity to head the editorial team that I was an integral part of for the last 4 years. I wholeheartedly thank my team for their efforts in bringing this edition to its conclusion. I thank Dr Sanjay Tejaswi for guiding me through this journey. I also thank all the residents who sent their articles and made this edition a success.

HAPPY READING FOLKS!

Regards,

Dr. Priyanka Karagaiah

Editor in chief

Yuvaderma E-Bulletin

TREASURY

DR RAVI RATHOD
PRESIDENT

PRESIDENT'S PREAMBLE

Greetings to all young budding Dermatologists

At the outset it gives me immense pleasure to write this message as the President of IADVL Karnataka

Yuvaderma E-bulletin is the brainchild of IADVL, Karnataka which was started in 2016, thanks to the then IADVL president late Dr. Umashankar.

This bi- yearly bulletin has become hugely popular mainly among young dermatologists not only in Karnataka, but all over India.

The hard work put by the editorial team is evident by quality articles published in the bulletin with great deal of professionalism.

I wish, 14th edition of this bulletin is going to be one of the most useful scientific publication and hope that it will benefit everyone academically and prompt more and more youngsters to contribute to the bulletin.

Wishing all the very best
Regards,
Dr Ravi Rathod
President, IADVL Karnataka

DR D P JAYANTH
HON. SECRETARY

FROM THE SECRETARY'S DESK

Dear residents,

Karnataka has 60 medical colleges and produces more than 300 dermatologists per year. Young residents are the backbone of Dermatology departments.

In this regard, Yuvaderma journal edited by Dr Priyanka Karagiah and team represents the issues and aspirations of the new generation. IADVL Karnataka Academy is conducting a Resimed online training program. This is in line with the Skill India mission of the Central government.

India has a huge demographic dividend (young population). If they are provided the right support and guidance, they can carry forward the legacy of Indian Dermatology. At present, India has 4 dermatologists per lakh population. Hence, the ability and skill set of each and every dermatologist adds value to society.

Residents must utilise every opportunity to learn and upgrade their skills. Hybrid learning is the norm. Yet clinical examination of patients guided by experienced faculty has no substitute. Hence, I request residents to attend Derma basics and Derma advance personally.

Editorial team has brought out a fine journal with good academic content. I wish them and all the young residents a bright future !

Long live IADVL!
With regards,
DR D P JAYANTH
Honorary Secretary
IADVL Karnataka 2021-2023.

DR SANJAY THEJASWI R
ADVISOR

ADVISOR SPEAKS

The Journey of a thousand miles begins with a single step- Confucius

Every resident, whose articles grace the pages of Yuvaderma journal, have travelled a Journey unique to their own lives. Each Journey is marked by hard work and diligence, but more so by grit and determination.

Pushing forward against the tides of time, all the contributors have cast their creations as their dice, ensconced in fact, shrouded in dedication and have crossed the fires of Review.

Present edition takes us back to the basics like explaining about the scourges of syphilis.

Rare case reports , latest review about Tomato flu has been discussed too. The most Awaited and Interesting interview with the guru of dermoscopy is indeed a motivating one. In order to bring the artist hidden in the budding dermatologists, art, photography, poetry has been introduced. A sincere appreciation to the Editor in chief Dr. Priyanka Karagiah who has bought out the journal with collective information.

I would like to render my humble thanks to the Honarary General Secretary Dr. D P Jayanth for the constant support and I also wish to thank our President Dr. Ravi M Rathod for the steady guidance.

The zest of life, lies is zeal and curiosity, leaving upto the readers to unreveal the new edition and to conquer the knowledge.

Dr. Sanjay Thejaswi R
Assistant Professor
Deputy Medical Superintendent
The Oxford Medical College and Research centre.
Bangalore.

Interview with the 'Guru of dermoscopy'

Dr Balachandra S Ankad

Dr Balachandra Ankad is an eminent dermatologist & an avid researcher of dermoscopy. He is a passionate academician, who is always enthusiastic about learning and teaching new things. He is one of the youngest HODs who has won many accolades and awards at a very young age. He has published many articles, chapters in books and a book on DERMOSCOPY. He has presented papers and given faculty lectures in many national and international conferences & workshops. He is not only a successful dermatologist but also a dance enthusiast.

It's my honor to interview my guide & it's fun to know about the dermoscopy expert. Let us know about his journey.

SN: Sir, when were you born and tell us something about your schooling?

Sir: I was born on 19th June, 1975 at Nidagundi village, Vijaypura district. I completed my schooling till 10th std in Kannada medium Govt. school at Nidagundi. I completed PUC in Basaveshwar Science College, Bagalkot.

SN: Sir, did you always think of becoming a doctor?

Sir: No, till my PUC, I wanted to be a district collector (DC) and solve the problems of my native. But then during PUC, one of my teachers said if we score 80% we can get into medical field, and my brothers were already into engineering. So I thought let's be different. With interest I joined MBBS at KIMS, Hubballi. Just see the destiny, now also I am a DC - Dermatologist to the Core.

SN: Sir, would you tell us something about your UG days?

Sir: I was excited and nervous as well during the initial days of my MBBS course. For the first 6 months I couldn't adjust, was quite afraid of studies, as I was from Kannada medium and was not well versed with English. After 6 months, I started studying, understanding the subjects along with enjoying life by hanging out with friends, playing cricket, watching movies & I feel those days were awesome.

SN: Sir, Dermatology was by chance or by choice?

Sir: It was by chance. I wanted to do MD in a medical college with a stipend. For my CET rank, I was allotted dermatology

in JJMMC, Davanagere where there was a stipend for PGs. This is how I entered the world of dermatology. Otherwise my subject of choice was Medicine. But after entering dermatology, I have never repented, in fact I am more than happy.

SN: Sir, How did you start your practice after completion of PG?

Sir: During PG days, I had decided to join medical college in Bagalkot (It was established in 2002). I completed my PG in 2005 & immediately joined as the Senior Resident at S.Nijalingappa Medical College, Bagalkot. In 2006 I became an Assistant Professor and simultaneously started my private practice. In 2016 I became Professor & HOD.

SN: Sir, what made you walk through the path of Dermoscopy?

Sir: Well, I attended 1st National Dermoscopy CME at Mumbai in Jan 2012, by which I was inspired. I started my work on dermoscopy. In April 2012, first dermoscope (DermLite 3) from New York was purchased. For the first 3 months I struggled to understand. By repeated efforts & reading many books I got through it. Finally we started to correlate the dermoscopic patterns with clinical and histopathological features. In 2013, we wrote (with my friend Dr Savitha LB) our 'FIRST' dermoscopy paper on pyogenic granuloma. Till date it is one of the highest cited articles. In 2015, I attended World Congress of Dermoscopy in Austria & presented a paper. People started recognizing me as a faculty in dermoscopy and they started calling me to present papers in various conferences. This is how my journey continued in the field of dermoscopy. There have been more than 110 presentations in conferences & workshops till now. Many workshops on dermoscopy were conducted. Recently, I completed 100 publications on dermoscopy. In 2016, I went to Germany to get FotoFinder dermoscope for my private clinic. I got trained for 2 days regarding its usage & operating system. In 2019, I got another DermLite Dermatoscope with DSLR camera; this is how my curiosity continued in dermoscopy to learn more and more new things. In 2018, We (with Dr Balkrishna Nikam And Dr Samipa Mukherjee) thought of writing a book on dermoscopy -histopathology correlation. In 2019, we started working on book. In 2021, we published a book on 'Dermoscopy- Histopathology Correlation' by Springer Publications. Meanwhile I became a convener, coordinator of SIG-Dermoscopy by IADVL. I could collaborate with internationally reputed dermoscopy stalwarts like Enzo Errichetti, Aimilios Lallas and many others. In 2022, we could (with Dr Yasmeen Bhat and Dr Kinjal Rambhia) write an IADVL Atlas of Dermoscopy in skin of color. We have a long road ahead to be explored and understand many new things in dermoscopy. Now I am working on another unique dermoscopy book (with Dr Enzo Errichetti, Dr Richard Usatine and Dr Aimilios Lallas) by Springer publications which will be launched in 2023.

SN: Sir, did you face any difficulty during this career journey?

Sir: Difficulties were there in managing the department, in paper presentation and writing. I could manage it well probably

because of love towards work. I believe in 'Work is Worship'. We need to have 'Bhakti Bhava' towards work, then the whole universe will fall in one line. But I could not give much time to my family and kids. Now I feel I am handling work and family both very well.

SN: Sir, why did you choose the teaching field?

Sir: Somehow I liked teaching. Interestingly, I always liked teaching and wanted to beithe same field, I belive that the ultimate profession is of the teachers.

SN: Sir, Do you have any dream to be fulfilled?

Sir: I am quite happy with whatever is being done in the field of dermoscopy. But, 'BEST IS YET TO COME'. There is an intense 'Itch' to publish many more books and articles, to spread & share the knowledge about dermoscopy among dermatologists. I, with my colleagues, need to frame dermoscopic criteria for many dermatological conditions in Skin of color, so that it becomes easy for everyone to understand.

SN: Sir, Can you tell me something about your awards and achievements?

Sir: Got selected as a Board member of International Dermoscopy Society (IDS-2018-2020), Convener (2018-2019) & Co-ordinator (2020-2021) of SIG Dermoscopy. I received a scholarship in World Congress Dermatology at Seoul in 2011. I had an opportunity to be Dr JN Shetty Oration Cuticon- Karnataka in 2020. I really feel rewarded whenever my resident publishes a paper or presents a paper in a conference.

SN: Sir, what makes you so dedicated towards work?

Sir: Passion towards work, inclination and love towards whatever I am doing. I feel happy doing that.

SN: Sir, how would you like to be with your residents, As a teacher or friend?

Sir: Ha ha haaa, I'm always a friend to my residents and would always like to be a friend. I feel love & affection always creates an arena that makes people at ease and comfortable to learn. Probably this creates a friendly atmosphere in the department.

SN: Sir, how do you keep your residents busy?

Sir: Honesty in teaching and learning. We need to make them observe things carefully and urge them to always write articles. This makes them read, keeps them updated and motivated towards work.

SN: Sir, how do you cope up with your stress?

Sir: No, even I feel tired of working. Most of the time, tiredness is a sign of lack of interest in the work. We need to be 'TOTAL' in whatever we do. When we work with

interest or love, there will be no stress at all.

SN: Sir, little about your family?

Sir: I am grateful to my parents for introducing me to this world. Hats-off to my wife Dr Roopa for the complete support in my journey of academic endeavors. She looks after the entire family. I owe her a lot. My elder son Tanush is in 6th class and my younger daughter Srijani is in 4th . I really enjoy playing cricket with Tanush and doing art and craft activities with Srijani. I am home by 7pm, from then onwards I am with my family. We enjoy our family time.

SN: Sir, opinion about dermatology 10 years ago, presently & 10 years later?

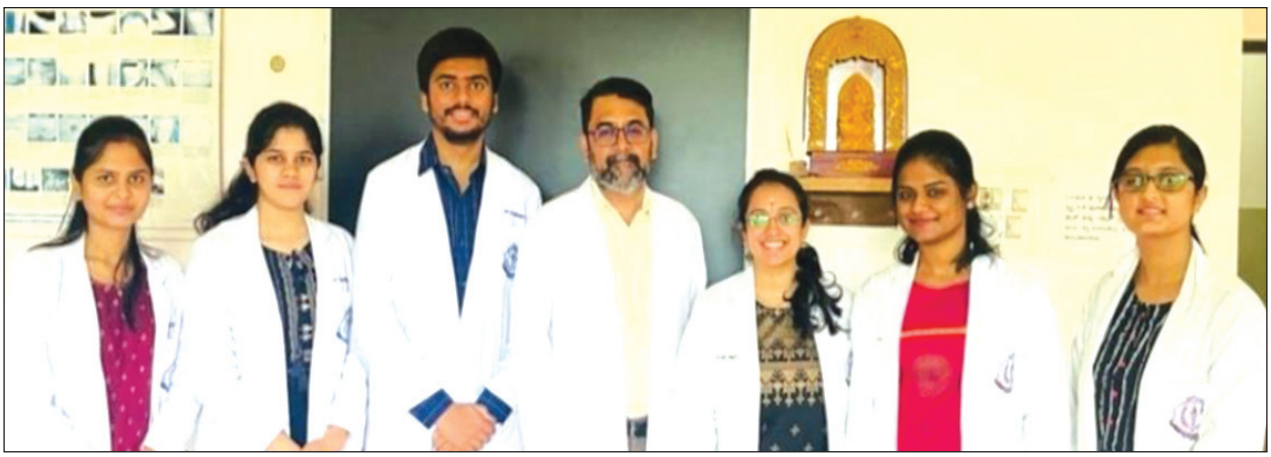
Sir: 10yrs back dermatology was a medical speciality without much reward. Presently, it has its imprints. We can offer good control over many inflammatory and infectious dermatoses. Thus, patient's quality of life has improved a lot now. Future is very bright with many molecules in the basket. Targeted topical therapy is the much speculated arena now. With many innovative technology and biological agents, dermatology will be leading from the front in the medical field.

SN: Sir, what do you think about research in our field?

Sir: Still many colleges lack research work & they should come up with many research projects. Compared to the past,the field of dermatology has a lot of ongoing research on topics such as biologics, aesthetics, dermoscopy and other imaging science. We, in the department, need to observe the diseases carefully, something would be noticed out of it. That becomes a stepping stone for new research and it could be published with proper scientific methodology. Every research work should be implied in clinical practice. It should help in the improvement of life of the patient being treated.

SN: Sir, what do you think about super-specialization after dermatology?

Sir: I think sub branches in dermatology are required. This makes us focus on one entity. Pediatric dermatology, dermatosurgery, dermatopathology, lasers, and cosmetology are very popular. Recently, dermoscopy was added to the list. But it should be remembered that we need to be a clinical dermatologist first.



This ensures a good foundation for the practice of dermatology.

SN: Sir, will teledermatology affect a dermatologist's practice?

Sir: Teledermatology is very much prevalent in the western world. It may affect the dermatology practice to a little extent. It is useful for remote places where it's difficult for people to travel to far places in search of a dermatologist.

SN: Sir, how to be a successful dermatologist?

Sir: Never work for success, work hard with passion & success will come to you. Keep walking; many mile stones would be crossed!

SN: Sir, expectations from your residents & any advice for the residents?

Sir: I think residents should exceed their teachers in academic achievements and in practice. This is the 'GURUDAKSHINE' in the real sense. Teacher should be known to the world by the student's name.

Regarding advice it's only Read, read, read. Always make some study hours, proper time table for reading, should refer to different books & articles and lastly, have passion towards whatever you do & keep enjoying every step. Whenever you face any difficulty just think of the path you walked so far & never give up. Always find positivity in every aspect, which will help you lead a beautiful life. It is like doing 'TAPASSU' (penance).

SN: Sir, last but not the least what's the secret behind your young & energetic look?

Sir: I am my favorite. I try to be myself, try not to bother about gossip or the outside world. I enjoy dancing, listening to music and meditating. Probably this makes me

feel relaxed and keeps me happy. Keep helping your fellow colleagues that gives a different feel altogether.

SN: Sir, let's have some Swift conversation.

Sir: Ok like?

SN: Bit about your likes or dislikes.

1) **Favorite food -** I'm poor at eating, not particular about food
2) **Favorite actor -** Dr Rajkumar
3) **Favorite actress -** Aarati (old actress)
4) **Favorite books -** Books by Beechi
5) **Favorite topic in dermatology -** Obviously dermoscopy.
6) **Favorite movie -** Yuvaratna, Rajkumara, Baahubali & many more. As I am crazy about watching movies, I have a home theater.
7) **Favorite place to hangout -** My Home
8) **Hobbies -** Dancing & watching movies.
9) **Like to work on holidays -** Nope not at all.
10) **Happiest moment of your life -** When I cleared my 1st year MBBS.

11) Strength - Passion for dermoscopy teaching.

12) Weakness - Have to learn many cosmetic procedures, quite poor in those.

13) Inspiration - Dr Arun C Inamdar from BLDE College, Vijaypura & Dr K Ravindra from JJMMC, Davangere.

14) How did you feel to be the HOD of the department? - As such nothing was normal as I was acting HOD from before so it didn't make much difference but then, I enjoyed being more than HOD because of my resident's company.

15) How it feels to be one among the popular dermatologists - Best is yet to come.

SN: Thank you so much sir for giving your precious time for this interview. It's bliss to have such a guiding light in our life & all the PG's feel lucky to have you as our Guru.

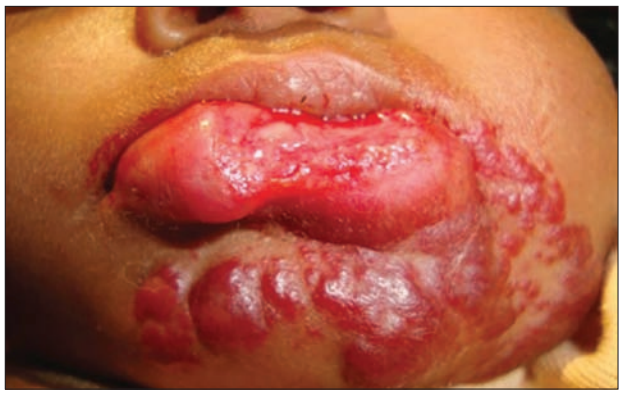
Sir: I extend my heartfelt thanks to Dr Priyanka, the editor of YUVADERMA. I feel honored for being selected for this column. I would like to remember and thank my departmental colleagues who are with me in difficult times and other colleagues who are sharing their experience and knowledge in bringing many scientific papers and books in dermoscopy. I thank

Dr Sushila for penning this interview. I thank all my residents (previous and present) for their constant love and affection towards me that makes me keep working in the department.

ದೇಶಕ್ಕೆ ಯೋಧ ನಾಡಿಗೆ ರೈತ
ಬಾಳಿಗೆ ಗುರುವೊಬ್ಬ ತಾನೇ!
ಅಕ್ಷರ ಕಲಿಸೋ ಅಜ್ಞಾನ ಅಳಿಸೋ
ಅವನೂನು ಅನ್ನದಾತಾನೇ!

Dr. Sushila Nagur
Final year PG
SNMC, Bagalkot

Hemangioma Spotter



1. Identify the lesion.

A: It appears to be a vascular anomaly, most likely a hemangioma involving the lower lip and the chin.

2. What is a hemangioma? What is a vascular malformation? How will you differentiate the two?

A: A hemangioma is a benign neoplasm of blood vessels characterised by early proliferation and spontaneous involution.

A vascular malformation is a developmental anomaly involving a particular vessel like the artery/vein/capillary/lymphatics. They do not involute spontaneously.

HEMANGIOMA	VASCULAR MALFORMATION
Benign neoplasm of vascular tissue	Developmental anomaly due to dysmorphogenesis of vessels
Not present at birth	Present at birth
Rapidly increases in size then spontaneously involutes	Increases with increase in age. Gradually progressive
Dynamic	Static
IHC- Glut1 positive	IHC- Glut 1 negative
HPE-Absent arterioles and nerves, endothelial cell proliferation	HPE-Thick walled arterioles/arteries, nerves visible
Eg: Infantile Hemangioma, Tufted Angioma, Pyogenic Granuloma	Eg:AV Malformation, Capillary Malformation-Nevus Simplex/Nevus Flammeus

3. How will you classify vascular anomalies?

A: Vascular anomalies are classified according to ISSVA Classification of 2014 with a 2018 update.

ISSVA stands for- International Society for the Study of Vascular Anomalies

ISSVA classification for vascular anomalies ©
(Approved at the 20th ISSVA Workshop, Melbourne, April 2014, last revision May 2018)

This classification is intended to evolve as our understanding of the biology and genetics of vascular malformations and tumors continues to grow

Overview table

Vascular anomalies				
Vascular tumors	**Vascular malformations**			
	Simple	**Combined °**	**of major named vessels**	**associated with other anomalies**
Benign Locally aggressive or borderline Malignant	Capillary malformations Lymphatic malformations Venous malformations Arteriovenous malformations* Arteriovenous fistula*	CVM, CLM LVM, CLVM CAVM* CLAVM* others	See details	See list

° defined as two or more vascular malformations found in one lesion
* high-flow lesions

4. List out some common benign vascular tumours.

A:

- Infantile hemangioma / Hemangioma of infancy
- Congenital hemangioma
  - o Rapidly involuting (RICH)
  - o Non-involuting (NICH)
  - o Partially involuting (PICH)
- Tufted angioma
- Spindle-cell hemangioma
- Epithelioid hemangioma
- Pyogenic granuloma (also known as lobular capillary hemangioma)
- Hobnail Hemangioma
- Papillary hemangioma

5. If the child presented to you (a)within a few weeks of birth, (b)at 7 years of age, which phase would it be in?

A: (a)Proliferative Phase (b)Regression Phase

6. List out the phases and clinical features of the different phases.

Clinical phase	Features
Prodromal phase	Premonitory lesions - circumscribed telangiectasia, anemic, or blue macule or blurred swelling
Initial phase	Loss of typical skin structures with increasing thickness and induration
Proliferation phase (6-8 weeks)	Bright red cutaneous infiltration with spreading of lesion. Exophytic or endophytic subcutaneous growth of thickness is seen. Firm and noncompressible
Maturation phase	Raised, bosselated crimson red lesions
	Surface complications such as ulceration seen in this phase
	Maximum size by 12-15 months
Regression phase (involution phase)	50% have normal skin at the site of lesion
	At times may leave fibrofatty residuum, telangiectasia, yellow hypoelastic patches, or may rarely scar
	Involution is usually complete by 5 years of age in 50% of children, 7 years in 70%, and most by 10-12 years

7. Can you name some syndromes associated with hemangiomas?

A:

PHACES: Posterior fossa malformations, Hemangiomas, Arterial anomalies, Cardiac abnormalities, eye abnormalities, sternal defects

PELVIS: Perineal hemangioma, External genital malformations, Lipomyelomeningocele, Vesicorenal abnormalities, Imperforate anus, Skin tags

SACRAL: Spinal dysraphism, Anogenital anomalies, Cutaneous anomalies, Renal and urologic anomalies, Associated with Hemangiomas of Lumbosacral origin

LUMBAR: Lower body hemangiomas and other cutaneous defects, Urogenital anomalies and ulceration, Myelopathy, Bony deformities, Anorectal malformations and Arterial Anomalies, Renal Anomalies

8. What are the common complications of hemangiomas?

A: Ulceration, haemorrhage, necrosis, heart failure.

Based on location, it can lead to impairment of vision, obstruction of airway, interference with feeding etc. It can be associated with syndromes as mentioned previously.

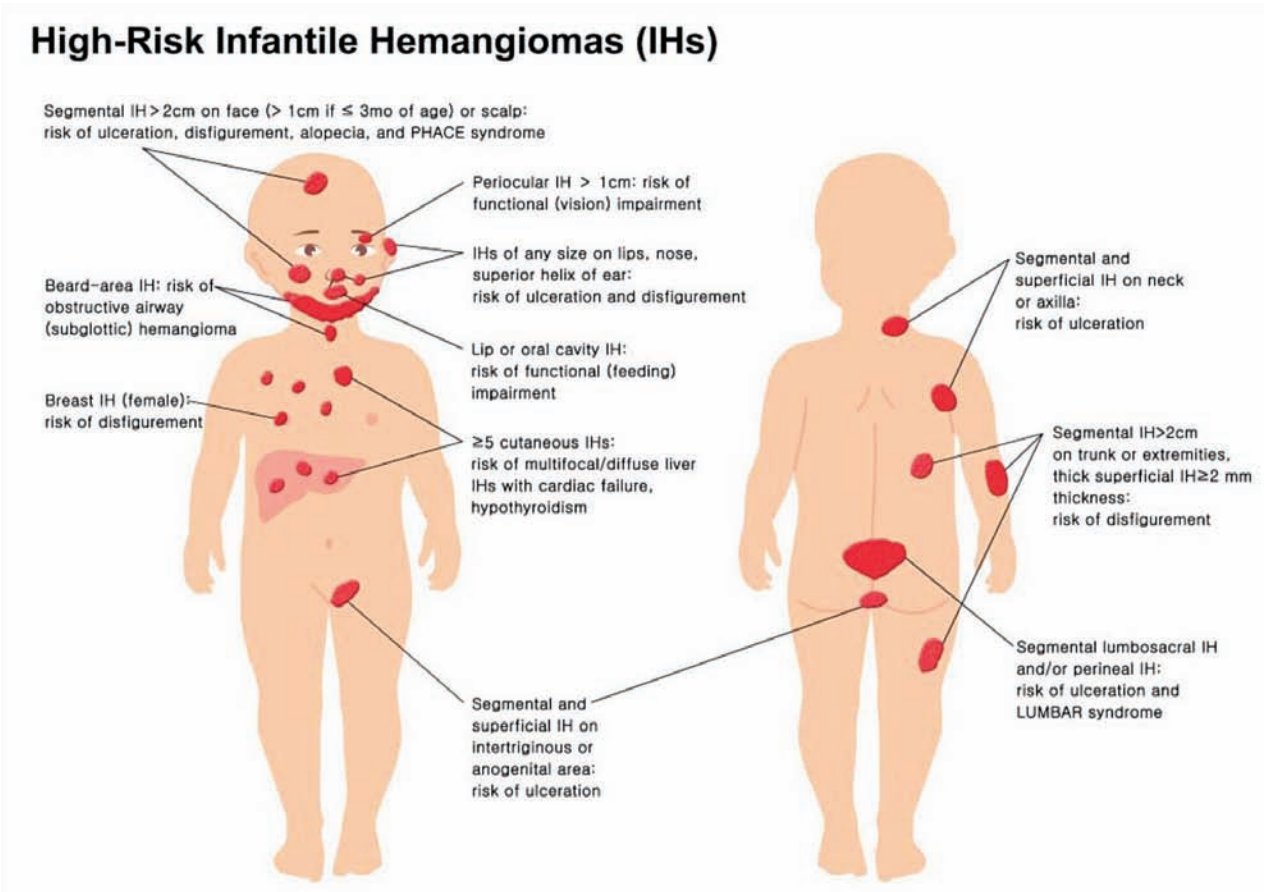
Local complications	Ulceration (common complication, seen in 5-13%), hemorrhage, infection, and necrosis
According to location	
Periorbital and retrobulbar	Visual axis obscured, astigmatism, amblyopia, and optic nerve compression
Auricular	External auditory canal obstruction, otitis externa, and hearing impairment
Nasal tip and large facial hemangiomas	Permanent scarring (e.g., Cyrano nose) and disfigurement
Mandibular/beard area distribution	Airway obstruction

Perioral	Feeding difficulties, ulceration, and disfigurement
Subglottic	Stridor and respiratory failure
Perineal	Ulceration
Multiple hemangiomas	Visceral involvement (liver and GIT)
Large hemangiomas	High-output cardiac failure
Associated anomalies	
PHACES syndrome	Posterior fossa malformations, hemangiomas (plaque such as segmental facial hemangiomas), arterial anomalies, cardiac defects, eye abnormalities, sternal clefts, and supraumbilical raphe
PELVIS syndrome	Perineal hemangiomas and external genitalia malformations
	Lipomyelomeningocele, vesicorenal abnormalities, imperforate anus, and skin tags
SACRAL syndrome	Spinal dysraphism, anogenital anomalies, cutaneous anomalies, renal and urological anomalies, associated angioma of lumbosacral localization

9. Which hemangiomas require further imaging?

A:

- Infants with more than 4 hemangiomas,
- Size >1cm in infants, >4cm in children above 3 years
- Segmental hemangiomas
- Complicated hemangiomas
- Doubtful diagnosis
- High Risk Subtypes:
  - Large facial
  - Nasal tip/ Ear involvement
  - Periocular/ Retrobulbar
  - Perioral
  - Segmental over lumbosacral spine
  - Anogenital area
  - Visceral involvement



10. What are the available treatment options for IH?

Medical modalities	Surgical modalities
Systemic	Surgical excision (elliptical intralesional resection)
Corticosteroids	Lasers (Pulsed dye, Nd: YAG)
Interferon alpha	Cryotherapy
Vincristine	Sclerotherapy (e.g., polidocanol)
Cyclophosphamide	Noncontact, low-frequency ultrasound therapy, and
Bleomycin	Embolization
Topical	Radiotherapy
Corticosteroids (topical creams/intralesional)	
Beta blockers (e.g., topical timolol)	
Becaplermin	
Imiquimod	

Nd: YAG: Neodymium-doped yttrium aluminum garnet

11. What is considered as the current drug of choice for high risk IH? What is the mechanism of action?

A: Beta-blockers- Topical Timolol 0.5% Eye Drops/Gel;
Oral propranolol at a dose of 2mg/kg/day.

Mechanism of Action:

- It causes vasoconstriction of supplying capillaries
- It inhibits angiogenesis by inhibiting pro-angiogenic factors like VEGF, MMP-2, MMP-9
- It also induces apoptosis of capillary endothelial cells

12. What are the precautions one must take while initiating oral propranolol?

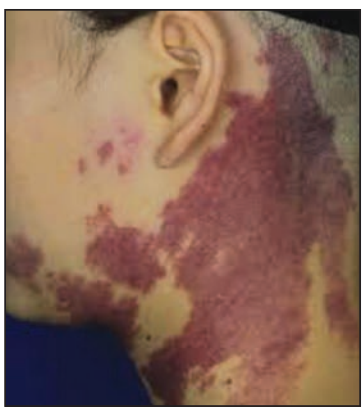
A:

- Exclude infants with evidence of bronchospasm / cardiac anomalies / suspected neurological involvement
- Conduct baseline investigations of blood glucose, blood pressure, echocardiogram and electrocardiogram
- 1st dosing should be administered in hospital settings with monitoring for 4-6 hours after the dose and for 1 hour after every increase in the dose.
- Ideally should be given just after feeding to prevent hypoglycemia. Frequent feeding should be encouraged.
- Watch out for early complications like sweating, tachycardia, hypotension. Late complications include lethargy, poor feeding, apnea, seizures and loss of consciousness.

13. What is Kasabach Merritt Phenomenon? Name 2 hemangiomas in which this phenomenon can be seen.

A: It is characterized by a coagulopathy with features including profound low platelets (thrombocytopenia), low fibrinogen (hypofibrinogenemia) and low level of red blood cells (anemia).
It is seen in Kaposiform Hemangioendothelioma and Tufted Angioma.

14. What is Nevus Flammeus? What are some syndromes associated with it?



A: Nevus Flammeus is also known as Port wine stain. It is a capillary malformation. Common syndromes associated with it are:

- Sturge-Weber Syndrome
- Klippel-Trenauney Syndrome
- Bannayan-Riley-Ruvalcaba Syndrome
- Proteus Syndrome
- Parkes-Weber Syndrome
- Rubinstein-Taybi Syndrome

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Dr. Punya Suvarna
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What's in a name ? Hand, foot & mouth disease (HFMD) or Tomato flu ?- A brief review

1. What is HFMD ?

Hand, foot and mouth disease (HFMD) is a viral exanthem, caused by the Enterovirus genus of the family - Picornaviridae. They are single stranded positive RNA viruses. The most common serotypes identified are Coxsackievirus(CV) A6, A10, A16 and Enterovirus A71 (EVA-71). The first case of HFMD was first detected in New Zealand and Canada in 1957, and thereafter, the Asia-Pacific region is by far the most affected part of the world, with a maximum number of outbreaks. HFMD caused by EVA-71 accounts for the largest number of cases with an unfavorable clinical outcome, including CNS complications and cardiovascular collapse. More than 80% of the severe cases and more than 90% of HFMD related deaths are caused by EV71.

2. Pathogenesis of HFMD

The Enterovirus initially is said to replicate in the oropharyngeal cavity and small bowel, giving rise to mild viremia. They later disseminate and replicate in other organs and mucus membranes, causing systemic symptoms. Tonsils are an important site for viral replication of the Enterovirus, which is why throat swabs provide a high diagnostic yield of the viral load. Molecular research on EV71 showed that it mainly causes neurological effects by inducing inflammation in the CNS, but not in other organs. The inflammation was mainly found in the hypothalamus, brain stem, spinal cord and cerebellum, indicating a retrograde spread through peripheral motor nerves.

3. How does it spread?

Even though HFMD mainly affects children in the age group of 2-8 years, cases among adults have been one the rise recently. It spreads through intra-familial, child-care and household exposures. This necessitates certain interventions like hand washing, avoiding close contact with infected individuals, disinfection of frequently touched surfaces, to reduce HFMD transmission.

4. Symptoms and signs of HFMD

HFMD is a benign, self limiting disease, with most cases presenting with a mild exanthem and fever. The incubation period is 3-5 days and the lesions last for around 7-10 days. The characteristic lesion is a vesicular rash on the hands, feet, mouth and buttocks, associated with low grade fever, sore throat and malaise. Other sites like knees, elbows, dorsal surfaces of hands and feet can be involved. The typical skin lesion are typically ovoid vesicles with the long axis parallel to skin lines, with an erythematous halo, that can rupture into painless shallow ulcers, which does not scar. Atypical skin lesions include hemorrhagic or purpuric lesions, bullae, genital involvement, desquamation of palms and soles. Oral ulcerations, which are typically painful, are found on the soft palate, tongue and buccal mucosa.

The characteristic nail change which occurs in HFMD is onychomadesis (3-8 weeks post infection). Beau lines and nail matrix arrest have also been reported.

Neurological and cardiopulmonary manifestations (Severe HFMD) :

Although extremely rare, signs of fatal disease like tachycardia, tachypnea, hypotension have been identified in children. EV 71 is associated with a wide spectrum of CNS syndromes including, aseptic meningitis, poliomyelitis - like paralysis, opsoclonus-myoclonus syndrome, brain stem encephalitis and acute flaccid paralysis.

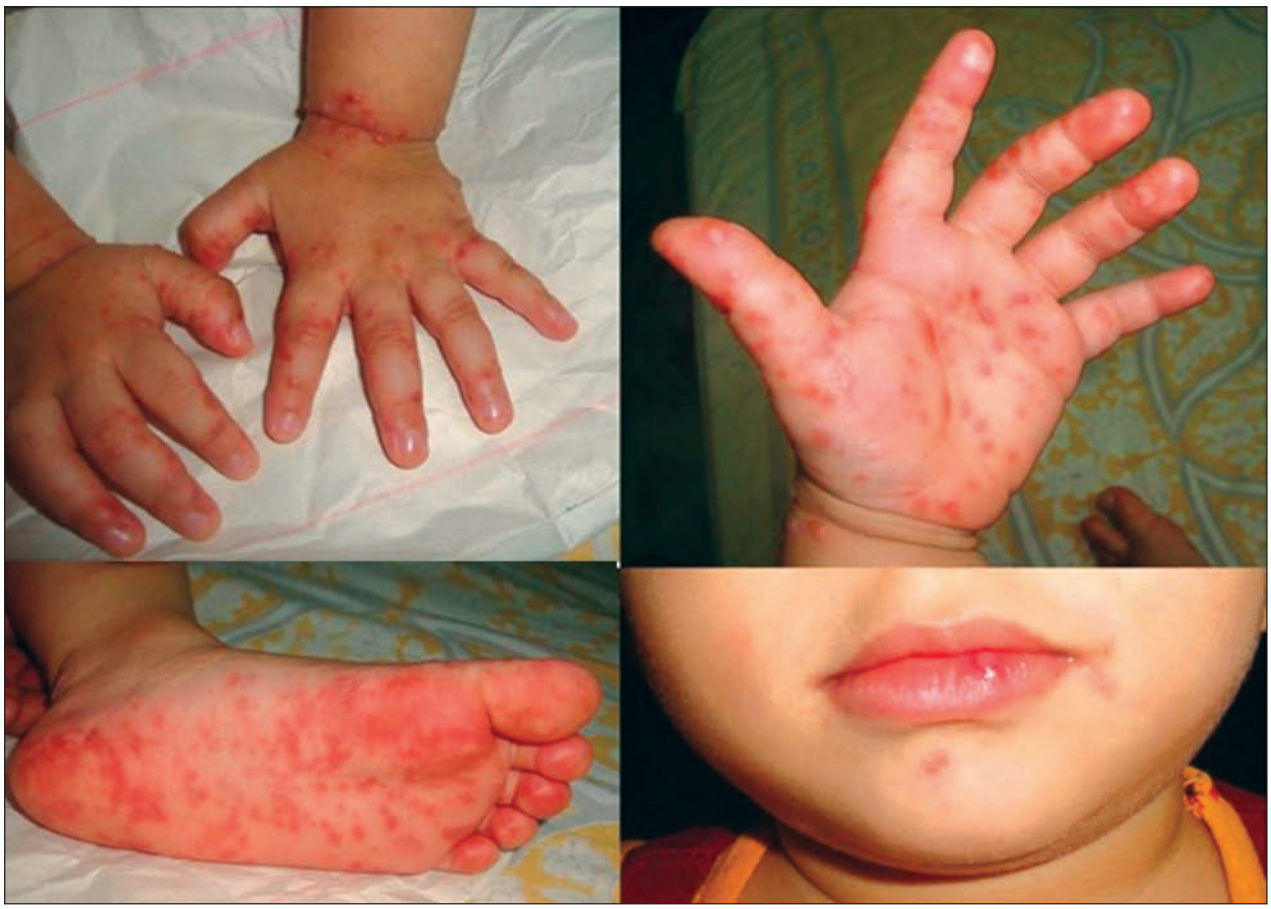

Fig1: Vesicular rash on the hands, feet and mouth

Table 1 : DIfferential diagnosis of HFMD.

	HFMD	HERPANGINA	VARICELLA	APHTHOUS ULCERS
CAUSE	CVA16, EVA-71	Coxsackie Virus A	Varicella Zoster Virus	Unclear
AGE GROUP	< 10 Years	3-10 Years	Preadolescent Children	Children And Adolescents
TYPICAL FEATURES	Ovoid Ulcers Over Hands, Feet, Oral Cavity	Ulcers Over Pharynx, Tonsils And Soft Palate	Vesicles With Central Umbilication (Dewdrops On Petal), Centrifugal Spread	Painful, Shallow Ulcers, Surrounded By Erythema

5. Diagnosis and Management of HFMD

Although HFMD is a clinical diagnosis , the virus can be detected from stool samples, rectal swabs and throat swabs. It can be detected in the stool for upto 6 weeks after infection and from the oropharynx for upto 4 weeks post infection. There are no specific antiviral drugs for the management of HFMD. Mild cases need symptomatic treatment in the form of rest, adequate hydration and NSAIDs for pain and fever.

Criteria for admission include (CDC guidelines)

1. Inability to tolerate oral feeds
2. Persistent hyperpyrexia (>38 degree Celsius) for >48 hours
3. Suspected CNS involvement- increased lethargy, drowsiness, change in sensorium, seizures.
4. Suspected CVS involvement- low pulse, low blood pressure, syncope.

Does Acyclovir help ? If yes, how so ?

Multiple studies reported a significant decrease in symptoms like fever and skin lesions, within 24 hours of starting therapy, when acyclovir was given within 1-2 days of onset of rash.

However, HFMD causing viruses like coxsackievirus, do not possess thymidine kinase, which is the principal enzyme upon which acyclovir acts and inactivates. The mechanism of action of acyclovir in HFMD is said to be an enhancement of the antiviral effect of the patient's own interferon !

In many countries with outbreaks of severe HFMD, IVIG has been used on a presumptive basis. When administered early, it was shown to halt CNS disease progression and decrease incidence of pulmonary oedema.

6. Vaccines for HFMD

Multiple vaccines are in the pipeline for protection against HFMD. Some vaccines currently in phase 3 clinical trials include formalin- inactivated EV 71 vision, inactivated CV-A16 virus, bivalent vaccine including both EV71 and CV-A16, and subunit vaccines using EV71 capsid proteins. Although the availability of vaccines against HFMD is important for lowering the disease burden, their long- term effectiveness, cross protection and efficacy needs to be extensively studied.

TOMATO FLU ?

Recent media reports, mainly from Kerala, have thrown light on an entity called - tomato flu in young children, described as a febrile rash with round, red skin lesions. A majority of the isolated samples taken from the children with the 'Kerala tomato flu' , demonstrated CVA 16, making it a variant of HFMD.

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AN APPROACH TO ACNE SCARS

Acne scarring is an untoward and permanent consequence of acne vulgaris, more often seen with inflammatory lesions. It is a highly prevalent complication having significant impact on quality of life of the patient and is a therapeutic challenge for the treating dermatologist.

Acne scars can be atrophic or hypertrophic; atrophic being the most common. The reason for atrophic scar formation with acne is loss of collagen tissue continuity, induced by injury, hormonal changes, or destructive inflammation (Propionibacterium acnes) in deep skin layers. In the areas with defective collagen fibers, the skin sinks; thus, atrophic scars are formed. Furthermore, due to the loss of integrity of collagen, sagging of skin is also noticeable.

Atrophic acne scars are divided into 3 main types:

1. Ice pick scars
2. Rolling scars
3. Boxcar scars

Scars are also be arbitrarily divided as fresh and old depending on the duration, as in:

- Fresh – less than 1 year old. These are considered to be more responsive to therapy.
- Old – more than 1 year old.

The scar becomes fully formed after 12 months and so the first 7–9 months are regarded as the most favourable period for treatment.

TOPICAL	INVASIVE	ENERGY BASED	SURGICAL	TISSUE AUGMENTATION
• Enzymotherapy • Hydration • Cryotherapy • Chemical peel	• Microdermabrasion • Rotatory dermabrasion • Dermaroller	• Laser correction • MNRF	• Subcision • Punch excision • Punch flotation	• Autologous grafts • Fillers

ENZYMOTHERAPY:

A modern method which involves injecting recombinant enzymes of non-animal origin, into the skin and subcutaneous tissue. These are predominantly proteolytic enzymes like collagenase, lipases, lyase and hyaluronidase.

Collagenases break down non-functional pathological collagen leading to formation of functional collagen, firming of sagging skin and reduction of scars.

Lipases degrade triglycerides from adipocyte.

Hyaluronidases and lyases depolymerizes polysaccharides that are causing water retention.

These enzymes alone or in combinations are injected into the concerned areas. The number of sittings varies between 2-5 done at intervals of 2-3 weeks.

HYDRATION / COLLAGEN INDUCTION THERAPY:

For hydration by collagen biomatrix applications, the treatment course is 3 months. Collagen biomatrix is a potent moisturizer and it is to be applied on fresh scars during the scarring period. Applications are given under occlusion for 40–120 min daily for 1–3 months. Mechanism of action is as follows:

- Maximal hydration of the scar formation zone;
- Reduction of transepidermal water loss;
- Acceleration of cell migration;
- Increase of fibroblast proliferation and synthetic activity.

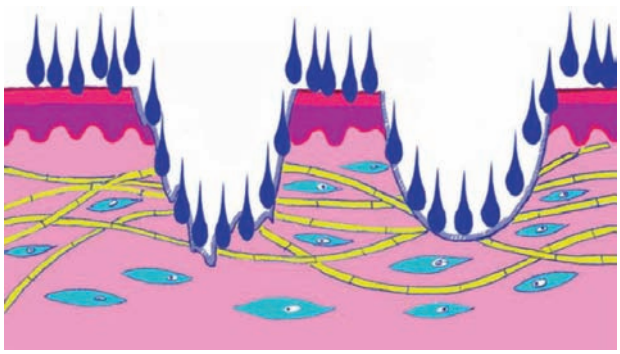
CRYOTHERAPY:

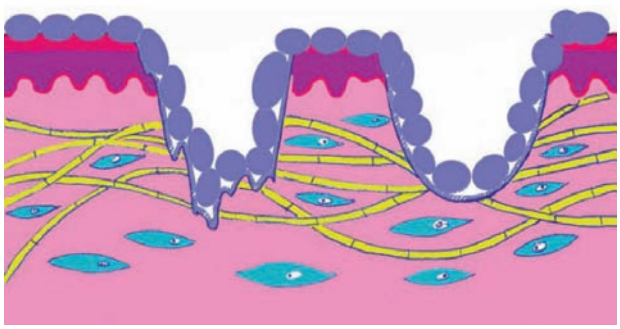
Cryotherapy in atrophic scar treatment is applied in the form of grinding and pinpoint cryodestruction by cryosurgical closed probes to even out the overhanging scar edges. It is performed in short 7s to 10s cycles. Cryotherapy is usually combined with microdermabrasion.

CHEMICAL PEEL:

Medium and deep peels are an effective method of fresh scar treatment. One to three sessions commonly suffice for fresh scar treatment at 1.5- 2-month intervals.

Fresh 30–50 % trichloracetic acid (TCA) CROSS is the most commonly done procedure for acne scars, specially for ice pick scars. Thorough and precise therapy requires that the acid shall be applied in two crisscross directions. The emergence of “frost” is stipulated by protein coagulation on the papillary dermis level. This coagulated protein coating prevents the deeper layers of dermis from acid penetration.



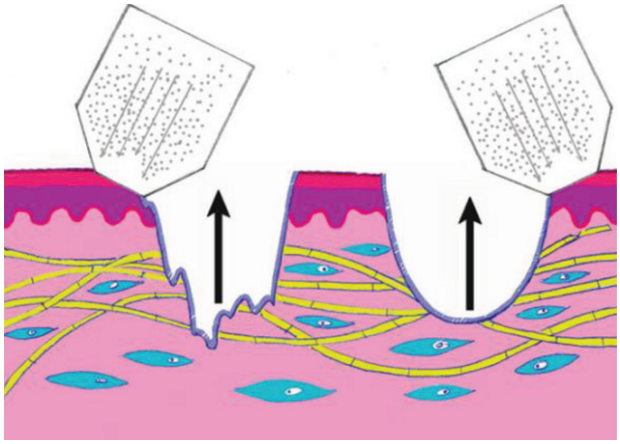


A common procedure for the treatment of atrophic scars is dermabrasion with aluminum oxide microcrystals. Advantages of dermabrasion are following:

- It evens and improves the scar surface
- It clears away the epidermal skin barrier of the scar area which enables topical products to penetrate freely into the skin.
- The scar bottom is elevated to the surrounding skin level by the vacuum, thus producing counteraction to myofibroblasts.

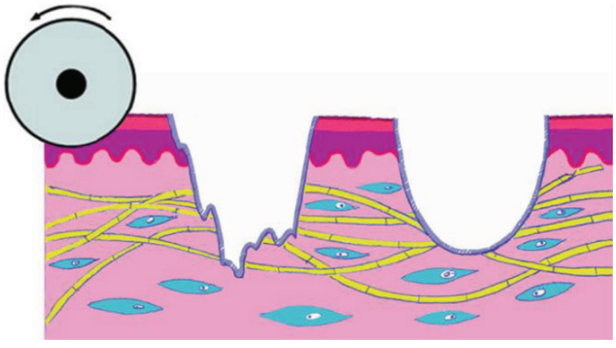
The procedure is performed multiple times (three to five times) at 3- 4-week intervals.

Along with scar maturation, connective tissues are formed, with the scar becoming less flexible and that's why performing early repeated microdermabrasion is more valuable. It is preferable to avoid MDA in IV–VI Fitzpatrick skin due to the risk of secondary hyperpigmentation.



ROTATIONAL DERMABRASION :

Rotational dermabrasion with nylon brushes and diamond cutters is another way of abrading the papillary dermis until there is pinpoint bleeding ("blooddew"). Unfortunately, nylon brushes and diamond cutters remove the normal skin surrounding the scar, lowering it to the level of the scar bottom. Hence this methos has lost its popularity.

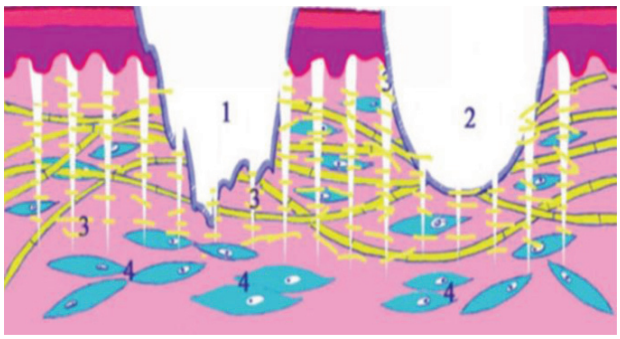


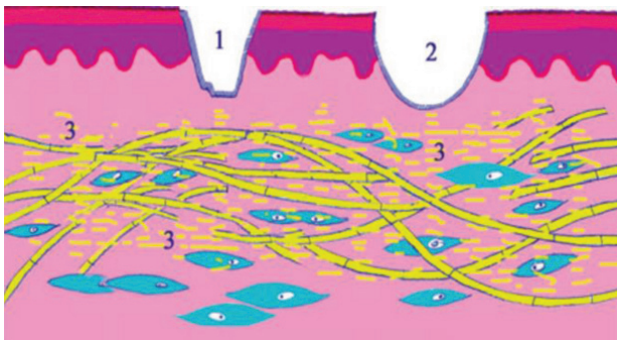
DERMAROLLER:

A dermaroller (mesoroller, mesoroll, top roller, skin roller) is a device with an abundance of small surgical needles that are 0.08–0.22 mm in diameter and 0.2–3.0 mm in length.

Mechanism of Action

When applying (rolling) the dermaroller, thousands of microscopic channels of diameter 0.08–0.22 mm and depth ranging from 0.2 to 2.5 mm are formed on the skin. Such channels induce a chain of healing processes leading to abundant fibroblast proliferation and collagen synthesis in the puncture areas. These fibroblasts "patch" microscopic channels, and a powerful supporting block (patch) is formed from its own collagen islets. Collagen islets gravitate to each other, gradually nearing the scar edges.





LASER CORRECTION:

All laser systems (ablative, nonablative) are applied in old scar correction with a varying success rate. The majority induce collagen tissue recombination and stimulate new collagen synthesis in the skin.

CO2 Laser (10,600 nm)

CO2 lasers when exposed to high energy, induce epidermis vaporization and partial collagen coagulation in dermis. Collagen fibrillae dehydrate and decrease finally resulting in tightening of the skin. The tightening effect facilitates further induction of collagen synthesis.

CO2 lasers can be either pulsed or fractional; the difference is that the pulsed laser vaporizes all the surface exposed to the laser beam, whereas a fractional laser creates microchannels in the skin that are interspersed with intact skin. Fibroblasts are activated at the bottom of these channels, synthesizing collagen. Therefore, fractional lasers are less traumatic and more efficient in atrophic scar correction.

Short-Pulse Erbium: Yttrium-Aluminum-Garnet Laser (2,940 nm)

Er:YAG laser vaporizes water from the reticular dermis and consolidates collagen. In this freed space the fibroblasts start synthesizing collagen. Unlike CO2 lasers, YAG lasers are characterized with significantly low thermal effect. This is the so-called cold ablation with minimal thermal damage, enabling minimization of the risk of new scar formation and pain.

Pulsed Dye Laser

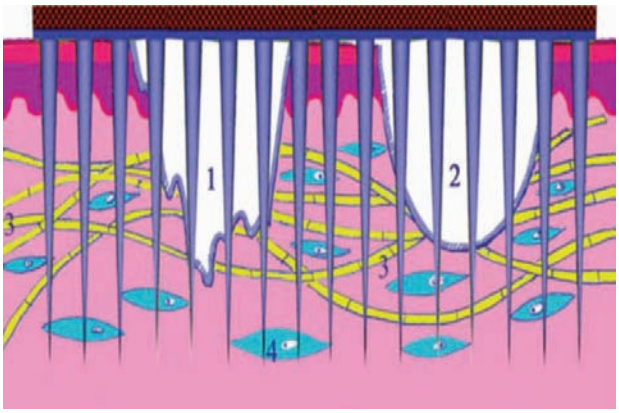
The PDLs at 585 or 595 nm wavelength are used in erythematic atrophic scar correction. The mechanism of scar vascular component removal lies in the RBC high-energy

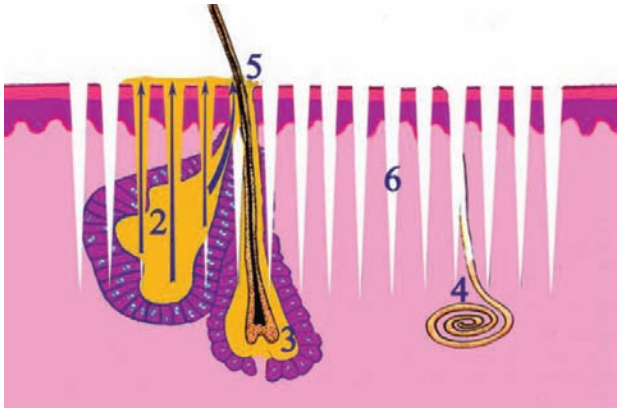
exposure in the lumen, which is the red scar substrate. A PDL is better applied as a final correction stage after resurfacing and/or cryodestruction because all machine-based relief correction methods facilitate the development of a venous capillary network in the scar.

MICRONEEDLING RADIOFREQUENCY ABLATION:

Microneedling radiofrequency device consists of a disposable tip with insulated needles that delivers bursts of RF energy. The depth can be adjusted from a minimum of 0.5 mm to a maximum of 3.5 mm. This tip creates an abundance of microscopic canals (RF thermal zones) allowing electrocoagulation of the dermis. The insulated needles ensure delivery of RF waves only at the tip

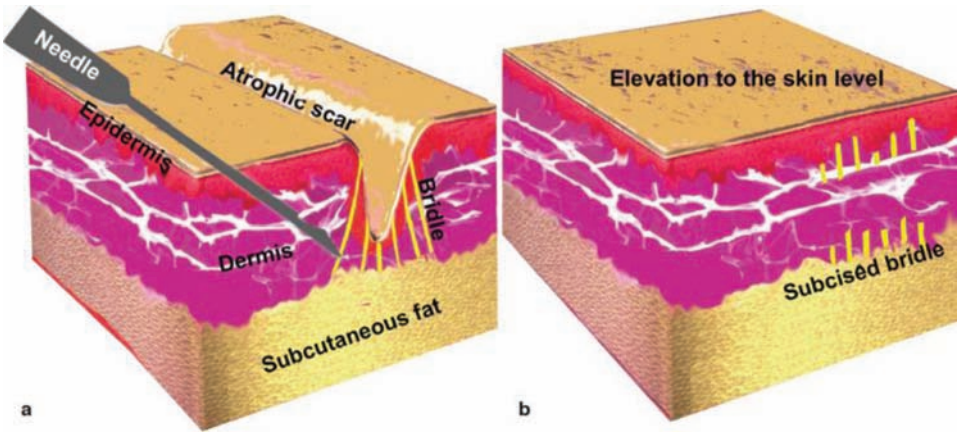
of the needle and hence damage occurs only in the dermis sparing the epidermis. Dermal damage induces neocollagenogenesis by stimulating the release of growth factors. An added advantage of this technology is the relative sparing of epidermis and adnexa which contribute to rapid healing and minimal downtime.





SUBCISION:

Scar subcision releases the bottom of the scar tissue and draws it to the surface. Subcision is performed by Nokor needle, preferably 18G. The edges are fixed with thumb and index finger and the needle is entered 3–4 mm away from the targeted scar. Tunnels are made by the needle and later it is moved from side to side, releasing the tied scar bottom from the connective bridle. When cutting connective bridle, a characteristic clicking is heard, which indicates that the procedure is being properly conducted. A recess filled with blood is formed in the course of subcision. Fillers such as Restylane or Juvederm may be injected into the recess, if required. It is more convenient to operate the needle with 1–2 ml cold saline solution as this helps in debriding the wound and also facilitates needle tunneling.



PUNCH EXCISION:

Punch excision (resection by punch, a circular knife) is applied in correction of deep ice-pick and boxcar atrophic scars. The size of the punch must be slightly bigger than the scar. Under local anesthesia with 2 % lidocaine solution, the punch is entered into the skin with circular movements

1.0–2.0 mm vertical to the skin surface. The resected skin cylinder is elevated by mouse-tooth forceps and cut off by scissors. The edges of the deficiency can be sutured by one stitch, glued by tissue adhesive or stripped in parallel to skin tension lines.

FILLERS:

Fillers are substances injected into an atrophic scar to align its relief and are divided into two groups: biological and alloplastic.

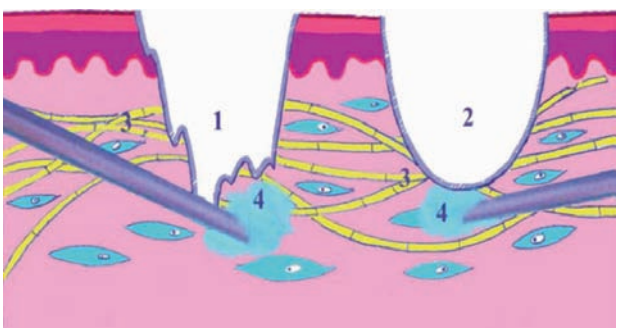
Biological implants - bovine collagen, autocollagen, allogenic collagen, autologic fibroblasts, and fat.

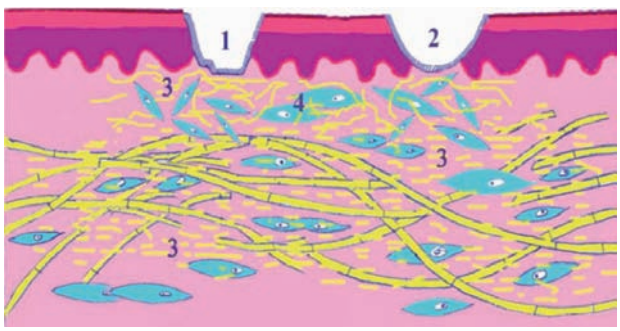
Alloplastic group - silicone, micronized alloderma, polymethyl methacrylate, and polytetra fluoroethylene.

Filler injections are performed as a final stage of atrophic scar correction. Peeling, dermabrasion, and electrophoresis are not to be performed after filler injections as these procedures may boost destruction of biodegradable filler and induce their complete resorption quickly.

TRANSPLANTATION OF FIBROBLASTS:

Fibroblast cultivation is carried out in special laboratories. A suspension is prepared from mature fibroblasts and the cultivated fibroblast suspension is injected directly under the scar intradermally. The needle must be directed maximally parallel to the skin surface. After the cell injection and a short adaptation period, fibroblast proliferation is initiated. When the fresh cells become synthetically activated, they start synthesizing collagen. The skin becomes elastic, and scars start leveling out.





To conclude, it is advisable to combine one or more methods to achieve maximal response.

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UNLEARNING

With hopes afresh, excited to fly on my own, I started my career after post graduation as a consultant in an aesthetic chain of clinics. I was always confident of what I could achieve, yet, I was so scared. I was scared not because I doubted my skills, I was scared because the aura of a clinic in private practice felt different. Trust me when I say, it's nothing like the protected environment during your PG days where you are handheld and taken through the nuances of the subject, where the liability of every decision you take and every mistake you make is borne by your professors. Here, you are on your own and solely responsible for your patients well and ill being.

In the very first month I met a plethora of characters in the form of patients. Everyone, as unique as a snowflake (pun intended). You meet different kinds of people here- the know it all, real time encyclopedias, patients of doctor google, reddit gangsta, infleunz(cer)a stricken, mixopathy enthusiasts, home remedy aunties (sexism not intended), filter fam - I can just go on. The funniest part about this is treating them scared the wits (****) out of me! More than my diagnostic/therapeutic skills, the need of the hour was people skills or soft skills. These skills are not something that you learn even in an Ivy league equivalent institution, unless you are into HR. These are skills that can only be mastered by observation, reciprocation and practice.

You may have had a 2 hour orientation class in the very beginning of your UG and PG days about communication, empathy and other 'stuff' that you conveniently slept through or even better, bunk'd. The importance of which is understated. What makes you sought after by the patients?

Your knowledge. Ofcourse. But most of us are equally knowledgeable and equipped to deal with any curveball of a case thrown upon us. We have a network of eminent colleagues to fall back to, when in doubt. What gives you an edge over the other competitors? (Now that we are competing with dentists, plastic surgeons, spas and everyone with an instagram degree in skin care). It's your "SOFT SKILL". Skill to empathize, communicate, understand what the patient wants, identify what the patient needs, differentiate between their wants and needs, communicate the same to them, convince them about the right treatment and most importantly setting the right expectations(Uffffffff). So in a way we are all mini psychologists by the end of the day.

The most important aspect of soft skill development is deciphering the patient's personality. There are 4 types of broadly categorized personalities.

1. Type A - The director
2. Type B- The socialiser
3. Type C- The thinker
4. Type D- The supporter

Why is it important to know these categories? Because understanding this helps you tailor your communication and save you time and trouble dealing with unhappy patients at the end of the day.

Type of personality	Qualities	What turns them off	What they expect out of treatment
Type A - The director	• Goal-oriented • Good under stress • Works well independently • Direct management style • Good delegation skills and administrative skills • Workaholic	• Long explanations or descriptions • Explaining things in emotional terms or more than once to the same person • Looking vulnerable • Being manipulated • Long waiting period	• Straightforward explanations • Quick consultations • Best and cutting edge technology • Instant results • Value for money
Type B- The socialiser	• Relationship-oriented • Outgoing • Enthusiastic • May try to do too much at once • Easily bored • Easily liked by most people	• Public humiliation • Being unappreciated • Appearing uninvolved • Nonsocial types • Appearing unattractive • Appearing unsuccessful	• Comforting words • Constant reassurance • Best aesthetic solutions • The latest trends • Look their best
Type C- The thinker	• Detail-oriented • Logical • Prepared • Likes to do things their own way • Worry about progress • Quality control	• Irrational acts • Indecision • Loss of control • Being subject to control or supervision by people they don't trust or respect • Distractions or distracting people	• Complete explanation • Evidence based treatment • Approved drugs/ methods only • Personal attention • Progress with each visit • Explain causes for failure of treatment

Type D- The supporter	• Shy, low-key and observant • Sincere • Consistent • Calm and stable • Fair and equitable • Avoiding confrontation • Resistant to change	• Risks • Pushy people • Change (especially frequent change) • Instability • Aggressive behavior • Disruption in routine • Surprises	• Comfort • Constant hand holding throughout the treatment • No frequent changes • Open to experimental treatment

The above table is an oversimplification of the more detailed Myer Briggs classification of the different types of personalities. People may have a combination of personalities but tend to have one dominant type.

The Type A/ The directors are the ones that come to you for quick solutions and in want of cutting edge technology. They don't like to wait as they are always busy and are also, very impatient. On the brighter side these are the ones looking for the most advanced treatments available and quite satisfied with visible improvement. As hard as it is to deal with them, it is also easy for them to be happy with quality treatment. All you need is to have a straightforward approach and a clear communication about the pros and cons of the treatment and let them decide what they need. Ultimately, they have the extreme need to be in control of their treatment/situation.

The Type B/ The Socialiser love to talk about themselves. Though they appear self-centered at the prospect, their motivation is to be liked by everyone and to forge strong relationships wherever they go. A good sense of humor and friendly conversation is a starter to win their trust. These patients are driven by trends and the onus is on you to break their bubble gently and offer an effective and safe treatment. But when treated well, they can make some of the most loyal customers and are most likely to be your organic marketing personnel.

Document every single detail, not just about the disease, but also the previous treatments, refusal to take a certain drug/ treatment, consent before the procedure, any side effects of the drug/procedures. Building this practice very early goes a long way in your career and prevents unwanted legal litigations.

The Type C / The Thinker are the most difficult patients you might come across. They are very outcome-driven and will be sticklers for following protocol. These are the kind of patients who research thoroughly about the disease and ask if the treatment is FDA approved. It's hard to bluff them and it's best to always go for the approved treatments and if available, follow the current guidelines for treatment. A thorough documentation can be a life saver if things go down south and don't be surprised if they ever sue you for negligence.

The Type D/ The Supporters are the easiest of patients and they mostly rely on your knowledge for best treatments and offer full cooperation.

They like to keep their routine constant and do not appreciate frequent change in treatment. They are the most loyal customers and do not like to change doctors often.

No matter what type of personality your patient belongs to, the key to a good relationship building is your honesty and knowledge.

Some of the practices to incorporate early in your practice are -

1. A warm greeting to begin your conversation with, addressing the patient by their name adds a touch of personal interest and can gain their trust.
2. A brief history about their work and place of occupation can give a fair idea about their lifestyle, and of late most conditions are lifestyle related.
3. Patient listening to their problems gives away the diagnosis in most cases and also helpful in meeting the emotional needs of the patient
4. By this time, if you have even developed a fair sense of what type of personality the patient belongs to, your job of explaining to them becomes very easy.
5. Communicating what abouts of the disease should be well thought of and made in a way that would suit their emotional demands.

For example, when a patient walks to you with generalized psoriasis and he/she is more of a director's personality, the best way to handle them would be to give them a very brief and crisp explanation about the disease and all the available treatments. They would probably be keen on trying apremilast/ even ready to take secukinumab as a last resort. They would want something advanced and less time consuming.

Whereas, a supporter would probably need a slow approach where you break the news more gently and in a less daunting manner. They would assume you know what's best for them and the burden of selecting the right treatment is entirely on you. Even if it takes longer they would be completely ready to trust you and comply as long as it's even remotely working.

Let's take another example of androgenetic alopecia. If you were to meet a socialiser he would probably come asking you for QR678 or scalp threads, as they are trending and frequently appear on ads. Not everyone is equipped with either of these methods, while they may still be performing other effective treatment options. You can't simply dismiss their whims, instead they need to be coaxed to undergo PRP or microneedling or even taking minoxidil. A friendly conversation with an occasional complement would do the tough job of gaining their trust.

But, instead if you were to meet a thinker he would come very well equipped with all the research and you should not be surprised if they ask about comparative studies and FDA approvals. In these cases knowledge is your superpower, all you have to do is lay out facts as it is and let them be an active part of decision making.

6. Never force anyone to take any form of treatment, especially long term systemic agents and procedural treatments.
7. Never give up on your chronic patients, take help from seniors/ colleagues but try to give them the best treatment possible.
8. Always try to be considerate about the patient's financial conditions and especially if they have made it clear that they cannot afford a certain medication. Try and suggest a cheaper alternative.
9. Document every single detail, not just

about the disease, but also the previous treatments, refusal to take a certain drug/ treatment, consent before the procedure, any side effects of the drug/procedures. Building this practice very early goes a long way in your career and prevents unwanted legal litigations.

10. Always take documentary pictures.
11. Always be courteous to your clinic staff. Never reprimand them in front of your patients. It not only sets a wrong narrative about your temper but also makes them doubt the competency of your staff.
12. Most importantly, PRACTICE ETHICALLY.

Sooner or later you will enter the world of independent practice and you will have to unlearn some of the ways of your post graduate world. You will have to learn to survive in the world which is fast changing. The demographics are changing and so are the concerns of the patients. Now they are more aware of the conditions and the treatments.

Our faculty is considered nothing less of a service provider. Ultimately you are dealing with a multiverse of patients day in and day out. The same old rambling of a treatment like a radio jockey will not help. You will also have to work on your personality, communication and soft skills. The first prerequisite to character building is unlearning the things you have known and done so far. Keeping an open mind and constantly adapting to the ever changing world is the key to staying relevant, not just in your career but also in life.

P.S: This is the author's personal opinion, and not to be considered a manual for private practice. :p

Dr. Priyanka Karagaiah
Consultant Dermatologist
Oliva skin and hair clinics &
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SYPHILIS - The Scourge Of Renaissance

- **Stokes definition of syphilis (10 points)**

Syphilis is an

1. infectious disease;
2. due to Treponema pallidum;
3. of great chronicity;
4. systemic from outset
5. capable of involving practically every structure in the body in its course;
6. distinguished by florid manifestation on one hand & years of completely asymptomatic latency on the other;
7. able to simulate many diseases in the field of medicine & surgery;
8. transmissible to offspring in man;
9. transmissible to certain laboratory animals;
10. treatable to the point of presumptive cure.

- **Etymology of syphilis**

Syphilis derives its name from a poem, syphilis sive morbus gallicus written by a physician, Girolamo Fracastoro. In the poem syphilis happens to be the name of a Shepherd who suffered from this disease as a curse for insulting the god Apollo.

It is also known as morbus gallicus, lues venereum, Neapolitan disease, English disease/ Firangi disease, French disease, Portuguese disease, the scourge of renaissance.

- **What are the different hypotheses explaining the origin of syphilis?**

1. **Pre- Columbian hypothesis -** this hypothesis claims that along with other treponemal diseases syphilis was widely spread in both old and new world.

According to this hypothesis yaws occurred as a consequence of mutation in Pinta and endemic syphilis emerged as a consequence of climatic changes in yaws and sexually transmitted syphilis emerged from endemic syphilis due to lower temperature of post-glacial era.

Mutation	Mutation
Pinta	yaws > endemic syphilis > sexually transmitted syphilis

2. **Unitarian hypothesis –** it is a variant of Pre-Columbian hypothesis. According to this theory, both syphilis and non-venereal treponemal diseases are variants of the same infections and the clinical difference happens only because of geographic and climate variation.
3. **Columbian hypothesis –** syphilis was unintentionally brought from the new world (New York) by Christopher Columbus and his crew after his voyage from America in 1493.

- **Named terminologies in syphilis**

Syphilis insontium

Accidental inoculation among medical personnel by prick on the digits or spilling of syphilitic material to the eyes leading to syphilis

Chancre redux

Relapse of primary chancre

Pseudo chancre redux

It occurs at the site of original chancre, however unlike true chancre redux, it is a non-infectious granulomatous lesion of late syphilis from which treponema cannot be recovered.

Condom chancre

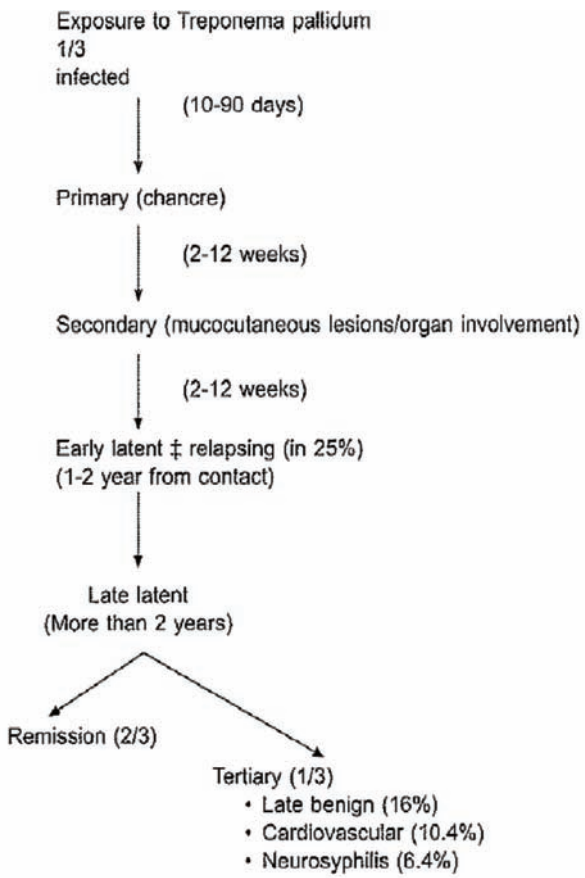
Chancre that occurs over the proximal part of the penis or the pubes or the groin. The distal part of the penis will be protected by the condom during the intercourse.

Syphilis d emblee

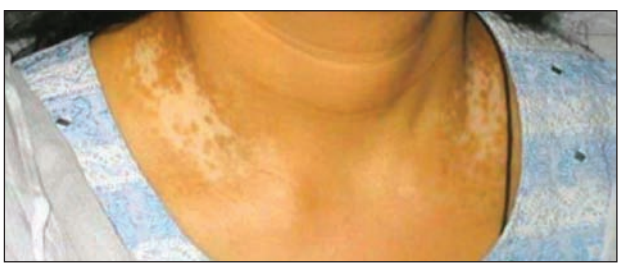
Syphilis resulting from deep inoculation of treponemes due to puncture wound or blood transfusion with infected blood. Primary stage is absent and the patient directly presents with signs and symptoms of secondary syphilis.

Syphilis incognito- form of latent syphilis where seropositivity has been detected without any past or present manifestation of syphilis.

- **Describe the natural course of untreated syphilis?**



- **What is Dory flop sign?**
  In primary syphilis induration of the ulcer on the inner side of the prepuce gives rise to a flip on retraction of preputial skin a.k.a dory flop sign.
- **Which strain is used for the preparation of antigens for treponemal tests?**
  Reiter's strain
- **What are the causes for biological false positive VDRL ?**
  Acute reactions (<6months) – associated infections, immunization, narcotic abuse, pregnancy.
  Chronic reactions (>6months) – Idiopathic, old age, leprosy, autoimmune disease, connective tissue disorders, narcotic abuse, malignancy, dysgammaglobulinemia.
- **Which is the earliest cutaneous eruption in secondary syphilis?**
  Macular syphilide/ Roseolar syphilide (appears at about 8weeks- more frequently involves palms and soles).
- **What is the Buschke ollendorf sign?**
  Deep dermal tenderness elicited over the rash on pressing the papule directly with a small blunt object.
- **What is leus maligna / malignant syphilis?**
  It is a form of pustular syphilide characterised by a prodrome of fever, headache, muscle pain, papulopustular eruptions that soon become necrotic, marginated ulcers covered with thick rupioid crust. Visceral involvement and severe toxaemia can be seen.
- **Identify the condition?**



Collar of Venus/leukodermacollisyphiliticum – cutaneous lesions of secondary syphilis heals with macular pigment loss on the hyperpigmented background on the sides of neck.

- **What is JARISCH–HERXHEIMER reaction?**
  After treatment for primary or secondary syphilis, about one third or two-thirds of patients, respectively, have a reaction characterized by chills, fever, arthralgias, headache, and transiently increased prominence of lesions. It occurs due to the release of treponemal constituents. The onset is within 4–6 hours after treatment, and it subsides within 24 hours. It is not an indication for discontinuance of treatment; most reactions can be managed by reassurance of the patient and aspirin or ibuprofen.
- **What is Hoigne's syndrome/ Procaine psychosis?**
  Acute psychotic episode, severe anxiety, agitation, vertigo, seizures, visual and auditory hallucinations. It is thought to be due to brain micro embolism secondary to inadvertent injection of crystals of procaine penicillin intravenously.
- **Which is the most sensitive and specific serological test for syphilis?**
  TPHA [TPHA is more sensitive than FTA-ABS except in 3rd to 4th week of infection]

- **Treatment of syphilis (CDC guidelines 2021)**

Risk category	Recommended regimen
Primary, secondary and early latent	Benzathine penicillin G 2.4 million units IM , single dose
Late latent syphilis	Benzathine penicillin G 2.4 million units IM , three doses 1week apart
Neurosyphilis, ocular and otosyphilis	Aqueous crystalline penicillin G 18-24 million units per day, administered as 3-4 million units IV every 4hours or continuous infusion *10-14 days Alternative Procaine penicillin G 2.4 million units IM + probenecid 500mg orally QID, both for 10-14 days.

- **How to manage syphilis in pregnancy?**
  Inj. Benzathine penicillin G 2.4 million units IM , single dose(after test dose).
  Pregnant women who are allergic to penicillin should be desensitised and treated with penicillin.
  WHO & NACO recommended alternate regimens are:
  Tab Erythromycin 500mg QID, orally *15 days
  Note : Erythromycin estolate is contraindicated in pregnancy as it is hepatotoxic. Only erythromycin base or erythromycin ethyl succinate should be used in pregnancy.

- **How to differentiate various pathogenic treponemes?**

Features	Yaws	Endemic Syphilis	Pinta	Venereal Syphilis
Organism	T. Pallidum ssp. pertenue	T. Pallidum ssp. endemicum	T. Pallidum ssp. carateum	T. Pallidum ssp. pallidum
Geographical distribution	Africa, Asia, Central America, Pacific Islands	North Africa, South East Asia, Arabian Peninsula	North Africa, South East Asia, Arabian Peninsula	Worldwide, more in developing countries
Occurrence	Endemic	Endemic	Endemic	Sporadic
Climate	Humid, warm	dry, warm	Semi-dry, warm	-
Age group	< 15 years (early childhood)	< 15 years (early childhood)	15-30 years	Adulthood
Spread by direct contact	Yes	Yes	Yes	Rare
Primary lesions	Common	Rare	Common	Common
Location	Extremities	Oral mucosa	Extremities	Genitals
Secondary lesions				
Extent	widespread	Limited	Limited	widespread
Location	Skin, bone	Intertriginous areas	Local	Skin, mucosa, systemic
lymphadenopathy	Common	Common	Rare	Common
Constitutional symptoms	Rare	Rare	Never	common

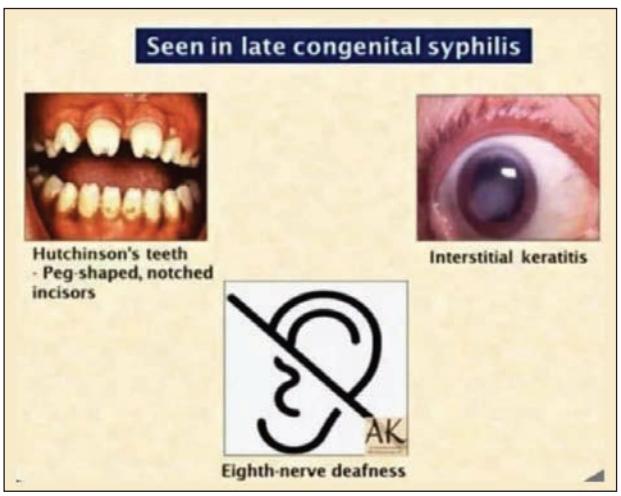
Congenital syphilis

- **Describe the laws of congenital syphilis**

1. **COLLES'S LAW(1837) -** States that syphilitic infants can transmit the disease to previously healthy wet nurses but never to their own mother.
2. **PROFETA'S LAW(1865) -** States that a healthy infant born to a syphilitic mother is immune to the disease.
3. **KASSOWITZ'S LAW(1876) -** If a woman with untreated syphilis has a series of pregnancies, the likelihood of infection of the foetus in later pregnancies becomes less.
4. **FOURNIER-FINGER CRITERIA** (FOR THIRD-GENERATION SYPHILIS) after Fournier (1891) and Finger (1900):

(a) Acquired syphilis must be demonstrated in grandmother and preferably also in grandfather.
(b) Prenatal syphilis must be demonstrated in the mother. Acquired syphilis must be excluded in her case and father must be proved healthy.
(c) There must be incontrovertible evidence of prenatal syphilis in the third generation.
(d) Manifestations must appear soon after birth in the second and third generations.

- **What are the components of Hutchison's triad?**



- **Vesiculobullous lesions are found in which type of syphilis?**
  Congenital syphilis
- **Signs in congenital syphilis**
  Barber pole appearance of umbilical cord- interspersion of blue and pink areas along with chalky white coloration on umbilical cord in spiral configuration due to necrotizing funisitis.
  Bucket handle sign - fracture through degenerated metaphysis.
  Bulldog facies - saddle nose, short maxilla and prominent mandible which are stigmata of early stages.

Celery stick sign - Plain film appearance of metaphyses characterized by longitudinally aligned sclerotic bands. Also seen in congenital rubella, toxoplasma and cytomegalovirus infections.
Clutton's joint - Chronic, painless, and insidious joint effusion of knees in late congenital syphilis.
Dubio's sign - Short incurved little finger that occurs as stigmata.
Ghost vessels - empty blood vessels extending from sclerotic to deeper layers of cornea occurring as stigmata of interstitial keratitis.
Ground glass cornea - Hazy cornea due to cellular exudation into deeper layers.
Hennebert's sign - nystagmus caused by pressure applied to a sealed external auditory canal. Also positive in Meniere's disease.
Higoumenaki's sign - unilateral enlargement of sternoclavicular articulation in late congenital syphilis.
Hutchison's teeth - Abnormal permanent upper central incisors that are peg shaped and notched and widely spaced.
Mulberry molars (Moon/ Fournier molars)- The biting surface of the first molars is dome shaped and has multiple underdeveloped and poorly enameled cusps.
Natiform skull (Hot cross bun skull) - Frontoparietal bossing along with prominent suture lines of skull.
Olympian brow (Beetled brow) - bony prominence of forehead.
Onion peel periosteum - Successive layers of bone are laid down on the surface of the cortex in regular fashion giving this radiographic appearance in early stages.

Parrot's nodes - Localized osteoperiostitis of skull vault leads to the formation of round bony swellings in the frontoparietal region followed by permanent thickening of bones in late congenital syphilis.

Sabre shin/ Fournier's sign - Anterior convexity of tibia occurs due to thickening of middle third of the shaft.

Salmon patch - Due to circumcorneal vascularization, a dull pink patch occurs at the periphery of the cornea.

Salt and pepper fundus - Chorioretinitis giving rise to tiny light specks interspersed among dark specks.

Stokes facies - Inalert, sleepy, tired, dreaming appearance of upper face.

Tower skull - high cranium

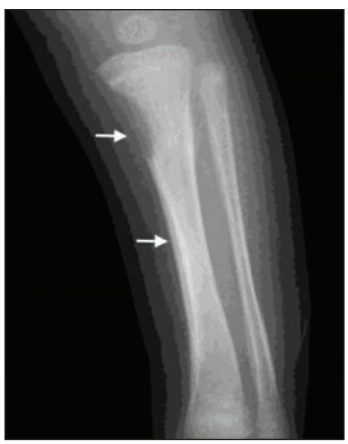
Virchow's sign - Tongue with smooth base

White pneumonia/ pneumonia alba - Firm and pale lungs due to inflammation and fibrosis in alveolar septa.

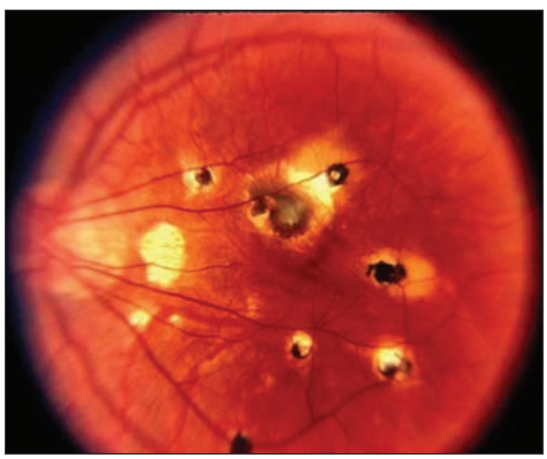
Wimberger sign/ Cat bite sign - Localized bilateral metaphyseal destruction of medial proximal tibias in early congenital syphilis.

Zig-Zag sign/Sawtooth metaphysis - serrated appearance at metaphysis of long bone.

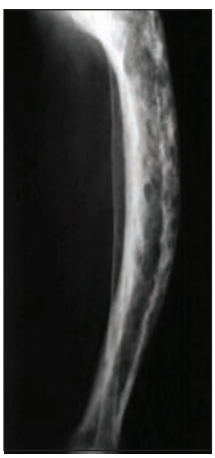
Dubio's sign



Wimberger sign



Salt and pepper fundus



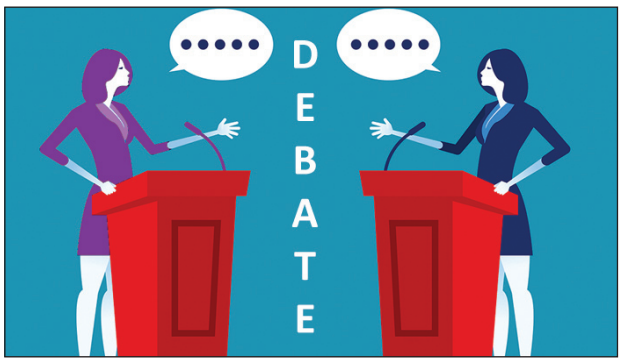
Sabre tibia

Dr Meghana C B
3rd year PG,
MIMS Mandya

Are millennial dermatologists straying far from clinical dermatology in pursuit of aesthetics?

This has been quite a controversial topic in the past few years. Is there really a need to focus on aesthetics? Has clinical dermatology taken a backseat? Dr. Ruta Joshi (Against) and Dr. Punya Suvarna (For) tackle this debate.

Dr. Punya Suvarna
Consultant Dermatologist,
The Skin Clinic, Mangalore.



Dr. Ruta Ujjval Joshi
Fellowship in Dermatosurgery
Assistant editor YUVADERMA

1. Change in patient profile

PS : We live in a visual world. Be it symmetry in nature, or brightness of colours on social media, our eyes are constantly taking in and analysing visual cues all around us. Being the most external feature in the human body, it is justifiable how people want to have better skin and hair so that they look their best and feel their best. As a result, it is only natural that people have become more aware about their appearances and are making a conscious effort to reach out to us dermatologists. Thus, along with patients lining up for treatment of various skin diseases, we also have individuals approaching us to look younger, fresher and aesthetically more pleasing. Being a part of the millennial generation, we are able to connect and relate more easily and can help achieve people's aesthetic demands. Thus, keeping in mind our clinical dermatology foundation, few millennial dermatologists have chosen to cultivate their niche in aesthetics.

RJ : In the past decade there has been tremendous change in the way skin disorders have been perceived. Previously skin disorders such as leprosy / psoriasis used to be neglected till it became absolutely life threatening and had to be treated. As we all know dermatology conditions are chronic and require regular follow-up. Due to lack of awareness patients used to be irregular at follow ups. This scenario has changed due to increase in literacy rates and also rise in social media awareness thus patients are motivated to follow up regularly. It is imperative for us to sharpen our skills in medical dermatology as that is our true identity and patients trust our knowledge.

2. Scope for Diagnosis and Research

PS : Aesthetic dermatology is an ever-expanding field. With the development of energy-based devices, we have made great progress in the treatment of many cosmetic conditions. Gone are the days when one would

have to live with scars after acne/persistent pigmentation/receding hairlines. Though the initial focus may have been on aesthetic and cosmetic indications, we have extrapolated the applications in clinical dermatology. Thanks to ongoing research, we have been able to deal with the sequelae of most dermatological disorders and extend the principles of practice into the general setting. For example, the use of hyaluronic acid fillers in morphea patients, fading away traumatic/ surgical scars, removal of marking tattoos in Holocaust victims and reformed criminals. Moreover, the pursuit of clinically and visually appreciable research always makes it more satisfying for investigators as well.

RJ : When it comes to diagnosis and research, there is ample work being done in these fields. Many young dermatologists are choosing interesting clinical topics for research and thus making their mark in the field of dermatology. With evolution of new biologicals and new treatment protocols the future for young dermatologists in the field of clinical dermatology appears to be very bright. A couple of decades ago psoriasis used to be treated with only conventional medicine which used to be very disappointing in patients with recalcitrant lesions but now with newer molecules dermatology has become more than steroids and ointments which in my opinion has been embraced by all young dermatologists.

3. Focused Practice:

PS : It has been very common for many years for people to first approach their general practitioner/their pediatrician/their gynaecologist for any skin related issue. With a basic medical background, most skin issues are easily diagnosed and treated by them. Only when no/minimal response is seen, patients are referred to a dermatologist. This might lead to only a fraction of patients actually reporting to dermatologists for skin and skin related issues. As a result, there is a need for dermatologists to focus and market themselves in a particular sub-field. The most popular, rewarding and satisfying being trichology and cosmetic dermatology, both which deal with aesthetics and enhancing one's beauty.

RJ : In recent times with growing awareness about skin diseases, patients are choosing to meet doctors whose expertise belong to the same field, may it be psoriasis or atopic dermatitis or pediatric dermatology. This has led to the younger generation choosing to pursue fellowships or observerships more in number. There are a significant number choosing to train in aesthetic dermatology but clearly doing so due to lack of exposure during residency. Recently many branches are gaining more popularity like pediatric dermatology, dermatopathology and trichology. Dentists, plastic surgeons and many other unqualified professionals claim to do the same work in the field of aesthetics. So, it is important to be unique and strengthen our core practice.

4. Need for further training and exposure

PS : In our current teaching program at the postgraduate level, not much emphasis is given on aesthetics and cosmetic dermatology. A brief basic introduction about energy-based devices, injectables and cosmetic procedures

"Have the courage to swim against the tide, have the courage to be happy". There are a lot of dermatologists who still have a passion for core clinical dermatology and are reluctant to practice aesthetics/ procedural dermatology. It's a choice that can be very fulfilling if done right and can be rewarding in the long run.

is done in most colleges. With the field of general dermatology being so vast, it is only natural that something equally as challenging as aesthetics requires a further observership/ fellowship/experience. The proper counseling and selection of patients for different therapeutic options is a skill that needs to be inculcated. Only once you are strong in your clinical knowledge, can you build up on your other skills. After all, even tinea patients are more worried about the residual pigmentation as opposed to the spread of the disease itself.

RJ : During residency a lot of emphasis is given towards diagnosis of clinical dermatology cases and very less towards treatment and the different protocols to follow. There is a rise in the need for revising current curriculum to incorporate all the newer treatment options so that young dermatologists develop interest in these fields and further pursue research as this is the future of dermatology.

5. Awareness about skin issues in public

PS : A booming increase in awareness about skin and hair related problems has led to more and more people approaching dermatologists. However, there is a tilt towards the aesthetic aspect of skin care than the medical based management of skin diseases. Even on social media, the general population is always keener to know tips and tricks to make oneself appear more pleasing. There is only a smaller set of people who might be suffering from a skin disease who will want to know about it. We can generalize anti-ageing to the general population but cannot do the same with something like psoriasis. As being part of the service industry, it is only natural that we cater to what our audience needs and wants to know about.

RJ : Social media has become one of the most integral parts of all our lives, it is the quickest and most efficient mode to be in touch with the general population and for getting noticed for your work. Through social media, awareness regarding aesthetic dermatology has increased. Aging and beauty are a common concern. But, there is a need to have more content about clinical dermatology which will help reduce quackery in treatment of serious disorders like pemphigus and psoriasis. IADVL SIG task force do have outreach programs for the same and many young millennial dermatologists are becoming a part of it. Social media is flooded with reels/ stories/ posts regarding skin care/botox/fillers, I think there is a need to change the narrative and focus on clinical dermatology issues cause most of the general population are unaware

of our field and consider it as a branch dealing with only beauty and glamour.

6. Procedural dermatology being used more for aesthetic indications?

PS : There is only a finite amount of results we can achieve with topical or oral medication. Sometimes, there is a need for stronger and more effective options which calls for the use of procedural based dermatology. With the easy availability of over-the-counter topicals, patients turn to us for other therapeutic options that can give equal/better/longer lasting results in a shorter period of time. Whether it be medical/aesthetic dermatology, interventional procedures can help in improving results.

RJ : Procedural dermatology has advanced by leaps and bounds in the past decade. More so being the last resort of treatment for many disorders. Considering vitiligo surgeries, there is so much social stigma surrounding this which leads to mental health issues. Some may view this as an aesthetic procedure but in reality it is far superior, it is a gift of new life to patients. Many conventional aesthetics procedures have found their way into clinical disorders such as botox for hyperhidrosis indicating that millennial dermatologists are not straying away from clinical dermatology but in fact are strengthening their roots.

Editor's note:

There are two sayings I want to quote here

1. "If you can't control the current of the river then don't fight it, just go with the flow". In an era where the demographics of a dermatology outpatient is changing rapidly, especially in the metropolitan cities and larger towns, most patients are looking for aesthetic solutions. Though the learning curve is easy, dealing with real life scenarios needs skills beyond formal training - that can only be learnt by embracing aesthetic dermatology as a part of your clinical practice (Sometimes even a larger chunk of it).
2. "Have the courage to swim against the tide, have the courage to be happy". There are a lot of dermatologists who still have a passion for core clinical dermatology and are reluctant to practice aesthetics/ procedural dermatology. It's a choice that can be very fulfilling if done right and can be rewarding in the long run.

I would like to conclude this debate by saying that no matter what your preferences are, it's time to acknowledge the fact that aesthetic and procedural dermatology is here to stay (for a very very long time) and one should always have a balance between both types of practices. A strong clinical foundation with precision hands can take you a long way in one's career. Frequent polishing and honing your skills and strengths is the way to a fulfilling practice no matter where you are practicing.

STATE-OF-THE-ART DERMATOLOGY!

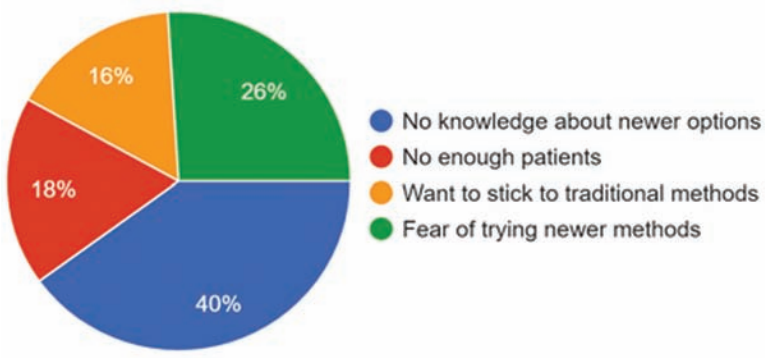
A survey on recent updates in dermatology among residents

Dermatology is a subject that continues to grow and expand. And it is important that we keep growing too, as dermatologists. Knowing the newer treatment options is as necessary as knowing the traditional methods. It is rather fair to treat a patient with a new drug than to keep using an old one just because we do not have the time to update ourselves. Of course we mustn't forget our basic medications but it is equally important that we upgrade our knowledge from time to time and the earlier, the better it is.

A small survey was conducted to know how well the residents are updated regarding the advances in dermatology and also to know what challenges were faced by them in doing so. An online questionnaire was prepared which included questions concerning various aspects of dermatology. A total of 63 residents all over Karnataka participated in the survey. We tried to assess their knowledge on advances and updates in dermatology from treatment of warts to dermatosurgical procedures and obstacles they went through during their residency.

IMMUNOTHERAPY IN WARTS : It was observed that almost 90% of residents use immunotherapy for warts and most preferred intralesionsal therapy is still MMR vaccine (71.4%), despite the availability of many newer agents. Although a good number of residents do use Vitamin D (52.4%), BCG vaccine (38%), Bleomycin (21%), Mycobacterium w vaccine (13%), Candida antigen (11%) and a small number of them use Hepatitis B vaccine and HPV.

The most common systemic therapy used was oral Zinc (87%). Other treatment options used were Autoimplantation (57%), Levamisole (38%), Cimetidine (20%), Ranitidine (3.2%) and Acitretin (1.6%).



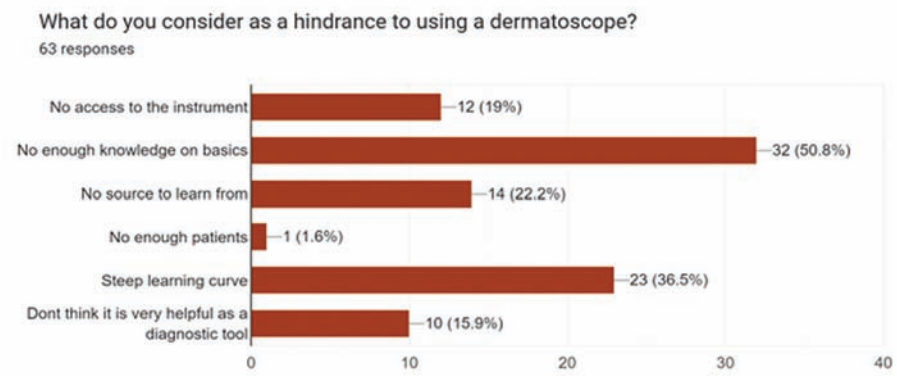
A noticeable number (27%) of residents do not use systemic immunotherapy to treat warts and the reason was that they had no knowledge about newer options. The other reasons were fear of trying newer methods, no enough patients and wanting to stick to traditional methods.

DERMATOSCOPY : Although a majority of residents did have easy access to dermatoscope, a significant number (21%) did not. It's surprising to see that 7.9% of residents have not used a dermatoscope at all.

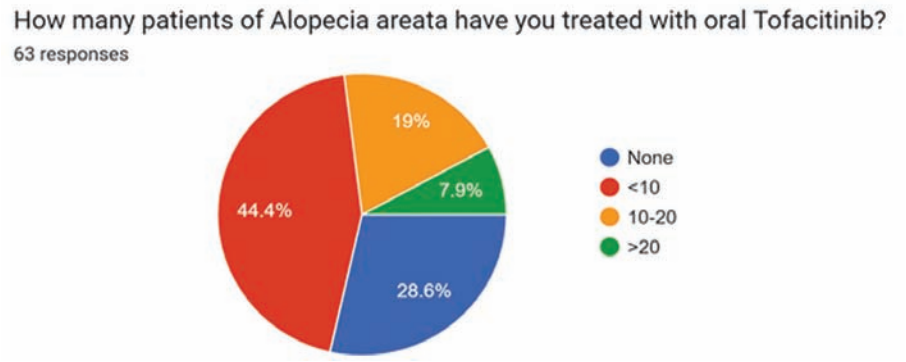
84 % of residents use a dermatoscope to diagnose cases during their OPDs. Only a small number of residents (7.9%) use dermatoscope on every patient whereas the majority (67%) uses it only when diagnosis becomes difficult.

51% believe that there is reduction in number of scalp biopsies in at least 50% of cases of scalp disorders since the advent of trichoscope. 12.5% think that there is not reduction whatsoever in the number of scalp biopsies. Majority of residents say that dermatoscope helps differentiate the different pigmentary disorders.

These are the reasons that prevented residents from using a dermatoscope.

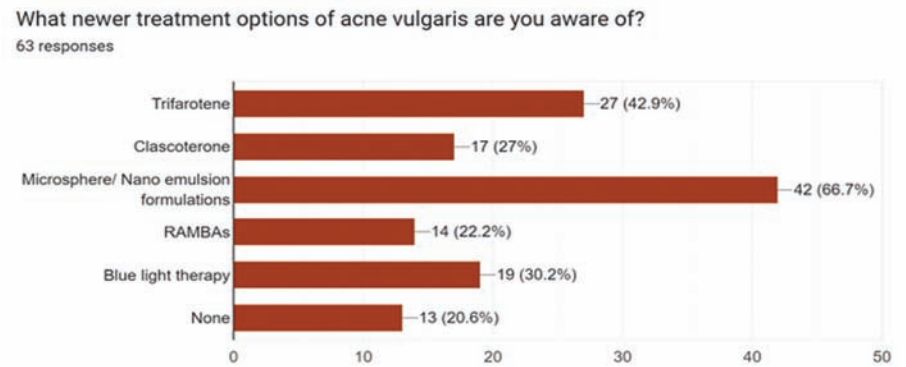


ALOPECIA AREATA : The most common medication being used is still corticosteroids (intralesional). The systemic medications used by residents are oral corticosteroids (85%), Tofacitinib (63%), Cyclosporine (38%), Apremilast and oral minoxidil.



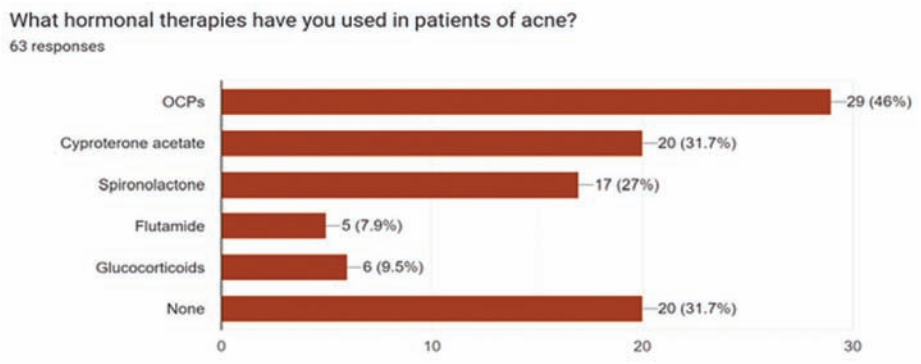
8% of residents have treated more than 20 patients of Alopecia Areata patients with Tofacitinib whereas 28% have not used Tofacitinib at all.

ACNE VULGARIS : When it comes to using topical retinoids for acne vulgaris, older and newer generation retinoids are equally used by the residents.



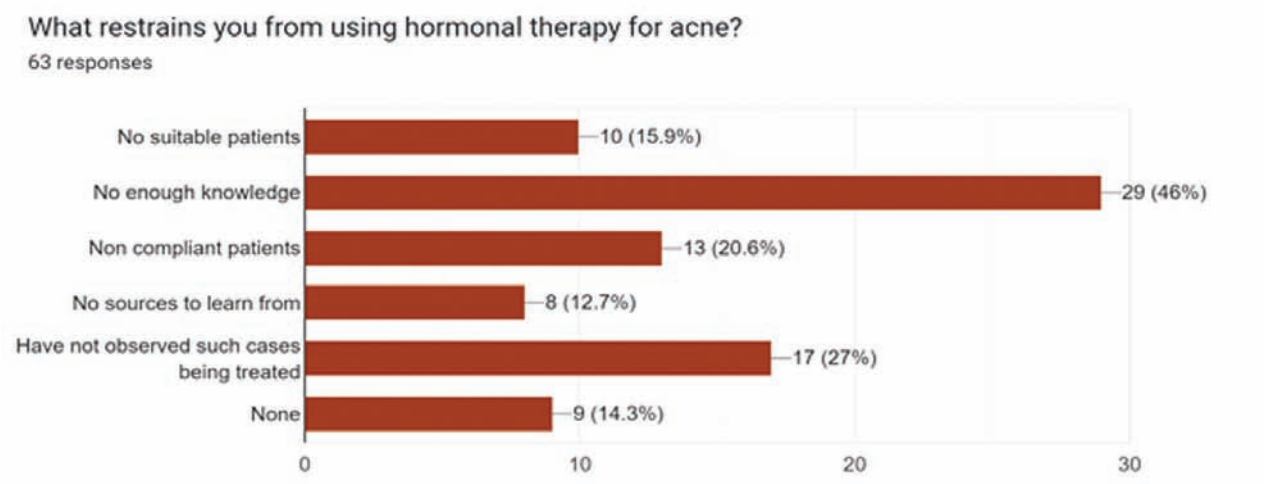
When asked about newer treatment options for acne, majority of residents were aware of them. But 21% of the residents were not acquainted with any of these newer agents.

Almost all residents have treated cases of adult onset acne. Most common hormonal therapy used was Oral Contraceptive pills (46%).



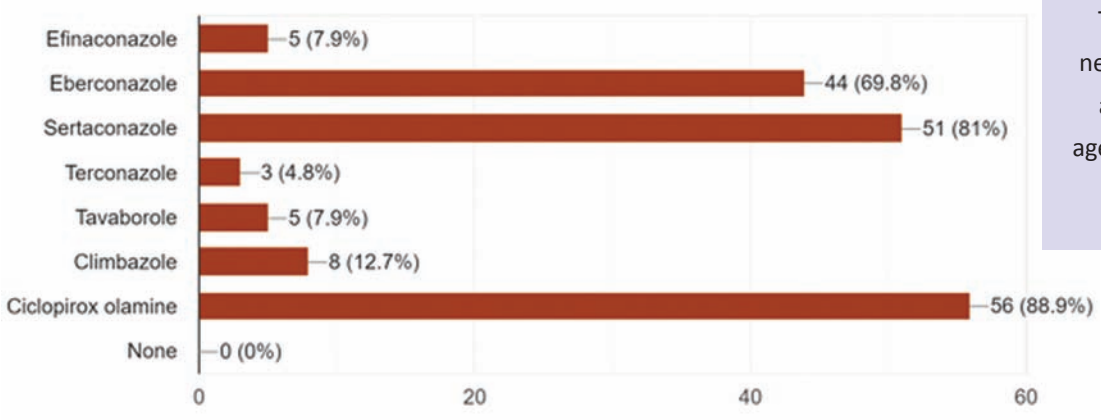
32% of residents have not used any of the hormonal therapies available.

The difficulties faced by the residents to use hormonal therapy as a treatment option for acne are as follows:



INFECTIONS : Despite the presence of many topical antibiotics, most commonly used is mupirocin. Other agents used are fusidic acid, ozenoxacin and silver sulfadiazine. Since the arrival of ozenoxacin, 22 % of residents prefer using ozenoxacin every day in their OPD practice.

5% of the residents are still unaware of the newer antifungal medications that are available. 60 % of them prefer using the newer antifungals and 40% prefer the older drugs.

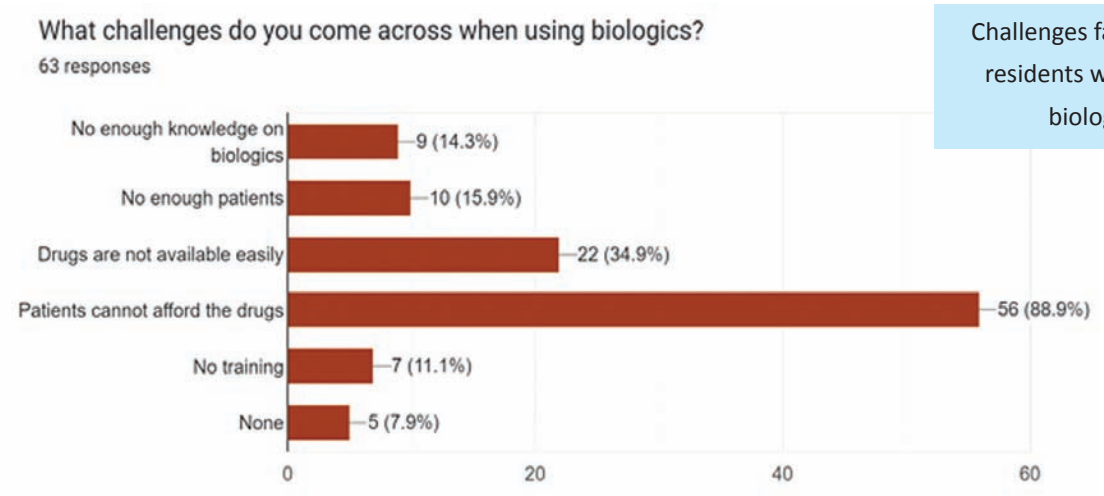


These are the newer topical antifungal agents used by residents.

ATOPIC DERMATITIS : Only a minority of residents use Tofacitinib and Crisaborole to treat atopic dermatitis. A huge number (67%) use neither. Tofacitinib (27%) is being used more commonly than crisaborole among residents.

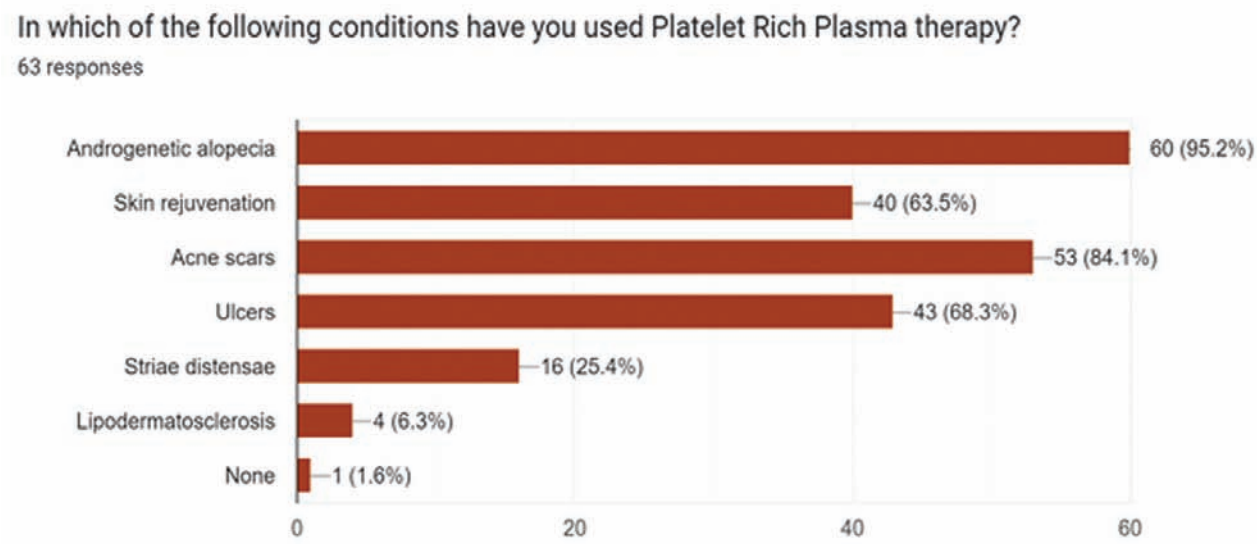
BIOLOGICS : The most commonly used biologic among residents is Rituximab. Other agents being used are Secukinumab, Adalimumab, Tofacitinib, Infliximab, Baricitinib, Ustekinumab and Golimumab. 17% of residents have not used any biologics during their residency.

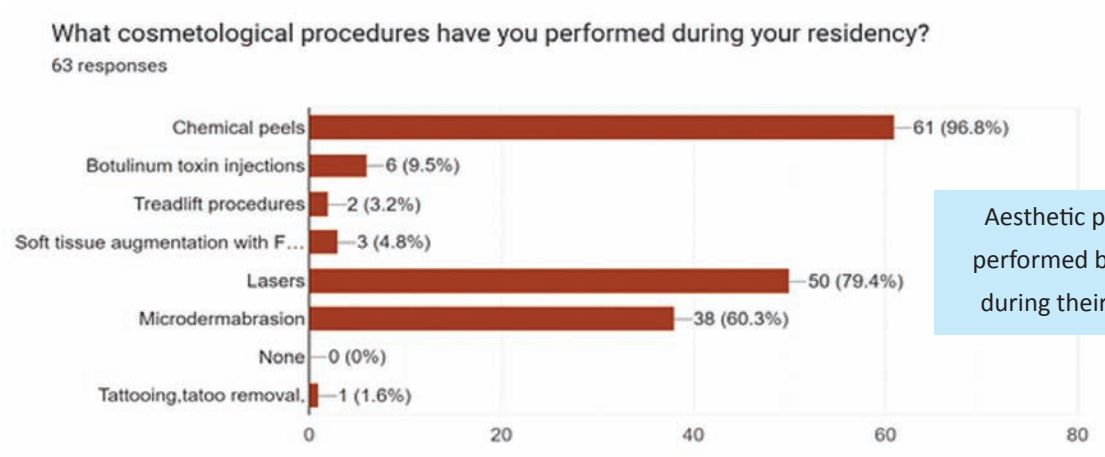
Majority (60%) of the residents prefer Rituximab over DCP in cases of vesiculobullous disorders.



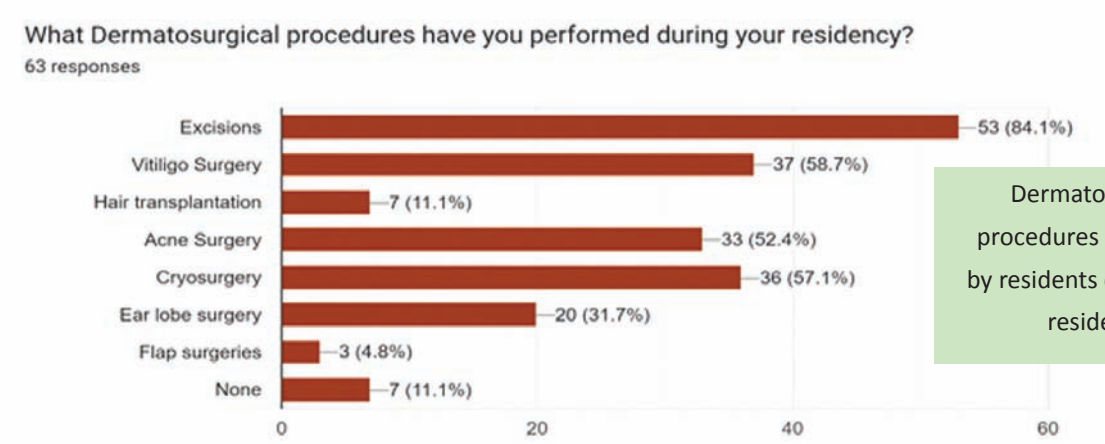
Challenges faced by the residents when using biologics.

AESTHETIC AND DERMATOSURGERY : Residents have used Platelet Rich Plasma therapy for variety of conditions:





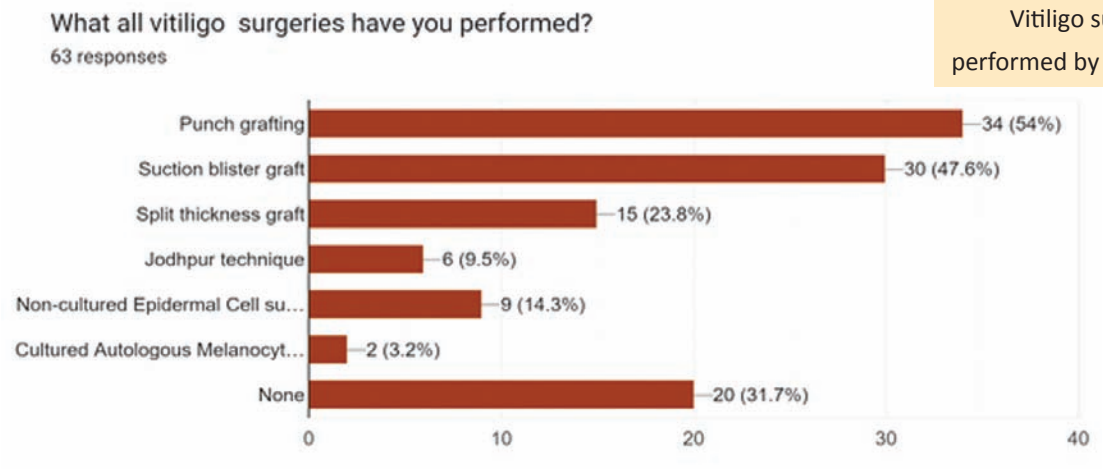
Aesthetic procedures performed by residents during their residency

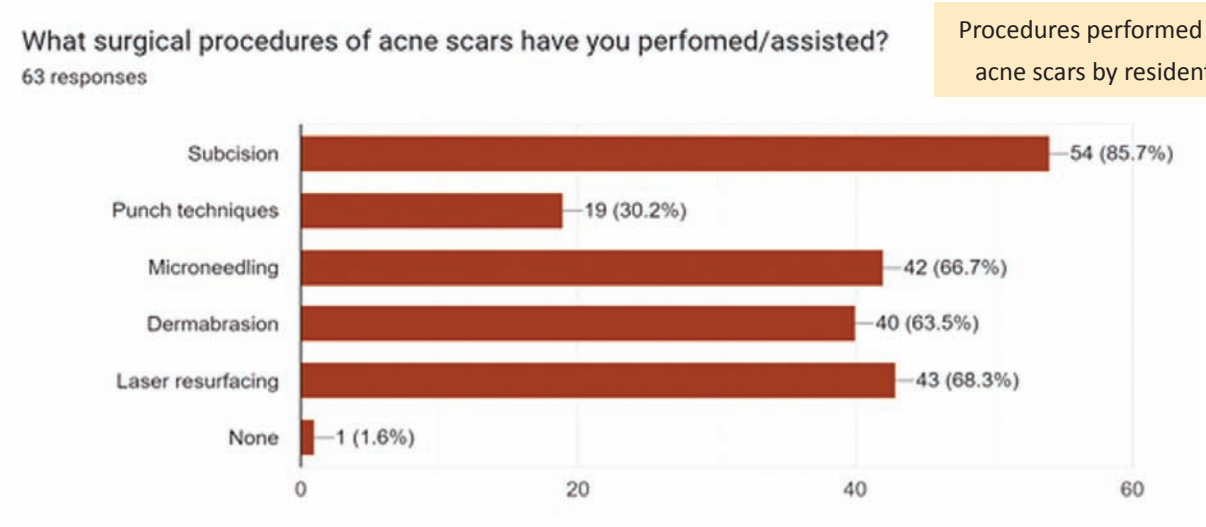


Dermatosurgical procedures performed by residents during their residency

Almost half of the residents have performed vitiligo surgeries during residency but a significant number (36%) did not. And majority (74.5%) prefers Tissue graft techniques over cellular graft techniques.

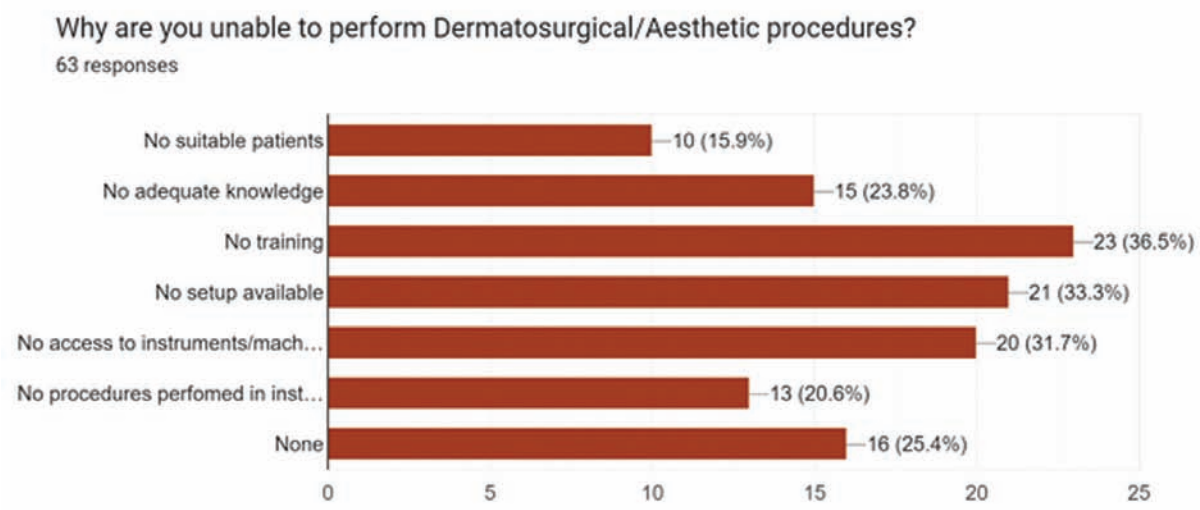
Vitiligo surgeries performed by the residents

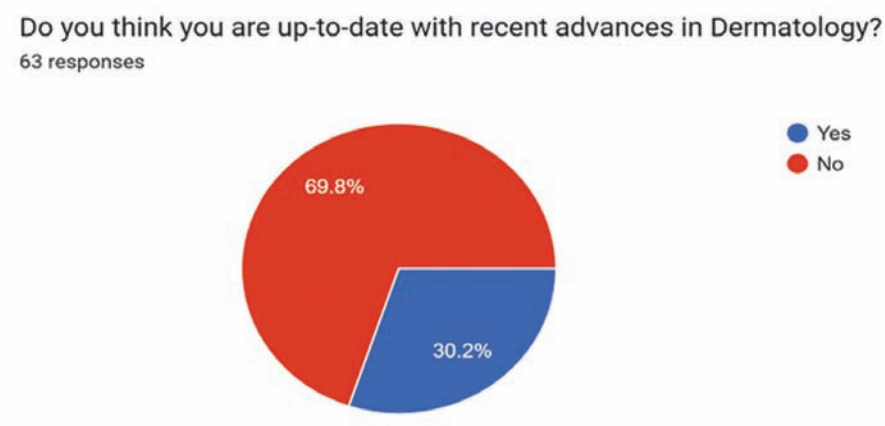




Procedures performed for acne scars by residents

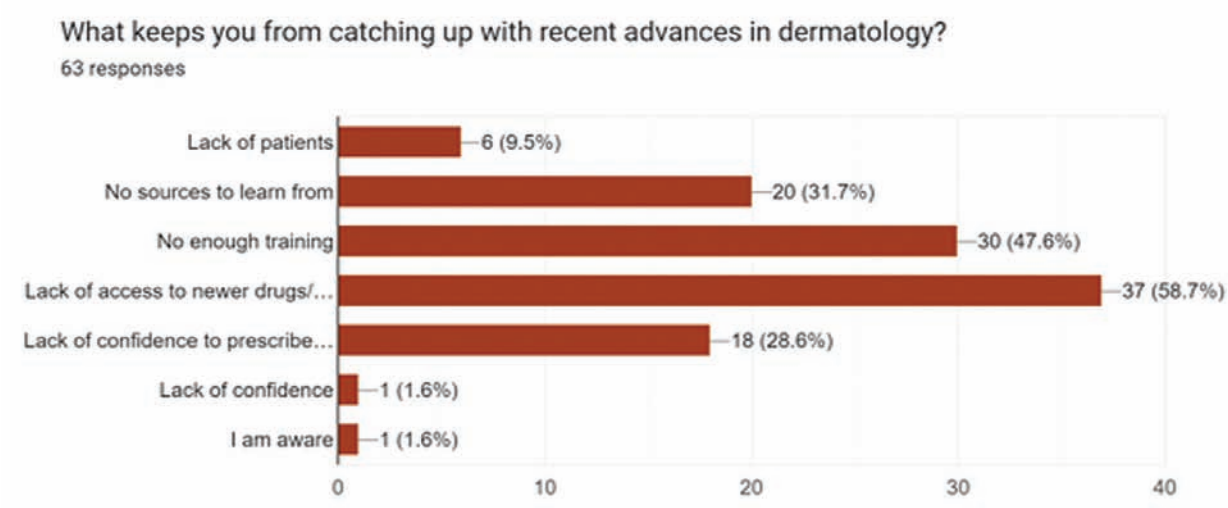
The reasons for not being able to perform aesthetic/dermatosurgical procedures during residency are as follows:





The residents were asked if they were up-to-date with recent advances in dermatology, a huge number (69.8%) believed that they did not.

The reasons that prevent them from catching up with the recent advances are as follows:



There are numerous problems faced by the residents. Some of which are inevitable like patient's affordability or lack of patients especially during COVID crisis. But there are a wide variety of journals, books, and unlimited internet access and of course our seniors and teachers who can be approached with no hesitation. To conclude, we must not limit our boundaries and keep learning new things each day from the time of residency.

Learn continually – there is always "one more thing" to learn!
- Steve Jobs

Concept by
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MIMS, Mandya
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The International Language of Dermatology

At the international conference, we witnessed dermatologists from all nationalities, cultures and walks of life. We participated in worldwide panel discussions that increased our understanding and expanded our perspectives. We paid attention, tried to comprehend, participated and devoured whatever new information was presented to us. Meeting and conversing with Dr Jean Bolognia, whose textbook we frequently testify for, was another delight.

Around 1.5 years ago, I read an archived edition of YUVADERMA that contained an interview with a distinguished professor, who urged post-graduates to participate in an international conference at least once while completing their residency. Needless to say, it took me until a year later, when a clinical case I had documented was chosen for an oral presentation at the Dubai Derma conference, for me to realize the significance of his words.

My department's requirement for attending a conference is having something to contribute to it in the shape of a pertinent poster or paper, thus my head of department and staff were encouraging and motivated us to go ahead with our endeavor. The doors of opportunity began to open for me and my fellow graduates at this moment.

Of course, as second-year post-graduates, we were nervous and apprehensive about what was to come. We were thrilled, but we were also unsure of our ability to compete with dermatologists from other parts of the world. Thoughts of our HOD's frequent encouragement to be fearless and self-assured, as well as his reminders that our competence in dermatology and strong interest in the field makes us inferior to none, reverberated in our heads constantly.

At the international conference, we witnessed dermatologists from all nationalities, cultures and walks of life. We participated in worldwide panel discussions that increased our understanding and expanded our perspectives. We paid attention, tried to comprehend, participated and devoured whatever new information was presented to us. Meeting and conversing with Dr Jean Bolognia, whose textbook we frequently testify for, was another delight.

And in that manner, a somber group of dermatology residents gave confident oral and poster presentations, grabbing the attention of everyone in the room. To top it all off, one of us even received an award for an outstanding poster presentation!

Five months later, we presented at the European Society of Pediatric Dermatology in Munich. While there, we had the chance to speak with renowned pediatric dermatologist Dr. Rudolf Happle, who had fascinating stories to share with us about his experience treating patients while visiting India decades ago.

I learned from my experience at international conferences that dermatology has no boundaries or constraints. It is a never-ending well of wisdom that has unlimited knowledge to impart to everyone across the globe. It taught me that even though our fellow dermatologists may come from many nations, have various cultures, and speak various languages, there is one language we all share and that is – the language of dermatology.

Photos with Dr Rudolf Happle and Dr Jean Bolognia

Dr. Andrea Rachel Castelino
JR3, KIMS, Bangalore.

DERM-CATCHERS!

1) I am known for things many
I am there when it's sunny
I am on faces, as whole or part
In Leus, Eyes-Ear-Teeth is my art
I can be true or false, as a sign
As a pioneer, I am just fine.

2) When disseminated, I am common
Present whenever there is sun.
In children, I am classy and dominate
Skin type 1 is my favourite.
Look for me in a Que at 12 and 15
In my family, I am the CAPtain.
Border likened to Great Wall of China
Cells with nuclei in a lamella.
Granular purple beneath me is absent,
Sometimes also known for being malignant.

3) I am a minor with a degree in major
And they call me iris,
I am mostly viral
But sometimes I have existential crises.
Whether typical or atypical
I never miss my target,
On skin I may be mild
Eyes you must not forget.
I am varied, in form and colour.
When I start, I blush
When atypical, I was known as Fuch.

4) I was born at the base,
I used to be a cell
My aim was to reach higher.
But then the disease arose,
And I am dead, known by many names
All I am now is a body,
But I am not sad and I have no blues
Because I am pretty, all dressed up in pink
You can still meet me, mostly at the junction.
And I serve even after death,
As an amazing clue,
For the things that happen at the intersection.

5) Relate the images for the diagnosis.

6) I can see it and feel it too
Them crawling all over
Burrowing and biting, they bug me all day and all night
They hurt me so bad, I scream and scratch
I try to secure them in a box

They don't exist, people claim
But I saw them just yesterday,
Millions of them moving like a troop of soldiers with a mission
I told people many, but none believed me
Then I found a friend, who could see it too,
All people said was 'it's just a madness shared by two'

I went to the doctors, not one but many
From city to city
They believed me just fine
But tried to convince me otherwise,
Like the bugs, my thoughts are stubborn
Neither leaves my mind, this malady no one can discern.

7) I told him, I fear the disease, where the rings accumulate,
And turn everything red
I told him, I also fear the sun that makes my skin fragile.

They divided me, called me by different names
Based on how I met-a-plight.
I knew I was not normal,
I looked at the first defect and cried ALAS!
By the end, I gathered some courage to face my life.

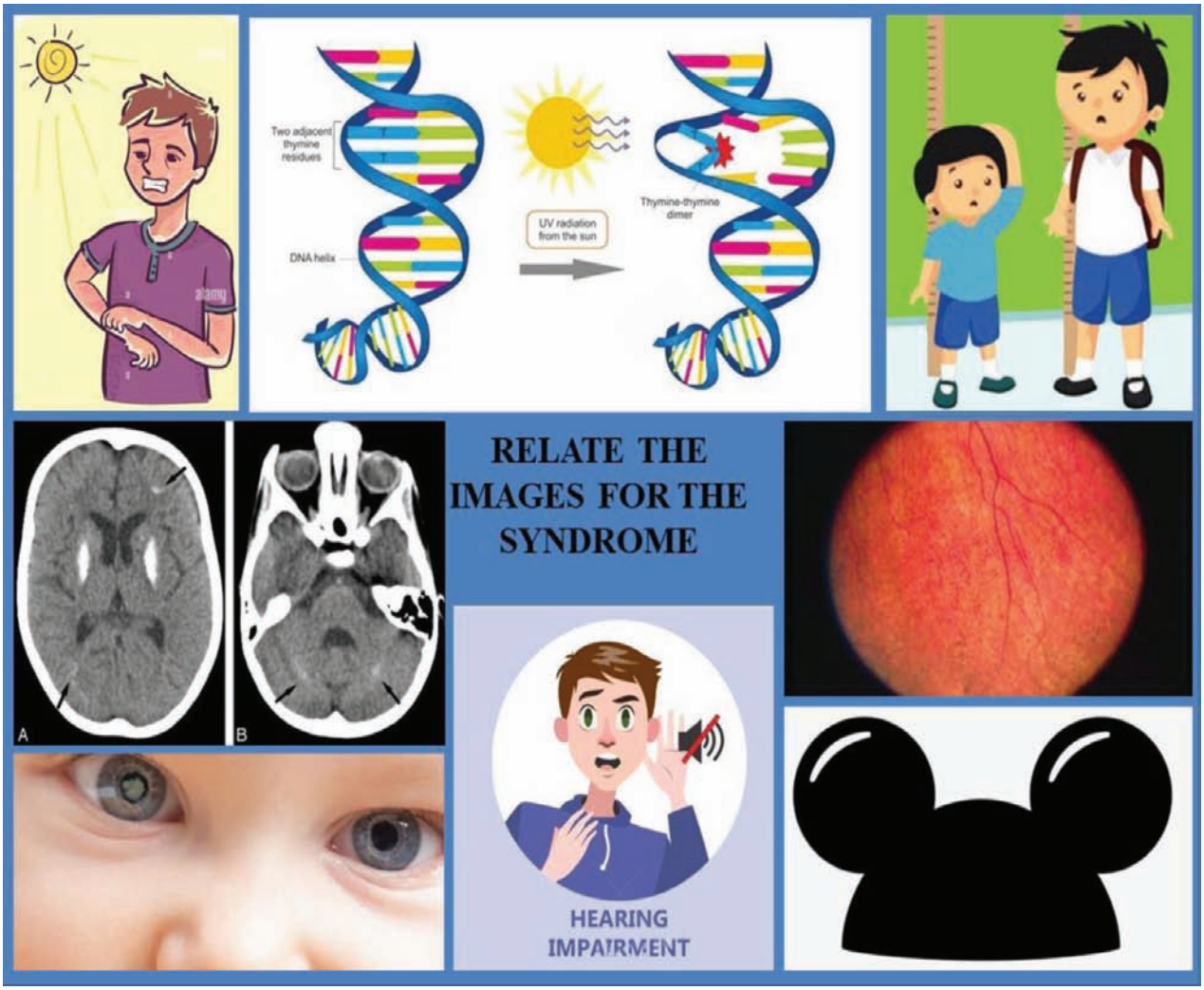
I have been to places many,
South Africa, Zimbabwe and Turkey
Met the Bantus, the werewolves and the monkeys.

When I was a child, I was given a Gun-threw it away as I grew up.
I have scars that make me look older than I am,
I have hair, too many, on face, body, everywhere

With the light of wood, you can see all my flaws.
Observe my skin too deep, you will see the festoons.
When you see me differently, you will find further clues.

Even if you are Sorry, 408 times, things don't change.
I may live or not, as one by one all my organs derange.

8) Related the images for the diagnosis

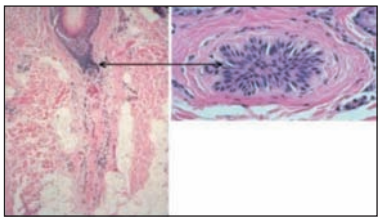
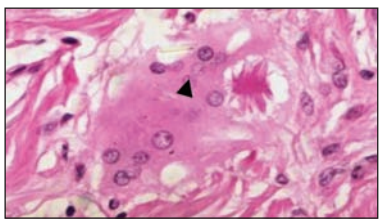
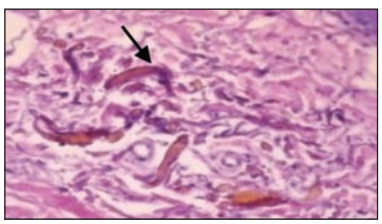
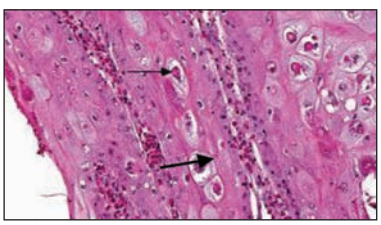
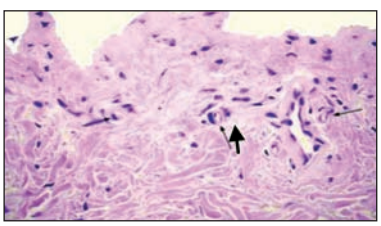


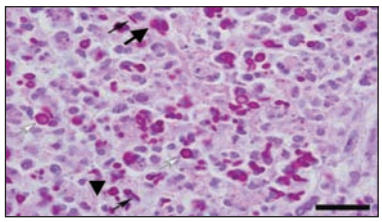
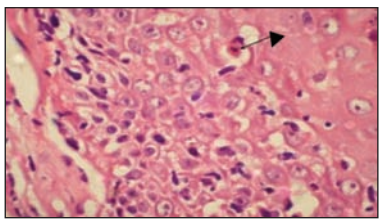
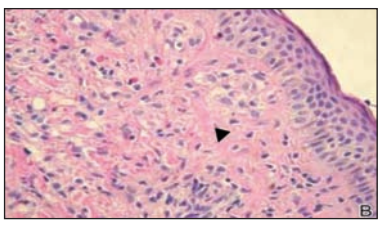
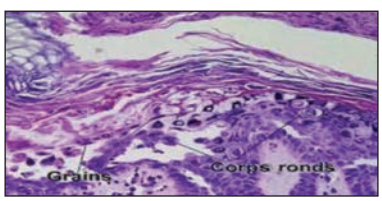
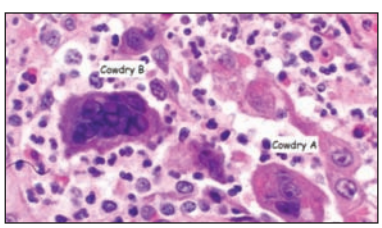
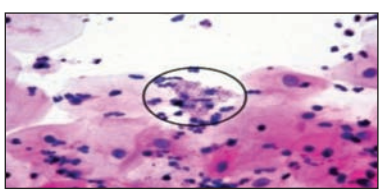
ANSWERS:

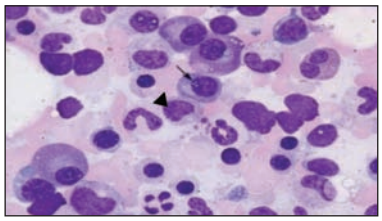
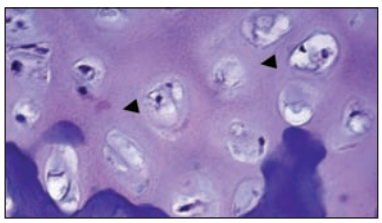
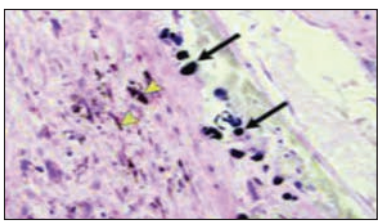
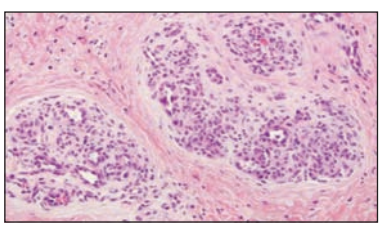
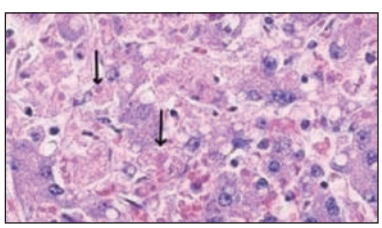
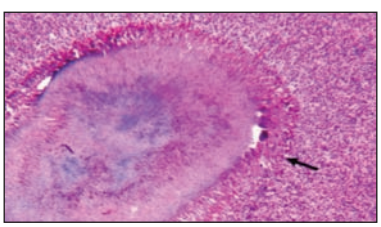
1. Sir Jonathan Hutchinson
2. Porokeratosis
3. Erythema multiforme
4. Civatte bodies
5. Bacterial vaginosis
6. Delusional Parasitosis
7. Porphyria
8. Cockayne syndrome

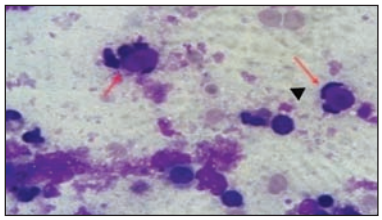
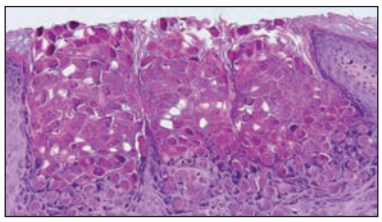
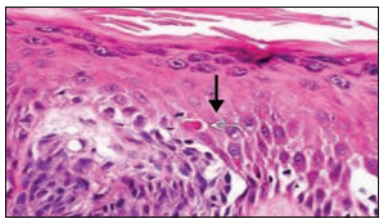
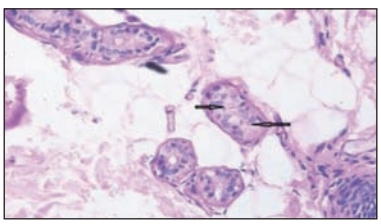
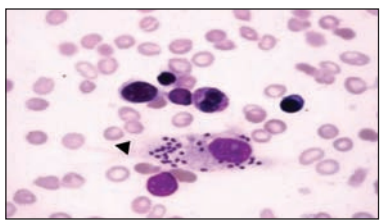
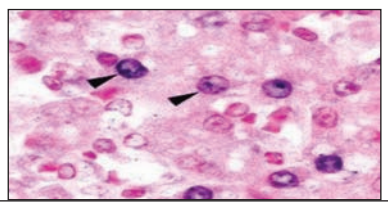
Dr Chinmai C Chikkalagi
Dermatosurgery fellow,
MIMS, Mandya
Assistant editor, Yuvaderma

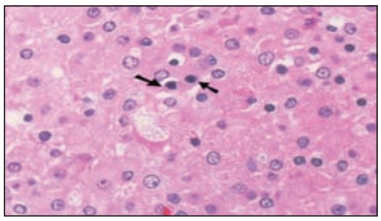
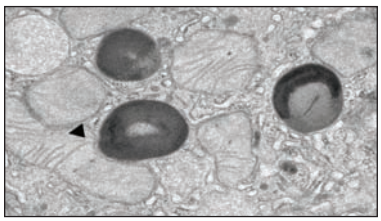
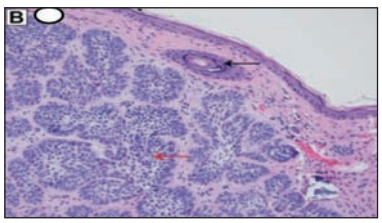
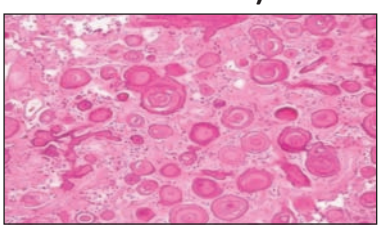
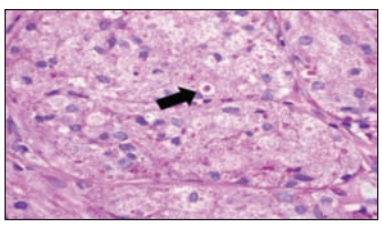
VARIED HUSK IN DERMATOLOGY

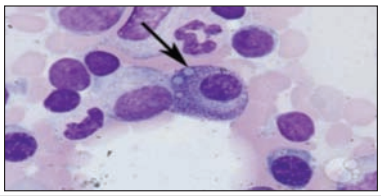
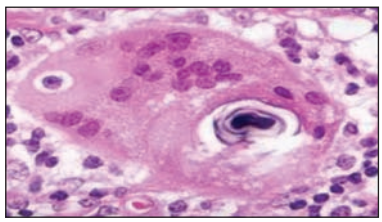
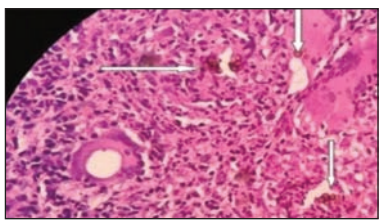
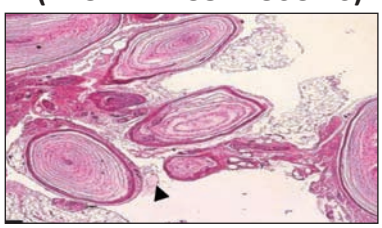
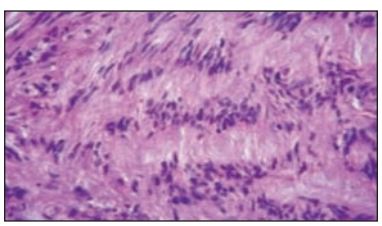
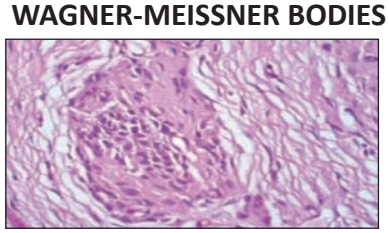
Sl NO.	BODIES	DESCRIPTION
1	**ARAO-PERKINS BODIES** 	These are clusters of elastic fibers formed within the lower part of follicular stellae like the rungs of a ladder during the catagen phase of a normal hair cycle and in Androgenetic alopecia.
2	**ASTEROID (STELLATE BODIES)** 	Small (3–5 µm), oval yeasts of Sporothrix schenckii may be surrounded by a thick, radiate, eosinophilic substance, which forms the asteroid bodies. Seen in Sarcoidosis, as star-shaped eosinophilic structures (10-15 µm). Other conditions include Foreign body giant cell reactions, Actinic granuloma, Necrobiosis lipoidica, Necrobiotic Xanthogranuloma, Fibroxanthosarcoma, & Cystic teratoma.
3	**BANANA BODIES** 	Yellow-brown or ochre colored, banana-shaped fibers in the papillary dermis due to the accumulation of homogentisic acid in alkaptonuria or following topical application of Hydroquinone, Resorcinol, Phenol, Mercury, or Picric acid.
4	**BOLLINGER BODIES** 	Large, granular, acidophilic, intracytoplasmic inclusion bodies observed in the infected epithelial cells of birds with avian pox. These are aggregates of Borrell bodies & are pathognomonic for fowl pox.
5	**CATERPILLAR BODIES** 	In subepidermal blisters of Porphyria cutanea tarda, the roof of the blister often contains PAS positive & diastase resistant eosinophilic bodies that are elongated & sometimes segmented resembling the larvae of butterflies. Also seen in Bullous pemphigoid, Junctional & Dystrophic epidermolysis bullosa, & Erythropoietic protoporphyria.

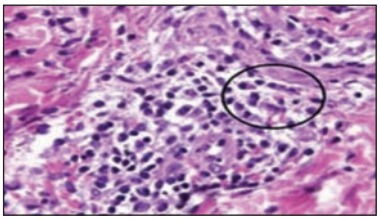
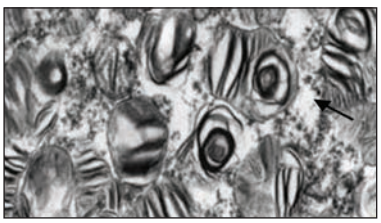
6	**CIGAR BODIES** 	In Sporotrichosis, Sporothrix schenckii appear as faintly stained, elongated or cigar-shaped free yeast cells within histiocytes, measuring 2–6 µm or more.
7	**CIVATTE (COLLOID, HYALINE, OR CYTOID BODIES)** 	Homogeneous, PAS positive & diastase resistant, eosinophilic rounded bodies of 10–25 µm in diameter, in the lower layers of the epidermis. Commonly seen in Lichen planus & other causes of interface dermatitis including Graft-versus-host disease, Lichen nitidus, LE, Drug reactions, & in Inflamed keratoses such as Lichenoid actinic keratosis & Lichen planus-like keratosis. They may even be observed in normal skin.
8	**COMMA-SHAPED BODIES** 	Seen within the histiocytes, formed by two electron-dense membranes of approximately 6 nm, separated by a light space of about 8 nm. Underlying conditions are Benign cephalic histiocytosis, Juvenile xanthogranuloma, & Cutaneous T-cell lymphoma.
9	**CORP RONDS & GRAINS** 	Dyskeratotic epidermal cells often observed in Darier's disease, Grover's disease, and Warty dyskeratoma. Corp ronds have a small pyknotic nucleus, a clear perinuclear halo, & brightly eosinophilic cytoplasm. Grains are small cells with elongated nuclei & scanty cytoplasm in the upper layers of the epidermis.
10	**COWDRY A & B BODIES** 	Intranuclear eosinophilic inclusion bodies are composed of nucleic acid & protein. There are two types: Type A (in Herpes infection & Yellow fever) and Type-B (in Polio & Adenovirus) Cowdry type-A inclusion bodies appear as droplet-like masses of acidophilic materials surrounded by clear halos within nuclei, with margination of chromatin on the nuclear membrane. Type-B bodies are not associated with any nuclear changes.
11	**DONOVAN BODIES** 	In Klebsiella granulomatis infection, macrophages contain encapsulated bacilli within vacuoles, which are seen as short bacilli, either singly or in clumps with bipolar condensations on Warthin-Starry or Giemsa staining.

12	**DUTCHER BODIES** 	These are PAS positive pseudo-inclusions formed due to invagination of immunoglobulin-filled cytoplasm into the nucleus being surrounded by clumped chromatin. Associated with Waldenstrom's macroglobulinemia, Diffuse large B-cell lymphoma, Multiple myeloma, & Chronic synovitis.
13	**FARBER BODIES** 	Curvilinear, tubular structures in cytoplasmic vacuoles due to the accumulation of ceramide in the lysosomes, seen in Farber's disease.
14	**GAMNA-FAVRE BODIES** 	Large basophilic inclusion bodies, composed of degenerated nuclear material, located in the cytoplasm of endothelial cells in Lymphogranuloma venereum.
15	**GLOMUS BODIES** 	Special arteriovenous anastomosis formed without the interposition of capillaries that is seen in finger pads, nail beds, palms, & soles. Being located within the reticular dermis & concerned with temperature regulation.
16	**GUARNIERI (PASCHEN) BODIES** 	Cytoplasmic aggregations of variola virus particles, which are also found in Vaccinia, Monkeypox & Cowpox.
17	**GUPTA BODIES (DUST BUNNIES)** 	Aggregates of filamentous, branching material with a wooly appearance with club formation at one end & seen in cervical smears in women, infected with Actinomyces israelii.

18	**HEMATOXYLIN BODIES (LE CELLS)** 	Basophilic extracellular aggregations of nuclear materials bound with immunoglobulins & are found in connective tissue diseases such as Systemic lupus erythematosus.
19	**HENDERSON-PATTERSON BODIES** 	Large, ellipsoidal, homogenous, intracytoplasmic proteinaceous inclusions found in the stratum spinosum & stratum corneum of skin infected with Molluscum contagiosum.
20	**KAMINO BODIES** 	Eosinophilic, red, globules with scalloped borders & crescent-shaped periphery present in the basal layer above the tips of dermal papillae. Found in Spitz nevus & Pigmented spindle cell nevus of Reed. The main content is type IV and type VII collagen which usually stain with PAS and Masson's trichrome stains.
21	**LAFORA BODIES** 	Intracytoplasmic, basophilic and metachromatic, PAS-positive & diastase-resistant, concentric target-like laminated inclusions found in the skin (excretory sweat ducts), neurons, muscle cells, & hepatocytes in patients with Lafora body disease.
22	**LEISHMAN-DONOVAN (LD) BODIES** 	Light blue, ellipsoid bodies, 2–4 µm long, with an eccentric nucleus & a smaller kinetosome at the opposite pole within large macrophages (Wright's cells), seen in Leishmaniasis.
23	**LIPSCHUTZ BODIES** 	Eosinophilic intranuclear inclusions with enlarged nuclei & clear halo in Herpes simplex infection.

24	**MICHAELIS-GUTMANN BODIES** 	Round cytoplasmic inclusions of 5–15 µ size with lamellar appearance that stain positively with PAS, Von kossa stain & Perl's ferrocyanide reaction. They are considered pathognomonic for Malakoplakia & are thought to represent the abnormal degradation of bacteria, with calcium & iron deposited on the remaining glycolipids.
25	**ODLAND/LAMELLAR BODIES** 	Membrane coating granules, of size 100-300 nm in diameter & are found within the cytoplasm of cells of the upper spinous layer & granular cell layer. By discharging their lipid components into the intercellular space, they play an important role in barrier function & intercellular cohesion within the stratum corneum.
26	**PAPILLARY MESENCHYMAL BODIES** 	Distinct fibroblastic aggregations around the basaloid islands representing the abortive attempts to form the mesenchyme of the dermal papilla around the hair bulb responsible for hair induction. These bodies are generally associated with Trichoepithelioma & Trichoblastoma & helpful to differentiate these tumors from Basal cell carcinomas.
27	**PSAMMOMA BODIES (SAND BODIES OR CORPORA ARENACEA)** 	Seen in Cystadenocarcinoma of the ovary, Papillary carcinoma of the thyroid, Meningiomas, Psammomatoid melanotic schwannoma, & Juvenile ossifying fibroma. On H & E stain, these appear as spherical concentrically laminated masses of gritty, calcified materials.
28	**PUSTULO-OVOID BODIES OF MILIAN** 	Accumulation of lysosomal granules appearing as large, round eosinophilic granules surrounded by a clear halo & found in Granular cell tumors.

29	**RUSSELL BODIES** 	Eosinophilic, large, homogenous immunoglobulin inclusions measuring 20–40 µm within plasma cells. They are seen in multiple myeloma, Helicobacter pylori infection, Periapical granuloma & Plasmacytoma.
30	**SCHAUMANN (CONCHOID BODIES)** 	Basophilic concentric lamellar structures, 100 µm in diameter, composed of lipomucoglycoproteins impregnated with calcium & iron, & they show central, bi-refringent crystals, seen in Sarcoidosis.
31	**SCLEROTIC BODIES (MEDLAR BODIES OR COPPER PENNIES)** 	Rounded cells with thick walls, found in chromoblastomycosis. In another condition Nephrogenic systemic fibrosis, sclerotic bodies consisting of elastic fibers surrounded by coarse collagen can be found in thickened & hyperpigmented skin.
32	**VATER-PACINI BODIES (PACINIAN CORPUSCLES)** 	Major mechanoreceptors, sensitive to vibration & pressure found in the skin of the fingers & formed by concentric layers of connective tissue with a central soft core in which the nerves are located.
33	**VEROCAY BODIES** 	Stacked arrangement of rows of elongated palisading nuclei alternating with acellular zones (made up of cytoplasmic processes of the Schwann cells & reticular fibres in between) found in Schwannomas. Such an arrangement of nuclei is probably due to the over-expression of laminins in the cells in an adaptive response to maintain cell to cell interaction.
34	**WAGNER-MEISSNER BODIES** 	Diffuse proliferation of nerve bundles in tactile corpuscles like pattern in the dermis, containing eosinophilic globules with parallel slits, typically seen in Neurofibroma.

35	**WINCKLER BODIES** 	Spherical structures found in skin lesions in patients with Syphilis.
36	**ZEBRA BODIES** 	Classically seen in mucopolysaccharidoses (e.g., Hurler syndrome) & lysosomal storage disorders such as Fabry's disease, Niemann-Pick disease, Sandhoff's disease, & Tay-Sachs disease. Characteristically, these bodies appear as metachromatically staining membrane-bound lamellated granules with a 5.8 nm spacing in neurons, schwann cells, macrophages, smooth muscle cells, endothelial cells, pericytes, glomerular epithelial cells, and hepatocytes.

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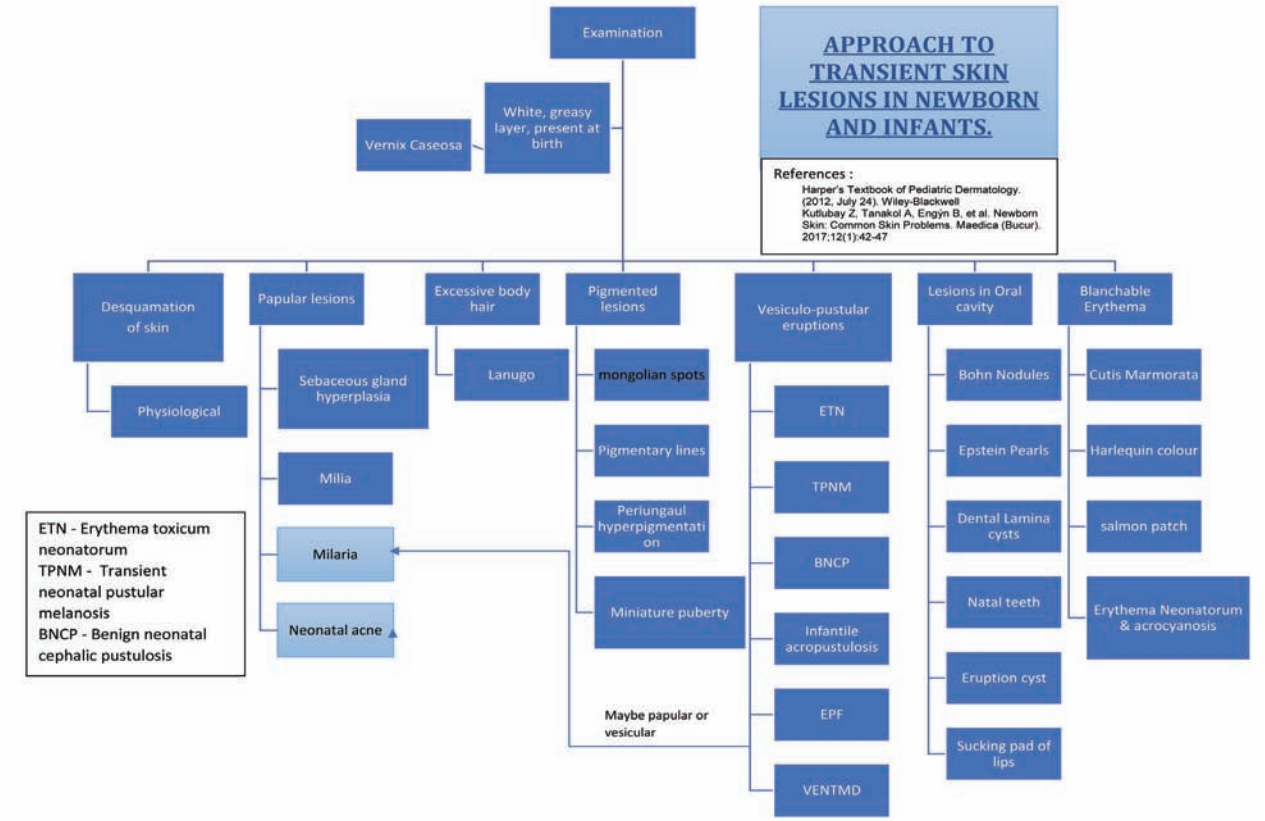
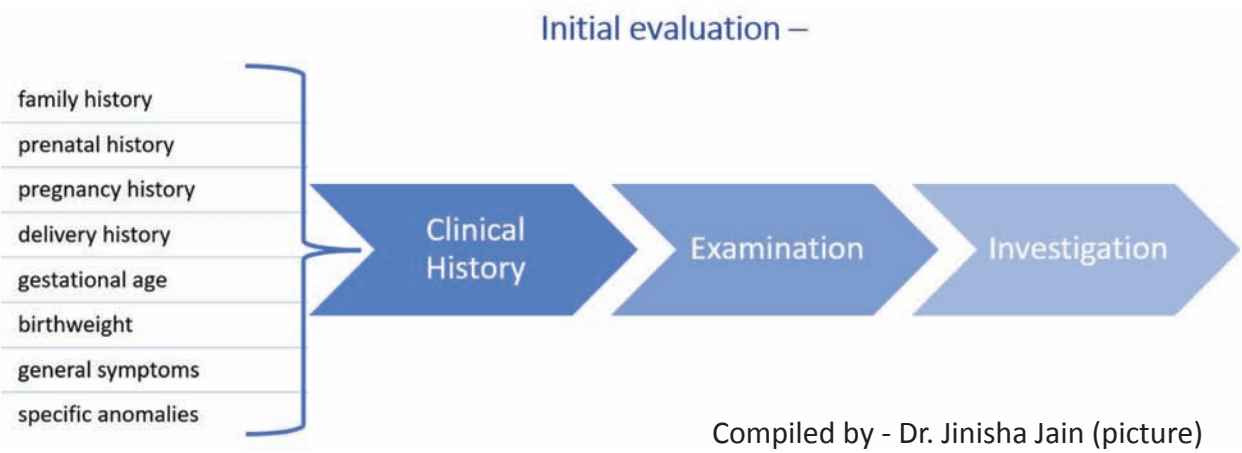
3rd year PG
SNMC, Bagalkot

APPROACH TO TRANSIENT SKIN LESIONS IN NEWBORN AND INFANTS

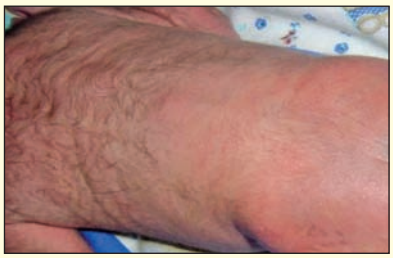
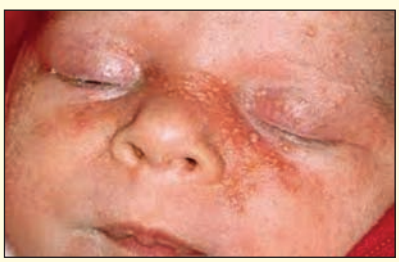


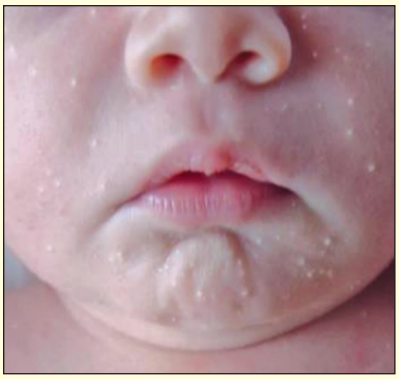
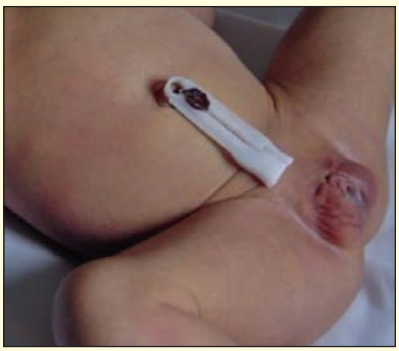
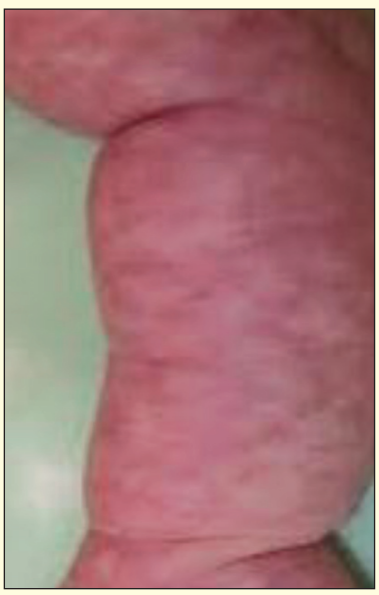
Skin lesions in first year of life are common, usually benign, physiological and transient. It is essential to differentiate them from serious life-threatening conditions which increase morbidity and mortality notably.

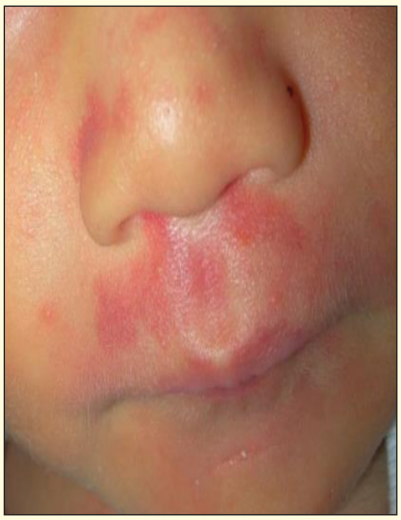
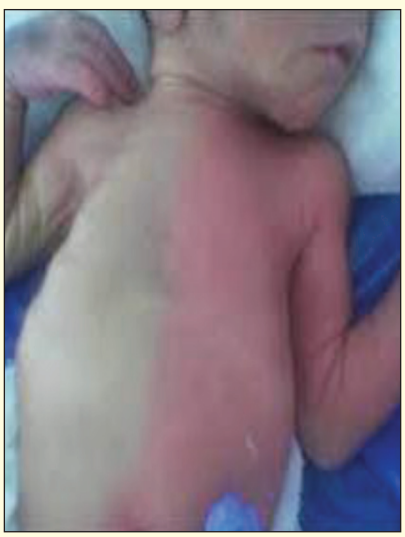
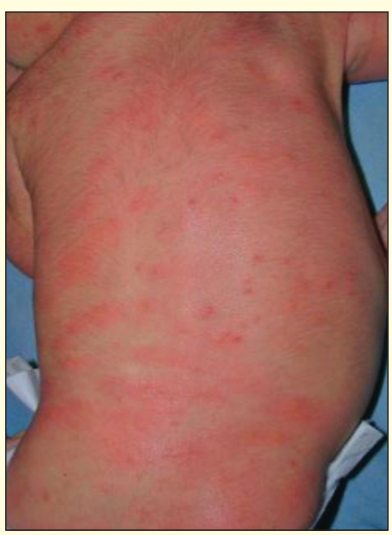
Here we will be discussing an approach to transient skin lesions in new-borns & infants and their differentials.

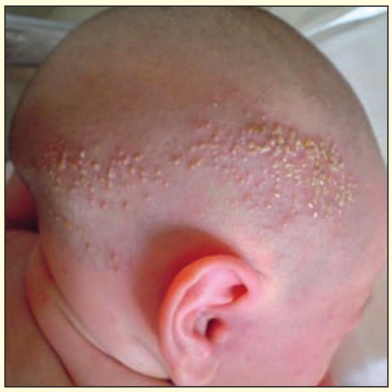
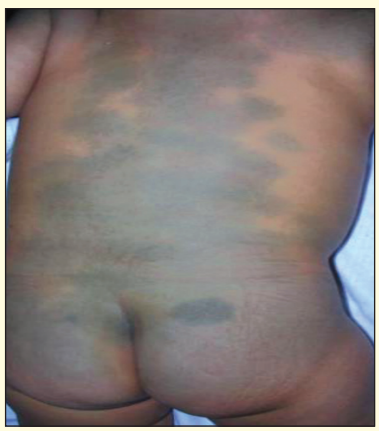
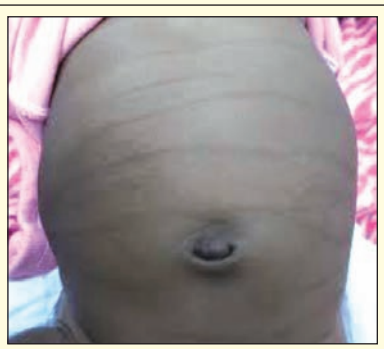


Compiled by - Dr. Jinisha Jain (picture)

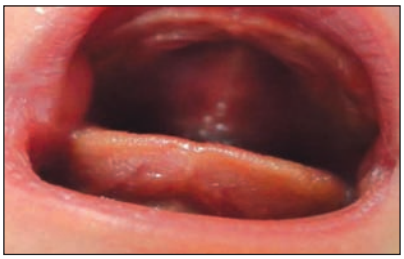
SKIN LESION	CLINICAL FEATURES	
Vernix Caseosa	White, greasy material which may cover the entire cutaneous surface of full-term babies or may be found concentrated in skin folds and on the back. After a few hours or days of life, it disappears. Vernix caseosa may be brownish-yellow if there was contact with meconium or as might be seen in cases of haemolytic disease. Vernix caseosa may have a noticeable typical odour in a case of neonatal sepsis.	
Lanugo	Newborn skin is often covered by fine, soft and nonpigmented immature hair termed lanugo. Lanugo is often located on the back, shoulders, face and scalp. Lanugo must be differentiated from congenital hypertrichosis lanuginosa, gingival fibromatosis with hypertrichosis, Cornelia de Lange syndrome and other rare disorders associated with excess body hair.	
Physiological desquamation	A fine desquamation occurs in newborns which may persist till the first 3 months of life. In full-term neonates this appears on the first or second day of life and in preterm infants it starts at 2–3 weeks of age. Desquamation is more evident on hands, feet and ankles. In post-term newborns it tends to be more generalized and thicker. Extreme cases when desquamation is persistent or severe congenital syphilis or ichthyosis should be evaluated.	
Sebaceous gland hyperplasia	Characterized by multiple tiny whitish- yellow papules located at the opening of pilosebaceous follicles in areas in which sebaceous glands are prominent such as around the nose, cheeks, forehead and upper lip. Differential diagnosis includes milia, miliaria cristallina and neonatal acne.	
Physiological jaundice	It is common and harmless jaundice seen in babies during the first days of life, with no significant underlying disease. It is common in the first week, affecting 60% of term and 80% of preterm newborns. Early onset & prolonged-mention duration jaundice may be a sign of pathological jaundice.	

Milia	Multiple superficial pinpoint white- yellowish papules 1–2mm on face, nose, cheeks, forehead and chin. Scalp, upper trunk and upper limbs may also be affected. May be single or multiple lesions. A solitary and usually larger milium may be present on the areola, genitalia or foreskin. Disappear spontaneously, usually within a few weeks of life without scarring, although they may persist for several months. In cases where profuse and persistent milia are noted or associated with other anomalies, an underlying genodermatosis must be suspected.	
Miniature puberty	Darkening of the linea alba, areolas and external genitalia, breast hypertrophy may occur in both sexes, as may neonatal acne and sebaceous gland hyperplasia. The enlarged breast gland may secrete a colostrum-like substance. Male genitalia may appear well-developed with hyperpigmentation of scrotum. Female genitalia may appear full with clitoral hypertrophy and darkening of the labia and vulva. Within a few days after birth, a whitish, creamy vaginal discharge can be seen. Rarely, on the third to fourth day of life, bleeding similar to menses may occur.	
Cutis marmorata alba	It is characterized by a transient, blanchable, bluish-red, reticulated mottling that in general lasts minutes, aggravated by cold and abated by warm temperatures. Physiological cutis marmorata improves spontaneously within the first few weeks. Persistent or severe cutis marmorata has been reported in several diseases including Down syndrome, trisomy 18, congenital hypothyroidism, homocystinuria, Divry–Van Bogaert syndrome and Cornelia de Lange syndrome. Frequently segmental or localized. Cutis marmorata telangiectatica congenita is a relatively uncommon capillary malformation that can mimic physiological cutis marmorata but does not disappear with warming.	

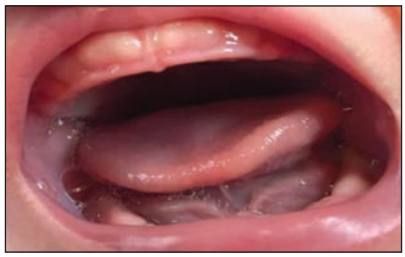
Salmon patch (Aka Stork bite & angel kiss)	It involves the median part of the face (glabella, forehead, eyelids, nasal alae and philtrum), the nape and occipital area and in some cases the sacral region Its colour typically varies from pale pink to bright red with undefined borders that become more prominent with heat, crying and physical activity. Facial lesions usually fade within the first 2 years of life. Lesions involving the nape may persist Patients with lumbosacral naevus simplex should have imaging performed to rule out underlying spinal dysraphism if the naevus is extensive or atypical, or other cutaneous anomalies such as lipoma, hypertrichosis or dermal sinus, among others, are also present.	
Harlequin color change	Consists of sharply demarcated erythema on one half of the body with simultaneous blanching on the contralateral half. A line of demarcation along the midline is well- defined. The change of colour, which generally fades within 30 seconds to 20 minutes, appears between the second and fifth days after birth, but may occur up to the third week. Harlequin colour change may arise as a single episode, it sometimes reappears. It is most commonly observed when the baby is in a lateral decubitus position. The colour characteristically changes location when the baby is rotated. Although it is observed in healthy newborns, it has also been reported in the context of hypoxia, such as prematurity or low birthweight. It disappears on crying or muscular activity.	
Erythema toxicum neonatorum	Although this rash may be present at birth, it usually begins 2–3 days after birth. Typical lesions consist of erythematous macules 2–3 cm in diameter with a central papule or pustule. Lesions can vary in number from one to several and be located on the face, trunk and proximal extremities, without involvement of palms and soles. The lesions are highly evanescent and usually evolve in crops that wax and wane, with spontaneous resolution of individual lesions within hours to days. Differential diagnosis includes transient neonatal pustular melanosis, miliaria and eosinophilic pustular folliculitis, congenital candidiasis, staphylococcal impetigo and neonatal herpes simplex.	

Transient neonatal pustular melanosis	It is characterized by fragile pustules or vesicles without surrounding erythema that are almost always present at birth. Lesions measure 1–5mm in diameter, are single or grouped in clusters, and may occur anywhere but are commonly seen on the forehead, mandibular area, neck, trunk, buttocks, thighs, palms and soles The vesicopustules are very fragile and break down easily, leaving pigmented macules surrounded by fine, white collarettes of scale. Brownish macules may persist for months, but in general fade spontaneously within a few weeks. The differential diagnosis includes erythema toxicum neonatorum, staphylococcal pustulosis, congenital candidiasis, syphilis, herpes simplex and acropustulosis of infancy.	
Mongolian spots	Characterized by single or multiple grey, blue-grey, blue-green or dark blue macules/ patches with irregular borders, located mainly on the sacrogluteal area. Extrasacral locations include the lower and upper extremities, upper back and shoulders. They typically fade in early to mid-childhood. Associated lysosomal storage disease associated with Mongolian spots is GM1 gangliosidosis 1, Hurler disease, Hunter disease, mucolipidosis, Niemann - Pick disease and mannosidosis.	
Pigmentary lines of newborns	Characterized by horizontal linear hyperpigmentation on the skin folds of the abdomen, back and extremities from birth. Spontaneous resolution occurs between 2 and 6 months of age.	
Periungual hyperpigmentation	Hyperpigmentation of the distal phalanges and the proximal nail fold.	

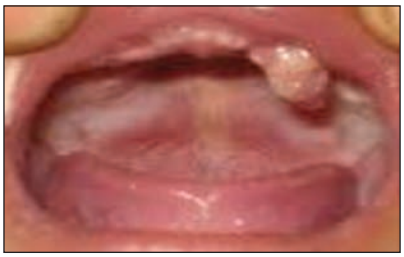
ORAL LESIONS



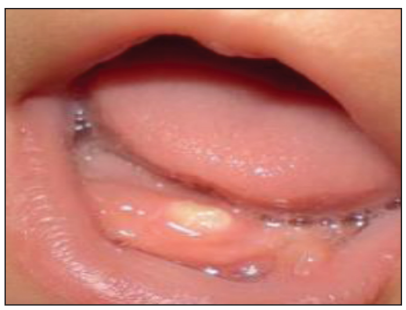
Epstein pearls are single or multiple, 1–3mm, whitish keratin-filled cysts, at the junction of hard and soft palates or at the palatal midline



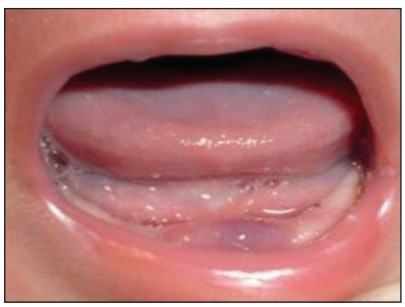
Bohn nodules are firm white cystic structures on vestibular and lingual surface of alveolar ridges. Found more on Maxillary arch.



Dental lamina cysts are small (1–3mm), pearly, firm papules on crest of alveolar ridges, may be single or multiple, located mainly on the maxilla.

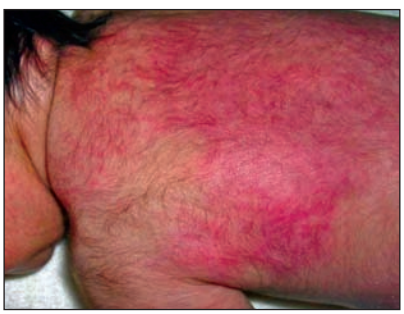


Natal and neonatal tooth

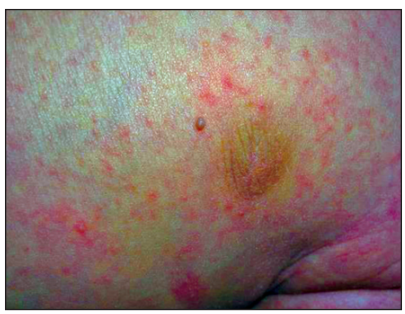


Eruption cyst is a translucent, flesh-coloured or bluish, dome-shaped, compressible lesion on the alveolar ridge of the mandible or maxilla.

MISCELLANEOUS



Subcutaneous fat necrosis - multiple indurated, erythematous or violaceous painful plaques and nodules located on the back, shoulders, extremities, buttocks and cheeks. Some lesions may become fluctuant with drainage of liquefied fat from nodules. Lesions resolve spontaneously within a few weeks, leaving on rare occasions atrophy, fibrosis, scarring, ulcers or necrosis.



Adnexal polyp of neonatal skin is a solitary, small, congenital polypoid tumour occurring most frequently on the areola of the nipple. It may also develop in other sites such as eyelid, cheek, scapula, arm, axilla, labia majora and scrotum.

Pedal papules of infancy are present at birth and consist of bilateral, symmetrical, asymptomatic, subcutaneous flesh-coloured nodules located on the plantar region of the heel.

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KAPOSI'S VARICELLIFORM ERUPTION IN SLE

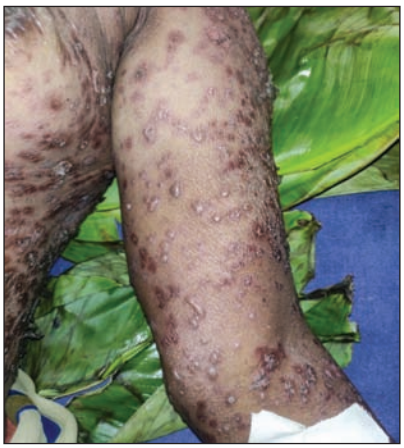
Kaposi's varicelliform eruption was first described by Moritz Kaposi in 1884 but the viral etiology was confirmed by Freund when he demonstrated cytoplasmic inclusions in the 20th century.[4] It is caused by viruses, most commonly herpes simplex virus 1 and 2, others being Vaccinia, Varicella zoster, Molluscum virus, Coxsackie A16.[5] Transmission occurs through contact with infected person or by dissemination of primary or recurrent herpes.[6]

ABSTRACT : Kaposi's Varicelliform Eruption (KVE) is a disseminated skin infection caused by herpes simplex virus in patients with underlying cutaneous disease. We report a case of a forty-five year old woman, a known case of systemic lupus erythematosus (SLE) who presented with herpes labialis and widespread vesicular eruption suggestive of KVE. Diagnosis was made based on clinical features. Patient responded well to intravenous acyclovir therapy. KEYWORDS : Kaposi's varicelliform eruption, SLE, Acyclovir

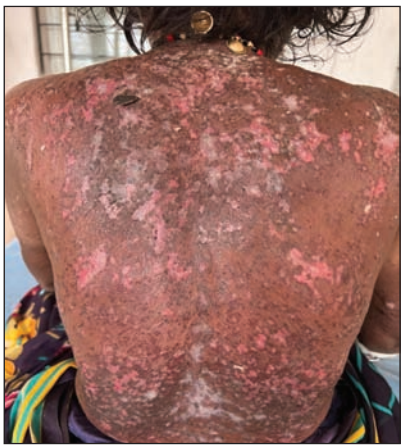
INTRODUCTION : Kaposi's varicelliform eruption is a cutaneous manifestation of disseminated viral infection in patients with pre existing dermatoses. Most commonly associated condition is atopic dermatitis, which gives it the name 'Eczema herpeticum'.[1] Clinically it presents with vesicular eruptions localised over the areas with pre existing cutaneous pathology and with a predilection for upper body and head.[2] Diagnosis is made primarily on clinical findings. Antiviral therapy is effective and should be started promptly to reduce morbidity and mortality.[3]

CASE REPORT : A forty-five year old female, a known case of SLE, on treatment since 1yr presented to the dermatology department with fever, insomnia, burning sensation and sudden eruption of fluid filled lesions over the body. The patient was apparently normal 5 days back after which she developed fluid filled lesions, initially beginning over the chest and progressed cephalocaudally to involve the back, abdomen, arms and forearms over the next two days. The lesions were associated with burning sensation and pain. Patient had a past history of recurrent herpetic gingivostomatitis.

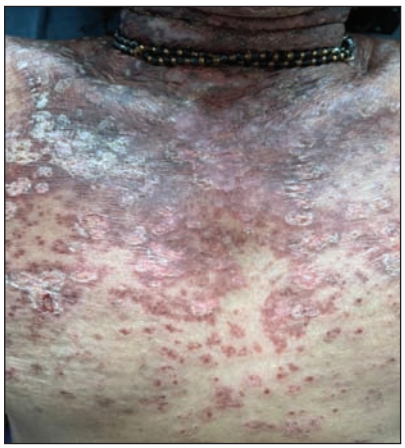
On examination there were bilaterally symmetrical grouped vesicles, many of which were umbilicated over the back, chest, abdomen, arms and forearm. There were multiple petechiae, purpura over the abdomen, lower limbs, palms and soles and non scarring alopecia over the scalp. There was crusting over the lower lip. In due course, the vesicles spontaneously evolved into widespread punched out erosions with serosanguineous discharge and crusting. The lesions were unresponsive to ongoing



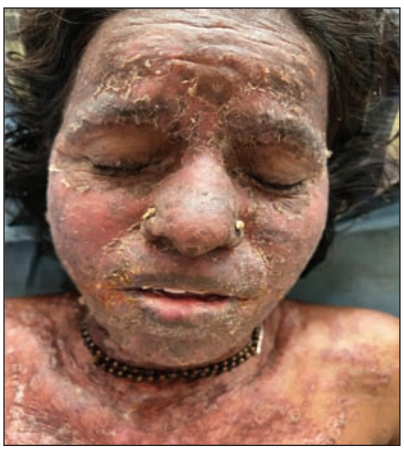
Multiple vesicles, few umbilicated over the chest, arms.



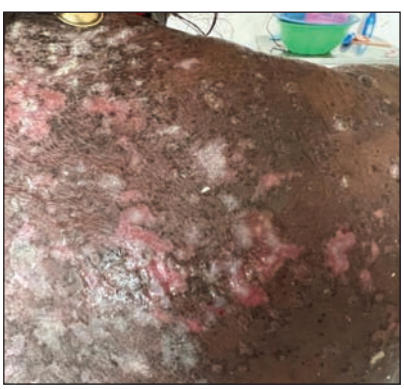
Widespread erosions with crusting over the back



Erosions and purpuric macules over neck and chest



Healing erosions over lower lip and erythema with desquamation and scaling over face



Close up view of the erosion with serous discharge and minimal crusting

treatment for SLE, i.e. steroids, hydroxychloroquine and dapsone. Blood counts revealed anemia (Hb-10.6g/dl), leukocytosis (TLC-14,200 cells/cu. mm) with neutrophilia 80%. Tzanck smear showed few lymphocytes but multinucleated giant cells could not be isolated. Viral culture and PCR could not be done due to the nonavailability of the tests in our institution. Based on the clinical findings, a diagnosis of recurrent herpes labialis with kaposi's varicelliform eruption was made. The patient was started on iv acyclovir 10 mg/kg tid for 7 days, following which the general condition of the patient improved and the skin and oral lesions subsided in 10 days.

DISCUSSION : Kaposi's varicelliform eruption was first described by Moritz Kaposi in 1884 but the viral etiology was confirmed by Freund when he demonstrated cytoplasmic inclusions in the 20th century.[4] It is caused by viruses, most commonly herpes simplex virus 1 and 2, others being Vaccinia, Varicella zoster, Molluscum virus, Coxsackie A16.[5] Transmission occurs through contact with infected person or by dissemination of primary or recurrent herpes.[6]

Atopic dermatitis is the most common underlying cutaneous pathology, others being pemphigus foliaceus, ichthyosis vulgaris, bullous pemphigoid, Hailey-Hailey disease, mycosis fungoides, psoriasis, pityriasis rubra pilaris, seborrheic dermatitis, contact dermatitis, burns, skin grafting, tinea etc.[6]

Damaged cutaneous barrier and altered immune response

are the predisposing factors for development of KVE. Possible mechanisms for KVE in SLE could be the reduced peripheral blood T cells, defective Th1 helper cell function and shifting towards the Th2 cytokine profile in SLE7 (similar to atopic dermatitis) which might favour viral infection and its dissemination. Immunosuppression due to the drugs, pre existing photosensitive rash could have been the other predisposing factors in this case.

Although rare, it is a potentially life threatening infection. Its presentation may vary from mild signs and symptoms to fulminant disease complicated by multiple organ involvement such as liver, lungs, brain, gastrointestinal tract, adrenal glands and eyes.6 The topography and distribution may delay the diagnosis as the eruption is often confused with the pre existing dermatosis. It is diagnosed primarily based on clinical features. Tzanck smear, viral cultures, Polymerase chain reaction can be helpful in doubtful cases.[3]

Treatment should be instituted promptly. Depending on the severity, oral acyclovir may be started 2 or in some cases high dose intravenous acyclovir may be necessary for disease control. Antibiotics are recommended as prophylaxis from secondary bacterial infection.[3]

CONCLUSION : An apparently mild viral infection can become disseminated in patients with pre existing disorders. Kaposi's varicelliform eruption should be suspected whenever vesicles develop suddenly over underlying dermatoses. Our case adds to the growing list of conditions that may be complicated by this eruption.

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YUVADERMA
E-Bulletin

A NOVEL USE OF TIMOLOL MALEATE EYE DROPS

A CASE REPORT

INTRODUCTION

- Angiokeratomas are well-circumscribed vascular lesions, a group of telengiectasias with prominent epidermal response.
- Clinically described as well-defined hyperkeratotic, red-to-black papules or plaques, and histologically appear as dilated blood vessels in the papillary dermis that form lacunae with associated acanthosis and rete ridge elongation.
- Multiple variants of angiokeratomas exist:

1. Angiokeratoma of Fordyce (located on the genitalia, including the scrotum)
2. Angiokeratoma corporis diffusum (associated with Fabry disease),
3. Angiokeratoma of Mibelli (located on acral sites)
4. Solitary or multiple angiokeratomas (often located on lower extremities) and
5. Angiokeratoma circumscriptum (congenital and occurs on the trunk and extremities).

- While angiokeratomas are typically asymptomatic, they can bleed, cause pruritus or pain, and impact psychological health, ultimately driving a patient to seek care.

CASE REPORT

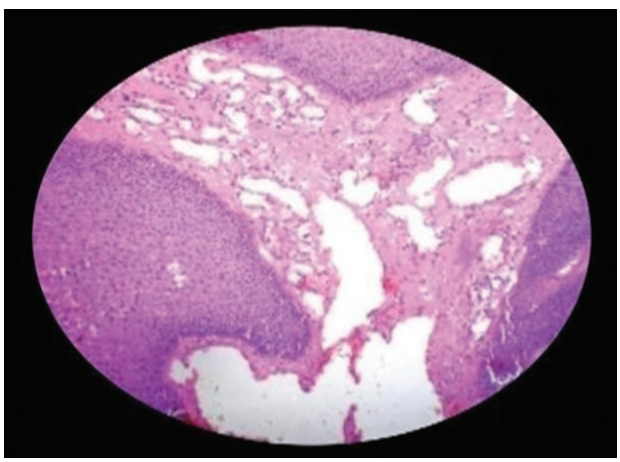
A 38-year-old male presented with multiple asymptomatic dark lesions over the scrotum since 3 months.

Patient noticed the lesions 3 months ago which presented as dark lesions over the scrotum initially few in number, within a few months the number increased gradually. No increase in size of the lesions. Not associated with pain or itching. No h/o bleeding on touch.

On examination, multiple (>20) grouped hyper pigmented (blue-black), hyperkeratotic papules firm in consistency of 2mm-4mm over the scrotum sparing the shaft of penis, inner thigh and abdomen. No active bleeding or urethral discharge.

A differential diagnosis of Angiokeratoma of Scrotum(Fordyce), Condyloma acuminate, Lymphangioma circumscriptum, Cherry angioma were made.

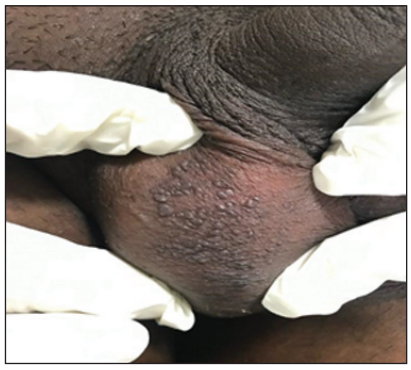
Routine investigations were normal; USG Abdomen & Pelvis was normal. Histopathology showed Epidermal hyperkeratosis,dilated congested thin-walled capillaries in the papillary dermis, acanthosis and papillomatosis confirming the diagnosis of Angiokeratoma of fordyce



TREATMENT

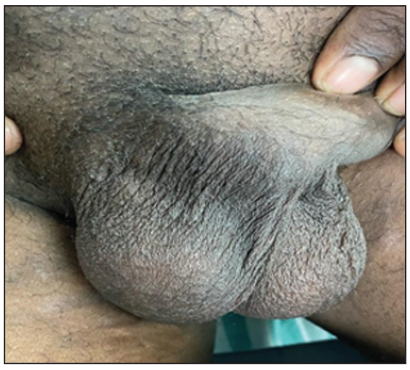
- The patient was unaffordable and expressed preference for topical treatment. Thus, he was started on off-label topical application of Timolol maleate 0.5% drops.
- The frequency of application was two drops twice daily for 1 month over the affected areas.
- Apart from initial transient mild itching at the treatment site lasting for approximately

10 minutes post-application, the medication was well-tolerated, and this transient itching sensation no longer occurred with continued use.



Pre-treatment – Angiokeratoma of scrotum.

- Patient had complete remission in a month and was followed up after 3 months with no new complaints.



Post treatment – Timolol drops twice daily for 1 month

DISCUSSION

- Timolol is a non-selective beta adrenergic blocker.
- Beta-adrenergic receptors present on vascular endothelial cells mediate peripheral vasodilation, activation of proangiogenic factors such as vascular endothelial growth factor and basic fibroblast growth factor, and inhibition of endothelial cell apoptosis.
- It is hypothesized that vasoconstriction of blood vessels within the lesion leads to size reduction as noted in infantile hemangiomas.
- Longer treatment may lead to regression, presumably because of inhibitory effects on vascular growth factors and promotion of apoptosis.

However, it must be noted that beta-adrenergic receptors are only weakly expressed in angiokeratoma of scrotum and in only 50% of them, as opposed to their strong expression in infantile hemangiomas.

CONCLUSION

- The reduction of vascular ectasias may be due to decreased blood supply to the capillaries resulting from interference with the active transport system or interference with prostaglandin biosynthesis.
- To the best of our knowledge, This is the first report describing topical timolol maleate 0.5% drops for use in treating angiokeratomas of fordyce
- Given the minimal side effect profile and the cost of therapy , timolol maleate 0.5% drops can be a promising alternative treatment for patients with angiokeratoma of fordyce who are unable to tolerate other therapeutic options.

Further studies are required to evaluate the true efficacy and safety, including the consequences of long-term use.

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BART'S SYNDROME

A RARE CASE REPORT

Introduction - Bart's syndrome, also known as Aplasia Cutis Congenita (ACC) type 6, is an exceedingly rare genetic mechano-bullous disorder characterised by congenital localized absence of skin, muco-cutaneous blistering lesions and nail abnormalities. [1] It follows Autosomal Dominant inheritance. Several sporadic cases also have been reported. The lesions are mainly unilateral, which involve the medial and/or dorsal surface of limbs, as sharply demarcated, glistening red ulcerations, extend upward from foot to the shin. [1]

Case description - A 3 days old male baby, born to 2nd degree consanguineous marriage, was brought to Dermatology OPD with multiple fluid filled lesions and wounds over the body since birth. No similar history in family members. On cutaneous examination, well demarcated, localised absence of the skin with bright red erythema and ulcerations, covered with slough and crusting noted over the antero-medial aspect of the bilateral feet, legs, extending upto left knee. Few tense bullae of varying sizes with superficial erosions seen over back, buttocks, dorsum of bilateral forearms and wrists. Superficial erosions noted on oral mucosa and lips. Anonychia present on medial two toes bilaterally in areas of involvement of the skin. Systemic examination and routine haematological investigations were within normal limits.

Tzanck smear from the base of the erosions showed few inflammatory cells. Patient attenders refused to give consent for skin biopsy. Clinical diagnosis of Bart's syndrome was made on the basis of localised absence of skin, spontaneous muco-cutaneous blisters and nail abnormalities and was managed with systemic antibiotics, topical fusidic acid cream and non-adhesive dressings. Baby succumbed to the disease due to sepsis on the 10th day of life.

Discussion - Aplasia Cutis Congenita is a group of heterogeneous diseases representing failure of the skin to fully develop. Frieden created a classification system for ACC consisting of nine groups based on the number and location of the lesions and the presence or absence of associated malformations. Group 6 of ACC is a genodermatoses clinically characterised by triad of congenital localised absence of the skin over lower extremities, any type of Epidermolysis Bullosa and nail abnormalities. [1] This triad is also called "Bart's syndrome", which was first described by Bruce J. Bart in 1966 in a family consisting of 26 members with these clinical features. [2]

The inheritance is autosomal dominant with complete penetrance but sporadic cases have been reported. Zelickson et al has performed genetic linkage analysis of the descendants of the original family, mapping the gene to chromosome 3p at or close to the site of the

gene encoding type VII collagen (COL7A1). Even though various hypotheses have been proposed, exact pathophysiology of ACC is not known. Proposed mechanisms include intrauterine vascular compromise, trauma, infections and medications. [3]

Diagnosis is usually clinical. Skin biopsy and genetic study help to confirm the final diagnosis. Histopathological examination of a fresh blister shows subepidermal blister formation (below the lamina densa) with poorly formed anchoring fibrils which are reduced in number, which is consistent with Epidermolysis Bullosa. Uninvolved skin has normal morphological appearance. [3]

More severe cases present with other congenital anomalies like pyloric atresia, ureteric stenosis, renal abnormalities, rudimentary ear development, flattened nose, broad nasal root and wide-set eyes.

Management is generally conservative including wound care, allowing the affected area to declare itself in order to optimise future reconstruction, control of infection, prevention and treatment of complications. Kuvat and Bozkurt discussed in detail conservative treatment for patients with Bart syndrome. They used daily hydrodebridement with 1/200 diluted povidone-iodine (100 mL povidone iodine/20 L of boiled water) and fusidic acid cream; the wound was closed with dexpanthenol plus chlorhexidine-impregnated sterile gauze bandages. These conservative methods led to rapid epithelialization. [1] Overall prognosis is good with normal life expectancy. [3]

Conflicts of interest – Nil.

Figure 1 : Clinical images of case.

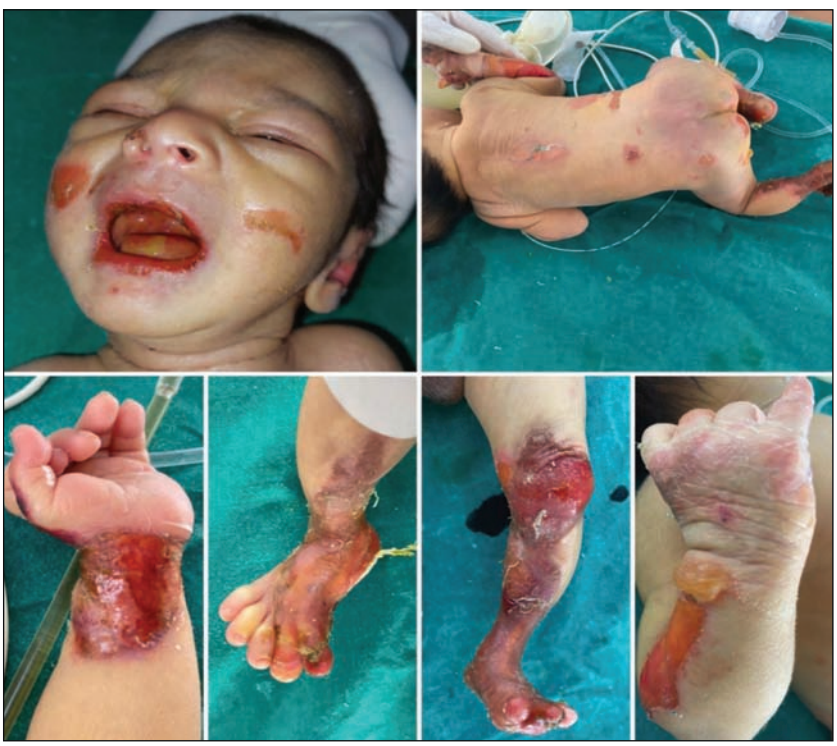

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Figure 2 : Classification of Aplasia Cutis Congenita adapted from Friedens classification.

Group	Definition	Inheritance
1	Scalp ACC without multiple anomalies	Autosomal dominant or sporadic
2	Scalp ACC with associated limb abnormalities (limb reduction abnormalities; syndactyly; club-foot; nail absence or dystrophy; skin tags on toes)	Autosomal dominant
3.	Scalp ACC with associated epidermal and organoid nevi	Sporadic
4.	ACC overlying embryologic malformations such as meningomyelocele, gastroschisis, and omphalocele	Depends on the underlying condition
5.	ACC with associated fetal papyraceous or placental infarcts	Sporadic
6.	ACC associated with epidermolysis bullosa (EB)	Depends on EB type: It may be autosomal dominant or recessive
7.	ACC localized to extremities without blistering	Autosomal dominant or recessive
8.	ACC caused by specific teratogens (methimazole, varicella and herpes simplex infection)	Not inherited
9.	ACC associated with malformation syndromes (Trisomy 13; 4p- syndrome; many ectodermal dysplasias; Johanson-Blizzard syndrome; focal dermal hypoplasia; amniotic band disruption complex; XY gonadal dysgenesis)	Various, depending on the specific syndrome

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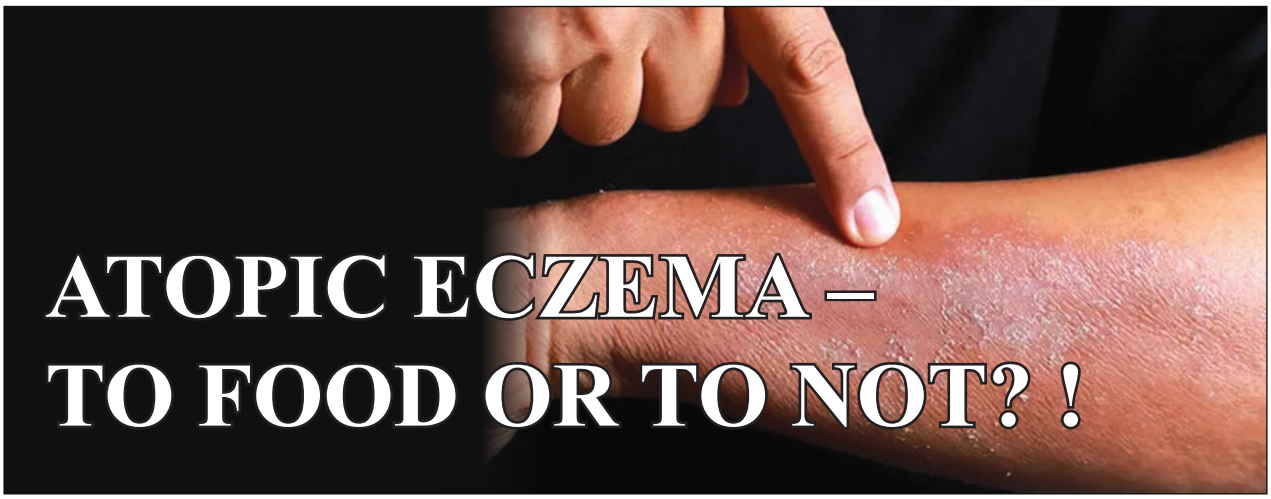

ATOPIC ECZEMA – TO FOOD OR TO NOT? !

Food allergy is basically "adverse health effect arising from a specific immune response that occurs reproducibly on exposure to a given food." Certain foods may trigger rapid, immunoglobulin E-mediated hypersensitivity reactions or may lead to late eczematous reactions.

Post graduation is a beautiful time when we explore the beauty of the subject, learn the practical aspects, learn to make informed decisions backed by our professors and seniors, counsel patients and assess treatment compliance of patients.

A 3 year old girl was brought to our OPD and it wasn't a herculean task to diagnose her as a case of atopic dermatitis. We started her on the required medications and counselled the father who was clearly worried about the prognosis and his face resonated with lingering thoughts of "how long?" side effects?" "What more?" Sensing this we gave him a thorough counselling regarding AD that included bath time, clothes, importance of moisturization and lastly food.

Call it the indian instinct; food grabbed his attention and I gave him a list of food that have known to trigger the condition. He looked surprised and replied saying most were from those used regularly in their kitchen! I thought on; how to tackle this ? Should we really concern patients and parents with this ? And how much importance should really be given ?

Atopic dermatitis has seen a rising trend as observed in India in last four decades owing to the betterment of the socioeconomic status, increased influx into urban areas, mothers becoming over cautious, increased nuclear families and shift from "time tested food".

With greater studies and evolving knowledge there has been a paradigm shift in our understanding of the pathogenesis of AD,

focus being attributed to barrier abnormalities, rather than immune dysregulation.

"Let food be thy medicine and medicine be thy food"- Hippocrates.

Nevertheless, the statement feels like a double edged sword!

AD and food allergy are clearly correlated and diet is likely to cause an exacerbation among infants or children with moderate-to-severe atopic dermatitis. Food allergies are more likely with earlier onset and increasing severity of AD.

Food allergy is basically "adverse health effect arising from a specific immune response that occurs reproducibly on exposure to a given food." Certain foods may trigger rapid, immunoglobulin E-mediated hypersensitivity reactions or may lead to late eczematous reactions.

Patients with chronic dermatitis, and many parents of children with (AD), are frequently concerned about if diet can either cause or exacerbate dermatitis. This concern has intensified as the prevalence of food allergy has risen in recent decades.

Some of the common Indian foods that seem to have a triggering effect are milk, egg, fish, wheat , soy and peanuts and these comprise the food that are commonly used in an Indian kitchen contributing to calories to a child.

Since malnutrition is rather common in an Indian setting; it is essential to elucidate a strong relationship between atopy and the food causing the allergy. Many food allergies tend to resolve in early childhood, especially with food like milk and egg and hence periodic food tolerability can be checked periodically.

Several tests are available like oral food challenge test and atopy patch test but the gold standard for diagnosis is the double blinded placebo controlled food challenge (DBPCFC) in which the suspect food and placebo are administered in the clinic or hospital setting and can be only if highly suspicious about a particular food. Maintaining a food diary may help in identifying a trigger.

Dietary modification in cases of proven food allergy helps to decrease severity but does not replace the standard topical or systemic treatment of AD.

Hence the idea is that avoidance of a particular food should be advised only after a clear history and diagnosis of food allergy. If there is an improvement of the symptoms during a diagnostic elimination diet, an oral food challenge should be considered and most importantly, if elimination diet is followed, these children should be supplemented with complementary foods to make up calories and nutrients!

Dr Madhurya s
Senior resident

DON'T JUDGE A DRUG BY ITS DRESS

Even the devil was once an angel, they say. There are certain drugs which have been regarded as villains of dermatology because of their horrid adverse effects such as SJS/TEN, erythroderma, DRESS and others. But some of them have another side to them where they have been used as treatment in the same field. Few of them are listed in brief below.

1. CARBAMAZEPINE –
- Post herpetic neuralgia
- Trigeminal neuralgia
- Diabetic neuropathy
- Neuritis and neuropathic pain- leprosy

2. PHENYTOIN –
- Topically as wound healing promoter of various ulcers, stasis, traumatic, diabetic, pressure, diabetic etc.
- Junctional and dystrophic epidermolysis bullosa
- Inflammatory conditions – linear sclero-derma, SLE, DLE, Sjogren's, pachyonychia congenita, lichen plants
- Neuropathic pain – trigeminal neuralgia
- Diabetic neuropathy

3. LITHIUM
- Topically for Seborrheic dermatitis
- Behaviour control in trichotillomania, skin picking disorder

4. SODIUM VALPROATE
- Neuropathic pain
- Inflammatory linear verrucosa epidermal naevus

5. FLUOXETINE
- Acne excoree
- Adjunct to PUVA in psoriasis
- Topically for wound healing

6. CHLORPROMAZINE
- Severe refractory neurodermatitis
- Dermatitis artefacta
- Trichotillomania

7. ALLOPURINOL
- Disseminated granuloma annulare
- Granuloma induced by fillers
- Sarcoidosis
- Psoriasis
- Acquired reactive perforating collagenosis

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A GLEAM OF HOPE?

In the midst of my hazy memories Stands a subtle spectacle
In the dawn like the buzzing bees An unsolved puzzle yet to crackle.

The dazzling dawn breaks Streaks of squidgy clouds amidst The blushing sun wakes,
Unfurling the latch of the night's mist.

Blooms of roses and periwinkles wink Fawning on the flies that hover over
The florid efflorescence of blushing pink After the last Sunday's sleepover.

A mother with her chicks, ferries Their bod to the nest in fine fettle
On the tree topped up with lush cherries With her kith and kin, full fledged to settle.

The two jubilant chicks in the nest they pule With their damp fluffs doused in rain
The momma stretches her floss of love; that out rule Everything; froth, frost, rain or pain.

Making up themselves in the mother's cosy hood Both the nestlings began to squish and squeak For the mother swooped off to fetch some food Into the red tint sky over to the aloof peak.

The forlorn frenzy had driven them mad Lazing and gazing at the floret sky
Awaiting their mother from the morn; so sad Wishing to see her in the blue somewhere high.

But as their jejune eyes reveal Their mom never turned back
A melancholic mystery they have to unveil; The story of the lost mother's hijack…

The terror of these two chicks on losing their mother reminds us of all the innocent little ones who spend their lives deprived of their parents who were killed by greedy humans. This is just my rendition of the ill effects of hunting and poaching.

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COURAGE , HOPE AND MAGIC!

I had a dream once
I have a dream now
Soul searching as they say
Not knowing where to sway
Looking down this big window
I can feel a wide open window inside me
Deluded demanding
Something astonishing
Like waves, they touch me and leave
Like rain they wet me and sweep
So here comes some thing deep
When we think everything is lost ,
Look for that flicker of courage to see
The window which is still open
Look down and deep
That's where the magic is, i believe
It lurks inside you
And make you wonder
How can we have all this thunder
Still can be so vain
Believing in superficial things is pain
So let it go let it flow
Keep this wondering keep this now
Because you beautiful fool
You are the soul
You are the magic
You still have to search for it
Thats very tragic!

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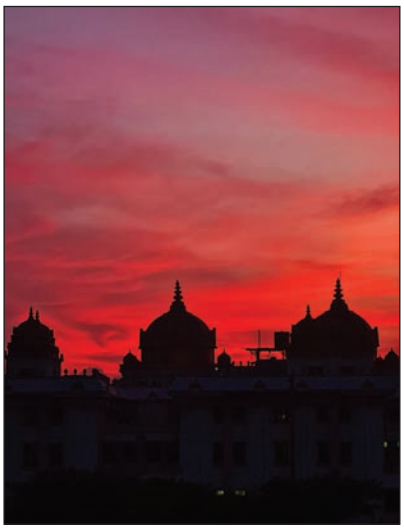
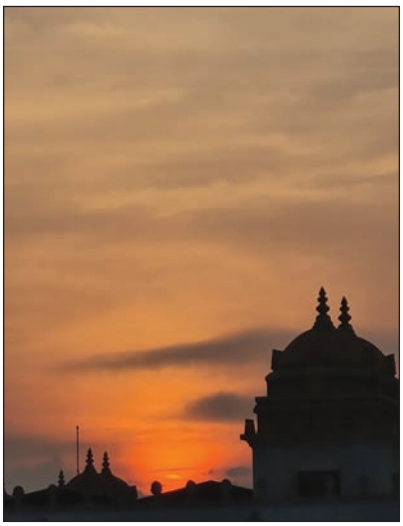


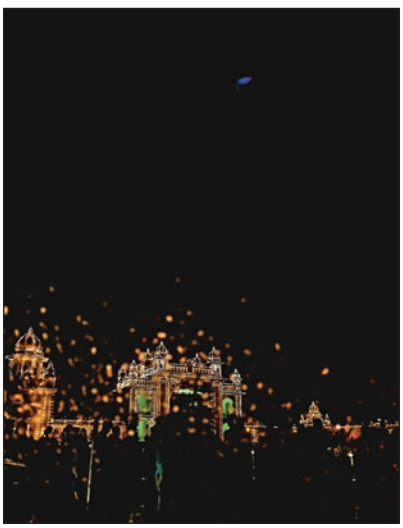
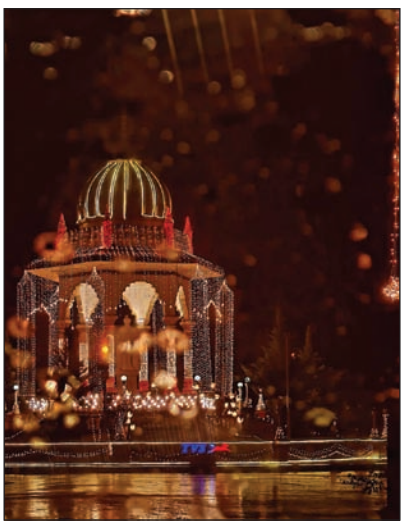


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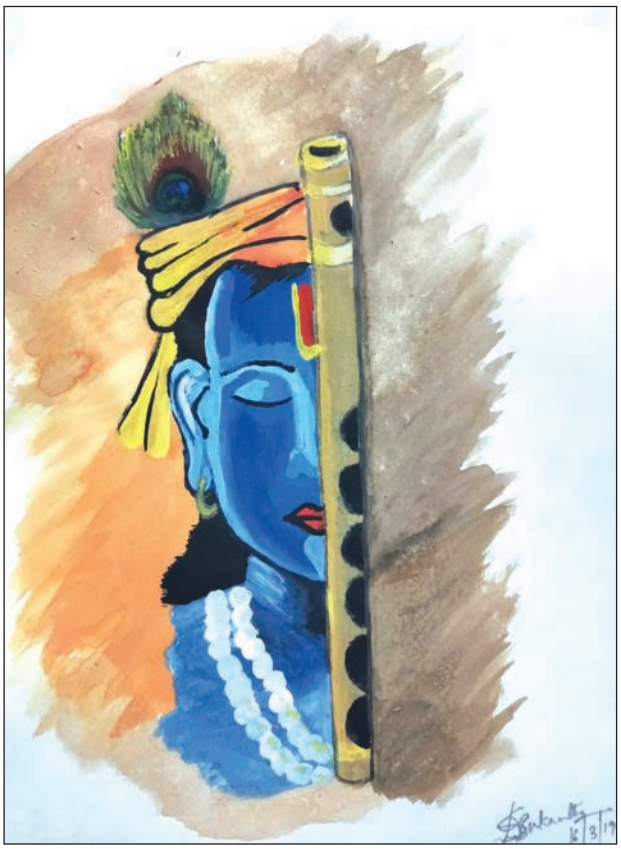

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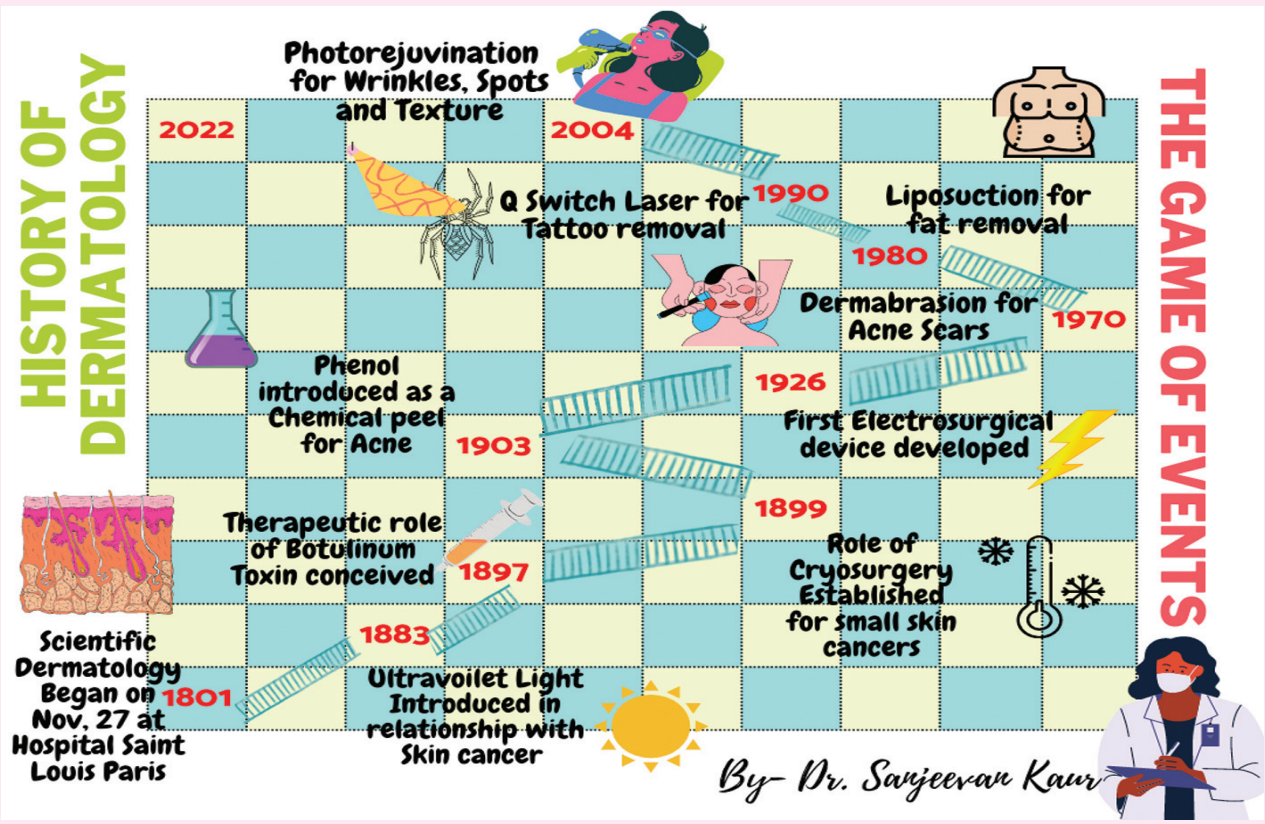

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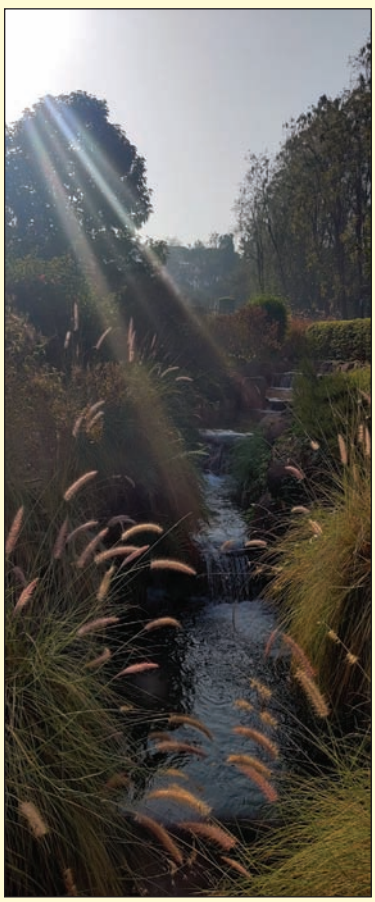

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We hope you have liked this effort of ours.

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Regards,

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