THE ESSENCE

The artwork reflects the essence of Yuvaderma, where young minds are nurtured and moulded into the building blocks of tomorrow.

It represents the journey to the key of enlightenment and success via the road of knowledge, team work, dedication and perseverance.

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IF YOU ARE NOT WILLING TO LEARN,
NO ONE CAN HELP YOU!
IF YOU ARE DETERMINED TO LEARN,
NO ONE CAN STOP YOU!!

Learning is a continuous process and as doctors we should always be ready to adapt and adopt newer things. Being the Editor in chief has made me realize this even more that learning is a treasure that will follow its owner everywhere.

I have been immensely blessed to continue being the Editor in Chief for the 8th edition of Yuvaderma e-bulletin. I would like to extend my gratitude to my mentors and Dr. Saloni Katoch for always guiding me in the right direction. Special thanks to Dr. Preeti Nayak for giving me this opportunity and for her timely advice. Words would fall short in describing my over-zealous team, who take up every task assigned with great responsibility and dedication to fulfill it, I am grateful to have you all, Dr Gagana, Dr Pranami, Dr Priyanka, Dr Sanjay, Dr Shibani and Dr Shilpitha.

We, the editorial team, at the outset take this oppurnity to thank the pillars of our strength Dr Satish Pai, Dr Manjunath R and all present and past respected members of the executive committee for their support and encouragement in all our endeavors.

We begin this issue with a salutation to the Pioneers of dermatology, next loading you up with the knowledge of Hair shaft disorders beautifully written by Dr Priyanka. Interviewing the man of great academic passion and dedication to dermatology, Dr Manjunath Shenoy, has been like an achievement in itself for Dr Shibani, go check out what she unveils. Relax your senses with the
colours of derma art galleria before you head on to the academic outburst with spotters and misnomers in dermatology.

Dermatology lens not only focuses on lesions but can also capture some mesmerizing landscapes. We are proud to have amongst our editorial team Dr Shilpitha, gold medalist RGUHS 2019, give her all your ears as she reveals the exam pearls. With the view of exams we have here, few ready reckoners you should go through to excel in your viva. Lastly we have the brainstorming crossword on misnomers in dermatology.

Happy reading,
Signing off,

Dr. Kirti P Katwe
Editor in chief
Yuvaderma E-bulletin.
PRESIDENTS PREAMBLE

Dear residents,

Very happy to know that the 8th edition of YUVADERMA is being released.

The team has been doing a great job. Congratulations!

I hope all of you know about the Inner circle program an initiative by IADVL Karnataka for residents. We had the first workshop at Father Muller’s Medical College, Mangalore which was well attended and was a great success. We will be having many more in the coming months in different parts of Karnataka. I request all the residents to make use of this program and acquire the necessary soft skills.

Some pearls in clinical Dermatology

1. Each man and woman is ill in his or her own way. You need to recognise their illness.

2. It is very much necessary that you follow a ritual while examining a dermatology patient. Ritual involves examination of entire cutaneous surface, mucosa, nail, scalp, palms and soles. Ritual also involves using a torch and magnifying lens. Rituals are all about transformation. It will help you to discover more about the patient. Ritual is at the heart of patient-physician relationship.

3. Do not short cut clinical examination. You may overlook simple diagnosis.

4. The secret of the care of the patient is in caring for the patient. “Francis W Peabody” (1881-1927)

5. Power of human hand is to touch, to diagnose, to comfort and to bring about treatment.

6. Patients never care how much you know until he knows how much you care.

7. Context, tone of voice and ritual of examination all these things are profoundly important.

It is very important that you are good in clinical dermatology before embarking on cosmetology or lasers. As a dermatologist you need to give something back to the community in the form of skin health education, awareness of common skin diseases and free services to needy and deserved people. This will go a long way in getting back the faith in doctors.

Wish you all the very best

Long live IADVL
Jai Karnataka

Dr Sathish Pai
President
IADVL Karnataka
Dear Residents,

You are all an integral part of the dynamic and vibrant IADVL. IADVL Karnataka has various academic activities for residents such as DERMABASICS, DERMAADVANCE and various CMEs. You can also make use of the online educations activities; Dermagyan, Digital Lecture Series and Dermatopathology Lecture Series. These are accessible on the members section of www.iadvl.org and www.iadvlkarnataka.org

You can also avail grants and scholarships to attend various national and international conferences. These are offered by IADVL national body and keep visiting our websites for regular updates.

Developing soft skills is an essential requirement for your future practice. To orient our residents to soft skills development, IADVL Karnataka has initiated ‘Inner Circle’, a soft skills development program for residents.

I congratulate Team YUVADERMA for successfully bringing out yet another issue of YUVADERMA E Bulletin and also for their other activities such as conducting poster competitions for residents, conducting YUVADERMA sessions etc. I thank YUVADERMA Editor in chief Dr Kirti P Katwe and YUVADERMA Advisor Dr Preethi B Nayak for leading this team. I also thank YUVADERMA associate editors Dr Gagana B Gopal, Dr Pranami Kashyap, Dr Priyanka Karagaiah, Dr Sanjay Thejaswi R, Dr Shibani Bhatia and Dr Shilpitha Srinivas for their support in all activities of YUVADERMA.

I request the first year residents to become Provisional Life Members (PLMs) of IADVL as soon as you join the department and the final year residents to upgrade your IADVL membership from PLM to LM immediately after passing the final examinations. I also request all the residents to actively participate in the various activities of YUVADERMA and IADVL Karnataka.

DR. MANJUNATHA R
Honorary secretary general,
IADVL Karnataka.
Dear Residents,
Warm greetings!

First and foremost, it gives me immense pleasure to know that YUVADERMA 8th edition is going to be released during CUTICON Gadag. I would like to congratulate the whole team; Dr Shilpitha, Dr Sanjay, Dr Priyanka, Dr Shibani, Dr Pranami, and Dr Gagana, for their hard work and dedication. This edition reflects the effort by zealous and highly talented, IADVL – Karnataka Yuvaderma, editor-in-chief, Dr Kirti P Katwe. Yuvaderma Karnataka is reaching greater heights with every passing year, this would not have been possible without, respected Dr Manjunatha R, Chairperson, Yuvaderma – KN, who has left no stone unturned in moulding this forum for the benefit of the residents.

Yuvaderma is for young budding dermatologists to showcase and nurture their talent. Dermatology being on top today, dermatology residents are among the brainy, skillful and competent minds. Given a right platform, at the beginning of their career, they can showcase and accomplish in plenty. Yuvaderma is one such platform, where the residents can express themselves, without any restrictions, to the best of their ability. Yuvaderma has consumed time, patience, hard-work and toil of a lot of residents and respected IADVL EC committee members, but at the end of it, when I look at the way it is shaping up, fascinates me and makes me happy to be a part of it.

Yuvaderma has been successful today due to the collective efforts, hence I request all the residents to actively participate in various activities conducted by National Resident Connect Committee, IADVL and Yuvaderma – Karnataka.

The 8th issue of the newsletter is here now; I am sure it will be a masterpiece and one of its kind.
Hope you have a good read!
Thanks & regards!

Dr Preethi B Nayak
Convener, National Resident Connect Committee, IADVL (2019-20)
Advisor, Yuvaderma Karnataka (2019-20)
Dr. YELLAPRAGADA SUBBAROW
(THE MIRACLE MAN OF MEDICINE)

Dr. Subbarow (January 12, 1895–August 08, 1948) was born in Bhimavaram, a small town in Andhra Pradesh. His childhood was traumatic due to the early death of his father and was bought up by his mother. He passed the matriculation examination in his third attempt and entered Presidency College, Madras. Being spiritually inclined, he spent most of his time at the Ramakrishna Mission. After passing the intermediate examination, he joined the Madras Medical College with the intent that he could serve in Ramakrishna Mission's hospitals after graduation.

He was denied of MBBS degree because of his practice of wearing khadi which incurred displeasure of his surgery professor and was awarded the Licentiate in Medicine and Surgery (LMS). Subbarow applied to join Madras Medical Service but was rejected as he did not have a graduate degree. He took up a job as a Lecturer in Anatomy in Ayurvedic College and began to engage in research to put Ayurveda on a modern footing.

He obtained his diploma from Harvard school of tropical medicine in October 1923 and gained interest in biochemistry. In 1940, he joined the world-renowned Lederle Laboratories as Director of Research and spent the rest of his life in the USA. He devised the now famous “Fiske-Subbarow method” for estimation of phosphorous in body fluids and tissues. It is an important tool to diagnose disorders of the thyroid and renal rickets. Subbarow discovered that adenosine triphosphate (ATP) provided energy for every biochemical process including muscle contractions. Subbarow next took up the challenge of pernicious anemia, which afflicted many. He extracted Vitamin B12 from pig liver which proved effective against anemia. A student of Madras medical college, his elder brother and younger brother both died due to tropical sprue in the span of 8 days. He subsequently discovered folic acid as a cure for tropical sprue. Research of obtaining folic acid in a pure crystalline form was done at Lederle Laboratory by the team called the “folic acid boys,” under the supervision and guidance of Dr. Subbarow.

He subsequently discovered folic acid as a cure for tropical sprue. Research of obtaining folic acid in a pure crystalline form was done at Lederle Laboratory by the team called the “folic acid boys,” under the supervision and guidance of Dr. Subbarow.
Penicillin was discovered in 1928. Dr. Subbarow was perceptive to this development and directed research into antibiotics. He succeeded in growing mold A-377 resulting in the development of chlortetracycline, a product of an actinomycete, the first of the tetracycline (Aureomycin) to be identified. Discovery of the drug was disclosed in a symposium held on July 21, 1948, just 2 weeks before Dr. Subbarow's demise. The antifilarial action of hetrazan (Diethylcarbamazine) was discovered and described in 1947 by Subbarow et al. while working with cotton rats infected with Litomosoides carinii. Hetrazan is on the World Health Organization’s “List of Essential Medicines,” most important medications needed in a basic health system. Dr. Subbarow led some of America’s most important medical research during World War II. He authored over 100 papers which have been published.

There are several unpublished works. He neither sold his scientific discoveries nor sought patents for any of the products. Despite trailblazing discoveries, the Nobel Prize eluded him. However, in 1950s, Subbarow’s colleagues had seen a portrait of him in “Karolinska Institute” in Stockholm, institute which awards the Nobel Prize in physiology and medicine.

American Cyanamid honored his memory with a plaque at its research laboratory and inaugurated the Subbarow library. A drug was named Subbaromyces splendens. Subbarow’s birth centenary was celebrated in 1995. India Post released a stamp in his honor, and a bust was erected in the campus of National Institute of Nutrition, Hyderabad.

Dr. Yellapragada Subbarow as “An eminent medical mind of the century.” Doron Antrim, a journalist, wrote in the April 1950 issue of “Argosy” (after the demise of Dr. Subbarow), “You’ve probably never heard of Dr. Yellapragada Subbarow. Yet, because he lived, you may be alive and well today.”
**Introduction:** Hair shaft abnormalities are structural defects of the hair shaft. They can be inherited or acquired. Inherited hair shaft abnormalities can be associated with increased hair breakage or can lead to unruliness of the scalp hair. Few congenital hair shaft disorders may be associated with involvement of other systemic components.

### Classification of hair shaft disorders:

<table>
<thead>
<tr>
<th>Hair shaft disorders with increased fragility</th>
<th>Hair shaft disorders without increased fragility</th>
</tr>
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<tbody>
<tr>
<td><strong>Congenital</strong></td>
<td><strong>Congenital</strong></td>
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<tr>
<td>- Congenital trichorrhexis nodosa (arginosuccinic aciduria)</td>
<td>- Piliannulati</td>
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<tr>
<td>- Monilethrix</td>
<td>- Pseudopiliannulati</td>
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<tr>
<td>- Pil torti (Bjornstad, Menkes)</td>
<td>- Woolly hair</td>
</tr>
<tr>
<td>- Trichorrhexis invaginata (Netherton syndrome)</td>
<td>- Uncombable hair syndrome</td>
</tr>
<tr>
<td>- Trichothiodystrophy</td>
<td>- Mary Unna hypotrichosis</td>
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<tr>
<td>- Trichoschisis</td>
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</tbody>
</table>

**Acquired**

- Bubble hair
- Acquired trichorrhexis nodosa

### Congenital Hair shaft disorders with increased fragility

<table>
<thead>
<tr>
<th>TRICHORRHEXIS NODOSA (TN)</th>
<th>TRICHOThiodYSTROPhy (TTD)</th>
<th>TRICHORRHEXIS INVAGINATA (TI)</th>
<th>PILI TORTI (PT)</th>
<th>MONILETHRIX</th>
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<tr>
<td><img src="image1.png" alt="Image" /></td>
<td><img src="image2.png" alt="Image" /></td>
<td><img src="image3.png" alt="Image" /></td>
<td><img src="image4.png" alt="Image" /></td>
<td><img src="image5.png" alt="Image" /></td>
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**Clinical findings**

<p>| Most common               | Diffuse nonscarring alopecia and various systemic defects | Hair that does not grow long. Most notable in the occipital scalp; Eye brows may also be sparse. | Hair that does not grow long and is easily broken; “spangled” appearance | Hair rarely grows beyond 1-2cm in length because of breakage. Topical minoxidil and oral etretinate may improve hair growth |
| Abnormal fragility of the hair becoming evident shortly after birth. | The hairs break with a clean cut (trichoschisis) | Tug test positive | Tug test positive | **|</p>
<table>
<thead>
<tr>
<th>Associated syndromes</th>
<th>Inheritance</th>
<th>Genetic defect</th>
<th>Other features</th>
<th>Microscopy findings</th>
<th>Dermo-scopical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Argininosuccinuria (AR) Argininosuccinate lyase deficiency</td>
<td>Citrullinemia (AR) Argininosuccinic acid synthetase deficiency</td>
<td>Argininosuccinate excision repair genes (XPD, XPB, TTD-A) TTDN1 (non-photosensitive TTD)</td>
<td>Low content of cysteine (sulfur) in cuticle and cortex</td>
<td>Frayed cortical fibers pushed up against each other. Alternating light and dark bands (&quot;tigertail&quot;) seen with polarized light. Distal hair shaft invaginating into the proximal hair shaft (&quot;bamboo hair&quot;). Flattened and twisted at 180° angles. Fractures occur at twists.</td>
<td>White nodules along hair shafts as well as fractured and frayed ends with brushlike appearance. Nonhomogenous structure of &quot;grains of sand&quot; and slightly wavy contour. Characteristic bamboo, matchstick and golf-tee hairs. Hair shaft with sharp bending at irregular intervals. Hair shaft beading caused by presence of elliptical nodes regularly separated by narrow internodes.</td>
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<tr>
<td>Crandall syndrome</td>
<td>Low serum copper and ceruloplasmin. Parenteral copper histidine in the first 8 weeks of life may be of benefit;</td>
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</table>
## Congenital: Hair shaft disorders without increased fragility

<table>
<thead>
<tr>
<th>Condition</th>
<th>Clinical findings</th>
<th>Associated syndromes</th>
<th>Inheritance</th>
<th>Genetic defect</th>
<th>Microscopy findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piliannulati (PA)</td>
<td>Alternating light and dark bands; only detectable in lightly pigmented hair. Hair grows long, non fragile. The bright bands correspond to abnormal air-filled cavities within the hair cortex.</td>
<td>AD, sporadic</td>
<td>AD</td>
<td>AD, sporadic/ Keratin defect (K6hf)</td>
<td>On a hair mount, these bright bands appear dark because transmitted light is not permitted to pass through.</td>
</tr>
<tr>
<td>Uncombable hair Syndrome (UHS)</td>
<td>Dry, shiny (&quot;spunglass&quot;), cannot be combed flat. Not as noticeable in dark hair.</td>
<td>Naxos disease AR/ Plakoglobin</td>
<td></td>
<td>AD</td>
<td>Longitudinal groove along hair shaft.</td>
</tr>
<tr>
<td>Woolly hair (WH)</td>
<td>Tightly curled hair in persons of non-African ancestry. Hair is lighter and thinner when compared to adjacent, normal hair in Wooly hair nevus.</td>
<td>Carvajal syndrome AR/Desmoplakin</td>
<td></td>
<td>AD</td>
<td>ruffling of the cuticle adjacent to the anagen bulb-&quot;floppy sock&quot;. The root sheath is absent or a small everted remnant.</td>
</tr>
<tr>
<td>Loose anagen Syndrome (LAS)</td>
<td>Hair is easily pulled from the scalp. Most cases are blonde girls &gt;2 years of age. Gentle hair care recommended</td>
<td>Naxos-like disease AR/Desmoplakin</td>
<td></td>
<td>AD</td>
<td>shows irregular twisting, and scanning electron microscopic examination shows longitudinal ridging and cuticle peeling</td>
</tr>
<tr>
<td>Maria Unna Hypotrichosis</td>
<td>Scalp hair is sparse or absent at birth, with variable coarse, wiry hair growth in childhood, affecting scalp and eyebrows. Late childhood, scalp hair is lost in a pattern resembling androgenetic alopecia with a scattering of coarse thickened hairs in the balding areas. Body hair is sparse to absent.</td>
<td>AD</td>
<td></td>
<td>AD</td>
<td></td>
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</table>
Acquired Hair-shaft disorders

Bubble hair
- Occurs when bubbles form within the hair cortex due to high temperatures from styling with blow dryers or curling irons
- Leads to hair breakage and alterations in hair texture

Acquired Trichorrhexis Nodosa
- More common than hereditary
- Proximal
  - breakage appears most commonly in African American women, usually after repetitive chemical or hot-comb straightening
- Distal trichorrhexis nodosa
  - is more commonly secondary to excessive brushing, back-combing, or sporadic use of permanent waves

Pohl-pinkus Lines
- Pohl-Pinkus lines are relatively common narrowings in hair-shaft diameter, which are frequently unrecognized
- May appear following serious illness, major surgery or antimitotic chemotherapy is due to early anagen hairs being sensitive to events that may produce a temporary disturbance of protein synthesis in the hair matrix
- Pohl Pinkus lines are the hair equivalent of Beau’s lines in nails

Peripilar Cast (peripilar keratin casts or pseudonits)
- Common sleevelike keratinous tubular structures of variable size that encircle the hair shaft seen in children and adults.
- Disorders of keratinisation and inflammation of the follicular ostium produces the casts.
- psoriasis and seborrhoeic dermatitis, lichen planopilaris or traction alopecia.
- The casts can also be of a non-keratin type; derived from chemicals (e.g., haircare products) or microorganisms (bacteria fungi) attaching to the hair.

Dermo-scopic Findings
- Shows alternating white bands that correspond to air-filled cavities.
- Shows triangular or reniform hair shaft with longitudinal groove or flattening
- Shows the image of hair shafts resembling a crawling snake with short wave cycles.
Figure 4 An algorithmic approach to hair shaft disorders

References:


Dr. Priyanka Karagaiah
PG-3
BMCRI, Bangalore.
Dr. Manjunath Shenoy is an eminent figure in the field of dermatology. He is the head of department, dermatology of Yenepoya Medical College, Mangalore. He has been the IADVL Karnataka president, ITART convener and IMA Mangalore president amongst all his other various contributions. He was presented with IADVL President’s appreciation award in 2018 for his pioneering work in ITART and was also awarded as the best HOD in Yenepoya Medical college this year. So here’s presenting his thoughts, valuable opinions, wisdom pearls with a peak into his personal life in his own words -

SB : Hello sir, to begin with, can you tell us something about your childhood? Where did you do your schooling from? Did you always want to become a doctor?

SIR : I would like to say that it’s a very nice initiative that Yuvaderma has been taking over the years. It’s a great honour for me to come in the list of people whom Yuvaderma has been interviewing. My childhood was spent in a village called Kukkundoor near Karkala. It’s a small village with less than 100 population. I did my schooling in Model School over there till 10th standard. Then I did my college in Karkala town, so that’s how my journey started. My parents always told me its either doctor or nothing! That’s the way they promoted me. We are 4 siblings and I am the youngest. My parents wanted all four of us to become doctors - but only my sister (who took up dentistry) and I went ahead with this profession. I am the youngest and had maximum pressure on me as no one else took up MBBS. But I am happy they took this decision for me as in those days in villages doctors were like god! So my parents felt it would be great and that was one of the reasons I took medicine.

SB : Why did you choose dermatology? Was there any other branch that you were interested in?

SIR : Dermatology was not my first choice. It was my belief that I am misfit for surgery; had a little bit fear for blood. My first choice was internal medicine but as I did not get it - dermatology was my second choice. Infact, after taking up dermatology I started preparing again but then I grew interest in the subject and continued and I’m happy for that today!

SB : Sir you have been the convener of IADVL Task-force Against Recalcitrant Tinea. Can you tell us a few things about ITART and about your experience?

SIR : This was one of the most important responsibilities which was given by IADVL to me because we are going through an epidemic of dermatophytosis. So, it was in focus for last 5-6 years. Hence, IADVL created this task force with respect to research, patient education and HCP education - so that was the motive behind this task force coming up. I took up as the convener as a I had some good experience in this field - I wrote the chapter on dermatophytosis in IADVL
reference book. I’ve done some original work on dermatophytosis as well and that’s why the association and the president felt I was suitable to take up the post. In two years, we did many things - patient education activities, focused CMEs (first one started in my institution itself called Dermato-fight), webinars, published a few articles in peer reviewed journals as ITART members and brought books apart from patient education leaflets as well. The major work was to bring about a consensus guideline on the management of dermatophytosis in current scenario. Which will be published soon. It is a compilation of the evidences we already have and on the basis of expert opinion.

SB : Any particular person in your life who has been an inspiration or guiding force in your life?

Sir: There have been many, I can’t name one. Some influenced me when it came to academics, some guided/influenced me when it came to hard work and some influenced me in leadership qualities. There isn’t only one. I have not only been inspired by people I know personally but also those I don’t know personally like movie stars, sports persons and politicians.

SB : Sir, what would be your advice to a first year resident - tips to become a good dermatologist in future?

Sir : Residency program has undergone a major metamorphosis in the past twenty years or so. The time when we were residents we learnt most of the skills from our senior residents/post graduates/junior staff - we were scared to communicate with our senior staff. But that gap has reduced now and there’s so much more interaction with the senior faculty of not only your own institution but also other college professors. So, the residents now have a lot of opportunity and comfort of learning a lot from their teachers. What is expected of residents is continuous work, as learning is a never ending process. Seeing patients in opd, working up cases in the ward and immediately going back in the evening and reading those cases – that’s more than enough. There’s no need for specific exam oriented learning because even an examiner checks how good a clinician you are rather than a bookworm. So working hard, seeing cases and reading them up - that’s what will make them a good dermatologist in future.

SB : And what would you recommend a final year post-graduate - is it wise to directly jump into private practice or to work in medical college/under a senior dermatologist initially?

Sir : Current breed of post-graduates are much more evolved than we were. I think you are all smart and quite capable. I think initially you should take up one sub-speciality - do a crash course or fellowship in it. Then start your own practice as soon as possible because the strength of a clinician is to practice early, one can even build their own institution on day one if they want to. The patients are going to do your publicity, so it’s better to introduce yourself early. Obviously in addition to this you can work with a senior doctor or medical college at the same time.
SB : Sir, you’re the head of department, an academician, consultant, have so many publications, been the IADVL Karnataka president, quiz master and so many other things on the list. How do you balance it all?

Sir : Balancing is not easy, it’s a tough question for me to answer as well. Especially because we all have families too. But having said, if we take one at a time it is possible – I have done that. When I was president of IADVL Karnataka I did not take up any other major responsibility from elsewhere. So if we do too many things at one time we will be in trouble, number one. And number two, I have been fortunate that I have gotten good cooperation from wherever I have worked – whether it be my colleagues or my team. Like when I was in ITART everyone was very supportive and nice, when I was the president we had a very wonderful and helping EC team, currently in my department I have a very good set of colleagues - that’s how it is possible. If you have friends and colleagues with whom you can work together its real fun.

SB : How do you like spending your time off from work? What are your hobbies?

Sir : Currently, the only thing I have been doing in my time off from work is travelling. As a speaker I travel a lot. It was a bit a tough initially, but now I’ve become used to it. In fact I enjoy it now, meeting new people and catching up with old friends. It’s one of the best things I enjoy now. It’s not true leisure but I have made it into one. Wherever I go, I make it a point to visit one new place. But only one place, so I see it properly and make the most of it. Second thing I like is music - I listen to all genres of music from classical to pop, everything! Though its frequency has come down. I used to be a good athlete during my college days. I would play volleyball and table tennis – but it’s been a decade since I played regularly!

SB : Sir, how important do you think social media/PR/marketing is in today’s practice? Is it as important as good treatment skills and knowledge of your subject?

Sir : It depends on the kind of practice you do. If you’re a consulting dermatologist running a small clinic – you probably don’t need any of those things. All you need to do is to treat your patients properly. As I said earlier, your patients and your colleagues only act as your PROs. But you need to be a member of your parent body organization - like one should be an IADVL member. You should be an active member because that way you will automatically learn new things which you can incorporate in your practice. In addition you should also become a member of some doctors union, like IMA. IMA is a very good platform, which helped me a lot indeed! And even other singular organizations like Lions or Rotary, even that helps. That’s best for a consultant. But if you’re starting your own institution then you obviously need more publicity because then you need to show what extra virtues you have in addition to being a regular dermatologist. Because otherwise the ill qualified or underqualified tend to override qualified people. So it depends on the kind of practise you do – as a consultant in a small set up or big city, good patient care is itself enough.

SB : Documenting each and every detail has become so important today. Are we loosing that personal touch with the patient because of documentation? What do you think sir?

Sir : I agree with you, your statement is correct. To some extent documentation is bringing down the relation with our patients and how our approach towards the patient has changed over a period of time. But having said that, I feel it’s also a deficiency from our side. We need to do both - because
YUVADERMA

documentation is also very important. From the point of view of academics and from a legal point of view. So you can’t run away from documentation. Things were different 20-25 years ago than now. You need to document and also develop a good rapport with the patient. I think we lack the skill because we have been doing only one side of it. Now we have to develop the skill and I think if we put in a little more hard work then it is surely possible!

SB : With so many sub-branches like paediatric dermatology, dermatosurgery, dermatopathology, cosmetology, trichology etc come up - do you feel the necessity of DM in these branches in future?

Sir : First of all in my opinion, dermatology should’ve itself been a DM. I think an internal medicine program followed by dermatology would be the best case scenario. But in India the medical education system is such that it cannot happen. Sometimes when we have cases like TEN or erythroderma - I feel we lack those internal medicine skills as it wasn’t enough at MBBS level. But coming back to reality I feel paediatric dermatology has the potential to become a DM since its very vast. There are plenty of things which are specific to paediatric dermatology like genodermatoses, it definitely needs some additional skills. The other field is dermatopathology - which is also vast. If we look at it, pathology has a few DMs to offer post MD. Infact I feel neuropathology and dermatopathology are equally vast. But then who should pursue the DM – a pathologist or dermatologist is debatable. But surely it does need to be a sub-speciality as there are around 4 to 5 thousand named diseases in dermatology - so maybe a DM could be considered. Other fields like trichology need to evolve more as a speciality to take it up for three years.

Coming to last and quite interesting :

Rapid Fire round -

1. Most suitable branch for a spouse of a dermatologist?
   Microbiology
2. Favorite dermatology book?
   Moschella
3. Coffee or Tea?
   Tea
4. Which other country would you like to settle down in?
   Australia
5. Next vacation spot on your mind?
   Plenty. Any place that I haven’t seen yet.
6. If not a dermatologist - what would you be?
   Internal Medicine
7. iPhone or Android?
   Android
8. If you had one superpower what would it be?
   Lot of responsibility. I wouldn’t want to take it - HIGHER POWER COMES WITH HIGHER RESPONSIBILITY!
9. Dermatologists stethoscope – dermoscope or hand lens?
   Hand lens for the senior doctors and dermoscope for the younger generation.

10. If you could eat one food for the rest of your life what would it be?
    Maggi

11. Favorite topic in dermatology?
    Fungal infections

12. As a student would you study late night or wake up early?
    As a student, it was always late night.

13. If you could invite 3 famous people/celebrities for dinner who would it be?
    Mahendra Singh Dhoni, Rahul Gandhi anddd one lady would be probably Priyanka Chopra

14. Movies or TV shows?
    Movies

15. Favorite viva questions as an examiner?
    Many.. But I won’t ask don’t worry! Few of them would be -
    1) In leprosy - nerve supply to intrinsic muscles of the hand and their actions
    2) Neurosyphilis (one of my favorite topics)
    3) Subcutaneous mycoses

    It was my absolute pleasure to meet and interview sir, that too on world psoriasis day. I was awestruck by how successful and humble he is at the same time which is so inspiring. The things that sir shared and the wisdom he imparted - I have gained so much from it today and I’m sure the residents reading this would too! Also, the entire dermatology department of Yenepoya Medical College was so welcoming - I was truly overwhelmed!!
Dr Varsha M
PG - 1
BMCRI, Bangalore

Dr Madhu M
PG - 1
SSIMS, Davangere

Dr. Shruti Appannavar, PG - 3, BIMS, Belgaum.
What is molluscum contagiosum (MC) ?
- A self-limiting skin infection caused by poxvirus, Molluscum contagiosum virus (MCV), commonly seen in children with a prevalence of 5%-11%.

What is the causative organism?
- **Family:** Poxvirus
- **Genus:** Molluscipox
- **Species:** Molluscum contagiosum virus
- MCV is a large, brick-shaped poxvirus (DNA) that replicates within the cytoplasm of cells.
- **4 types:** MCV 1,2,3,4.
- Most common being MCV-1 (76–97%), MCV2 infection seen in adults and HIV patients.

What are the MCV genes which contribute to an impaired immune response to this virus?
- Homologue of a MHC 1 heavy chain, which may interfere with antigen presentation;
- Chemokine homologue that may inhibit inflammation;
- Glutathione peroxidase homologue that may protect the virus from oxidative damage by peroxides.
- **Hosts:** Humans, chimpanzee (one report)

What are the common age groups affected?
- Three groups are primarily affected:
  - young children (2 and 5 years)
  - sexually active adults, and
  - immunosuppressed persons, especially those with HIV infection.

What is the mode of transmission?
- Skin-to-skin contact (Swimming pools and shared bathing facilities)
- Sexual transmission

What are the predisposing factors?
- Atopic eczema (Topical steroids and also topical calcineurin inhibitors have been suspected as a contributing factor)
- **Widespread lesions:** immune compromised, such as HIV (10 and 30% of AIDS patients not receiving antiretroviral therapy), haematological malignancy, sarcoidosis, idiopathic CD4 lymphocytopenia, hyper-IgE syndrome, DOCK8 deficiency and in those receiving immunosuppressive therapy suggesting that cell-mediated immunity is significant in the control and elimination of the infection.

What are the clinical features of MC infection?
- Smooth-surfaced, firm, dome-shaped, pearly papules, 3–5 mm in diameter.
- “Giant” lesions - up to 3cm.
- Central umbilication is characteristic.
- Asymptomatic / pruritus in patients with underlying atopic dermatitis.
- Pseudo koebnerization is seen
- Chronic conjunctivitis and punctate keratitis in patients with eyelid lesions.
- **Irritated lesions**: Crusted and even pustular, simulating secondary bacterial infection. This may precede spontaneous resolution. "BOTE" sign (for Beginning Of The End) - inflammation as an expected variant in the evolution of molluscum immunity.

- **Meyerson phenomenon**: an eczematous lesion around a central molluscum and is often called "molluscum eczema". It is thought to be one of the inflammatory phenomena that precede the disease resolution, hence, one of the variations of BOTE sign.

- Rarely associated with erythema annulare centrifugum.

- Rarely, may present as a cutaneous horn (molluscum contagiosum cornuatum).

- Untreated HIV disease, lesions favor the face and genitalia. Few or numerous, forming confluent plaques. Giant lesions (may be confused with a basal cell carcinoma).

- **Involvement of the oral and genital mucosa**: indicative of advanced AIDS (helper T-cell count less than 50).

Describe the histopathological findings

- Histopathological appearance on scanner view resembles Septate tomato.

- Epidermis - acanthosis

- Intracytoplasmic inclusion bodies the so-called molluscum bodies/Henderson Patterson bodies: minute, ovoid eosinophilic structures - in the lower cells of the stratum malpighii, increase in size as infected cells move toward the surface.

- The molluscum bodies in the upper layers of the epidermis displace and compress the nucleus so that it appears as a thin crescent at the periphery of the cell. At the level of the granular layer, the staining reaction of the molluscum bodies changes from eosinophilic to basophilic. In the horny layer, basophilic molluscum bodies measuring up to 35 Åµm in diameter lie enmeshed in a network of keratin filaments.

- The basal layer remains intact.

**What is the cause for central umbilication?**

- In the center of the lesion, the stratum corneum ultimately disintegrates, releasing the molluscum bodies. Thus, a central crater forms.

Name the differential diagnosis of MC

- Verrucae
- Pyogenic granuloma
- Papular granuloma annulare
- Epidermal inclusion cyst
- Sebaceous hyperplasia
- Appendageal tumors
- Basal cell carcinoma
- Amelanotic melanoma
- Cryptococcosis/histoplasmosis/penicilliosis

**Differential diagnosis for umbilicated lesions**

1. **Infections**
   - **Viral**: Molluscum contagiosum (MC), Cowpox, Orf, Milker’s nodule (MN), Eczema herpeticum.
   - **Fungal**: Disseminated cryptococcosis in HIV, Histoplasmosis, Penicilliosis.
   - **Bacterial**: Histoid leprosy, Methicillin-resistant Staphylococcus aureus (MRSA) septicemia.

2. **Noninfective granulomas**
   - Granuloma annulare (GA)
   - Sarcoidosis.

3. **Perforating disorders**
   - Reactive perforating collagenosis (RPC)
Perforating lichen nitidus (LN)
Perforating folliculitis.

4. Soft-tissue tumors
- Keratoacanthoma
- Pilar sheath acanthoma (PSA)
- Trichofolliculoma
- Desmoplastic trichoepithelioma (DT)
- Sebaceous gland hyperplasia (SGH) and sebaceous adenoma.

5. Malignancies : Leukemia cutis (LC)
6. Others : Acrokeratoelastoidosis of Costa, Vesicular pityriasis rosea (PR)

Disease course and prognosis
- Most cases are self-limiting within 6–9 months, some may persist for 3 or 4 years
- Depressed scars or anetoderma like lesions can remain when mollusca clear.

Investigations
- Diagnosis is mainly clinical. The umbilication can be seen more easily with a dermoscope or after freezing.
- Confirmed by direct microscopy or electron microscopy of the papule contents, by histopathology or by molecular analysis

Mention the management options
- Most cases self-resolve in 9–12 months but lesions often treated to speed resolution; can be persistent/ refractory in immunocompromised individuals.
- The risk of dissemination of the infection can be minimized by reducing scratching.
- Associated dry skin or eczema should be treated with emollients and possibly a weak topical steroid.
- Transfer of infection to another individual may be reduced by avoidance of shared towels, contact sports and communal bathing.
- Treatments aim to destroy the infected epidermal cells, stimulate an immunological response or act directly against the virus.

Treatment ladder

First line
- Caustic destruction : cantharidin, trichloroacetic acid, diluted liquefied phenol
- Irritant : salicylic acid, adapalene, nitric oxide cream, potassium hydroxide, benzoyl peroxide, lemon myrtle oil, tea tree oil
- Surgical irritation

Second line
- Immunological : diphencyprone, imiquimod, interferon, cimetidine, intralesional immunotherapy
- Surgical removal

Third line
- Cidofovir, Paclitaxel

References :
Acanthosis nigricans
Velvety hyperpigmentation, seen commonly in the flexures and neck. Histology reveals hyperkeratosis and papillomatosis but only slight irregular thickening of stratum spinosum, despite the term acanthosis. There is no increase in the number of melanocytes. The dark color is due to hyperkeratosis rather than increase in melanin.

Acne keloidalis nuchae (Folliculitis keloidalis nuchae, Folliculitis Nuchae Scleroticans)
Chronic, inflammatory process involving the hair follicles of the nape of the neck, leading to hypertrophic scarring, papules, and plaques. The lesions do not occur as a result of acne vulgaris and neither are they keloidal.

Acne necrotica miliaris (Propionibacterium folliculitis)
Essentially a folliculitis, the lesions typically distributed throughout the scalp. It is an inflammatory reaction to Propionibacterium acnes and in severe cases to Staphylococcus aureus.

Acne rosacea (Rosacea)
The epidemiology, etiology, and pathology of rosacea are quite distinct from acne vulgaris. The condition was named acne rosacea in the past due to involvement of the face and presence of erythematous papules and pustules akin to acne vulgaris.

Actinomycetoma
Mycetoma is derived from the Greek word ‘mykes,’ which means fungus. Actinomycetoma is a chronic, suppurative, granulomatous disease with discharging sinuses, similar to mycetoma. It is caused by aerobic species of bacteria actinomyces and not by fungi as its name suggests.

Adenoma sebaceum (Facial angiofibroma)
Symmetrically distributed small, red angiofibromas of the face seen in tuberous sclerosis. In the past, they were mistakenly called adenoma sebaceum due to its distribution in the sebaceous area of the face.

Adult colloid milium
These are asymptomatic, shiny, smooth, firm papules 1–10 mm in diameter, involving chronically sun-damaged skin of adults. Microscopically, the papules show severe elastosis involving most of the papillary dermis. Thus the term papular-elastosis has been suggested.

Alopecia mucinosa (Follicular mucinosis)
Presents as hair loss in hair-bearing areas; however, lesions can also occur on glabrous skin. The acute form is seen as erythematous papules and plaques with variable scaling and patulous follicles. The chronic form is widespread and of diverse morphology, with scaly patches, papules, plaques, and nodules. Histopathology shows deposition of mucin in the hair follicles and sebaceous glands.

Auspitz phenomenon
The eponymously credited sign was not first discovered by Heinrich Auspitz (1835-1886). Both Devergie Jeune (1860) and Hebra (1845) observed this clinical sign earlier, as did Robert Willan (1808), Joseph Plenck (1776), and Daniel Turner (1736).

Axillary freckling (Crowe’s sign)
Freckles are pigmented macules seen in sun-exposed
Characterized by single or multiple cutaneous nodules, usually occurring over the face. Occasionally, extra-facial involvement is also noted, most often on sun-exposed areas. Histopathology shows mixed inflammatory infiltrate with a predominance of neutrophils and eosinophils, mainly in the upper half of the dermis. No granulomas are found.

areas. Thus, the term freckling applied to pigmented macules in the axilla, which is a characteristic sign of neurofibromatosis, is not appropriate as axilla is a commonly photoprotected area.

**Botryomycosis (Granular bacteriosis, bacterial pseudomycosis)**
Chronic granulomatous reaction to bacterial infection, in which bacteria like Staphylococcus, Pseudomonas, Escherichia coli, Proteus, and Streptococcus species form granules. It is not a fungal infection as the name indicates.

**Cavernous hemangiomas (Venous vascular malformation)**
Vascular malformations are localized defects of vascular morphogenesis always present at birth. They have a quiescent endothelium and do not exhibit the markers of proliferation seen in hemangiomas of infancy. As they are not proliferating lesions, the suffix ‘oma’ (meaning ‘tumor’) has been deemed inaccurate. Other similar misnomers are sinusoidal hemangioma and verrucous hemangioma.

**Dissecting cellulitis of scalp (Perifolliculitis capitis Abscedens et suffodiens)**
Chronic suppurative disease of unknown etiology. It is not a bacterial infection. Follicular blockage is proposed to be the initial event that leads to retention of material and dilation of follicles. On subsequent rupture of the follicle, released keratin and organisms initiate a neutrophilic and granulomatous response.

**Dyshidrotic eczema (Pompholyx, vesicular eczema of palms and soles)**
Pompholyx is referred to as dyshidrotic eczema due to the connection with sweat-gland activity, as the condition is worse in hot weather. No causal relationship with the sweat glands or sweating has been demonstrated.

**Erythema migrans chronicum (Erythema migrans)**
About 90% patients develop erythema chronicum migrans at the site of tick bite. Even if untreated, the lesion fades, usually within a few weeks to months. It is not a chronic condition.

**Erythema toxicum neonatarum (Erythema neonatarum)**
The term toxic means a physiological state produced by toxins or other poisonous substances. Erythema toxicum neonatarum is a common benign vesiculo pustular neonatal eruption, which affects 50% full-term neonates. The condition resolves spontaneously over 1-2 days.

**Fibroepithelioma of Pinkus**
It is generally regarded as a rare variant of basal cell carcinoma (BCC). Therefore, fibroepithelioma is not an accurate term for this disease.

**Follicular atrophoderma**
Characterized by follicular indentations occurring over the extensors of upper and lower limbs. On histopathology, no abnormalities of the elastic fibers have been found (nor any evidence of atrophy
of the epidermis, hair, or dermis), making the term atrophoderma (atrophy of the skin) a misnomer.

**Granuloma faciale**
Characterized by single or multiple cutaneous nodules, usually occurring over the face. Occasionally, extra-facial involvement is also noted, most often on sun-exposed areas. Histopathology shows mixed inflammatory infiltrate with a predominance of neutrophils and eosinophils, mainly in the upper half of the dermis. No granulomas are found.

**Granuloma fissuratum (Acanthoma fissuratum)**
Reactive skin process resulting from chronic trauma, commonly from ill-fitting eyeglasses and is seen over the intertriginous area of contact between the ear lobe and the tempero-occipital area. There is acanthosis with attenuation of the rete centrally, and dermis shows variable chronic inflammatory infiltrate without any granulomas.

**Granuloma gluteale infantum**
Erythematous to violaceous nodules and plaques appear in the napkin area of infants as a complication of diaper dermatitis, frequently at a time when the dermatitis is improving. On histopathology, there are no granulomas.

**Herpes gestationis (Pemphigoid gestationis)**
It is a rare autoimmune bullous dermatosis of pregnancy. The disease was originally named herpes gestationis on the basis of the morphological herpetiform feature of the blisters.

**Hidradenitis suppurativa (Acne inversa)**
The primary pathology is not suppurative inflammation of the apocrine sweat glands but an occlusion of the hair follicles, comparable to acne vulgaris. It is acne inversa because, in contrast to acne vulgaris, the disease involves intertriginous localizations.

**Horn cysts**
These intra-epidermal whorls of invaginated keratin found in seborrheic keratoses are not true cysts, because each connects to the skin surface via a prominent ostium. Horn pseudocysts is a more appropriate term.

**Hot-comb alopecia (Central centrifugal cicatricial alopecia, follicular degeneration syndrome)**
It is a progressive vertex-centered alopecia, seen most commonly in women of African descent, who use hot combs to straighten hair. There are reports of the condition occurring without the use of hot combs.

**Impetigo herpetiformis (Pustular psoriasis of pregnancy)**
It is not bacterial infection unlike impetigo, which is caused by Staphylococci or Streptococci.

**Kaposi sarcoma**
Kaposi sarcoma is a multisystem angioproliferative disorder characterized by proliferation of spindle-shaped cells, neo-angiogenesis, inflammation, and edema. There is controversy as to whether these proliferative cells truly represent a malignant neoplasm versus an inflammatory hyperplasia.

**Keratoderma blennorrhagica**
Vesiculo-pustular waxy lesion with a yellow-brown color, seen commonly on the palms and soles in reactive arthritis. Blenorrhagia, which means excessive flow of mucus, is an old term for gonorrhea. However, reactive arthritis occurs commonly due to non-gonococcal urethritis.

**Keratosis follicularis (Darier’s disease)**
Darier’s disease is characterized by keratotic papules predominately affecting the seborrheic regions such as the upper trunk and the head and neck areas. Most papules in this condition are non-follicular in location.

**Knuckle pads**
Benign, well-circumscribed, skin-colored papules or plaques, located over the dorsal aspects of the
metacarpophalangeal (MCP) and interphalangeal (IP) joints. In most reported cases, lesions occur over the proximal interphalangeal (PIP) joint, not over the knuckles.

Leser–Tre’lat Sign
The eponym has been wrongly credited to two European surgeons, Edmund Leser and Ulysse Tre’lat. While they apparently observed cherry angiomatosis in patients with cancer, it was Holla¨nder who first linked internal cancer with seborrheic keratoses in 1900.

Lupus miliaris disseminatus faciei/LMDF (‘FIGURE’ facial idiopathic granulomas with regressive evolution)
The term LMDF, also known as acne agminate, resulted from a historical classification of this condition as a presentation of tuberculosis. It is now known that the condition is neither tuberculous in etiology nor related to acne and is a distinct entity.

Lupus anticoagulant
Originally described in patients with systemic lupus erythematosus (SLE) and clinical bleeding. LA causes increased risk of both arterial and venous thrombo-embolic events. The riddle of the anticoagulant effect in vivo and the apparent procoagulant effect in vivo remains unsolved.

Lupus pernio
Large bluish-red and dusky violaceous infiltrated nodules and plaques that occur on the nose, cheeks, ears, fingers, hands, and toes in sarcoidosis. Perniosis describes localized inflammatory lesions on acral skin, which occurs as an abnormal reaction to cold. Lupus pernio has no association either with tuberculosis or with cold exposure.

Lymphangiosarcoma (Stewart-Treves syndrome, hemangiosarcoma)
Rare, cutaneous angiosarcoma that develops in long-standing chronic lymphoedema. Lymphangiosarcoma seems to arise from blood vessels instead of lymphatic vessels. A more appropriate name is hemangiosarcoma.

Melanoacanthoma
Histological variant of seborrheic keratosis. As most of these lesions resolve spontaneously in a few months, the term melanoacanthoma, implying that it is a tumor, is inappropriate.

Moniliasis
The term monilias is used as a synonym for candidiasis, and it is a misnomer as moniliasis refers to a stage of ascomycetes and has no relationship to the genus Candida.

Mycosis fungoides (Cutaneous T Cell Lymphoma)
Most common variant of primary CTCL, generally associated with an indolent clinical course. The exact etiology of mycosis fungoides is not yet established. It is not caused by fungal organism.

Necrobiotic granuloma (Palisading granuloma)
The term necrobiosis refers to change in collagen associated with an accumulation of histiocytes.
Necrosis refers to collagen change and biosis to cellular proliferation. Necrosis can occur only in cells, not in collagen fibers.

**Nevus depigmentosus (Achromic nevus)**
The areas of leukoderma are hypomelanotic and not amelanotic.

**Palisaded encapsulated neuroma**
Histopathology of PEN shows a bulbous expansion of a peripheral nerve, with Schwann cells forming uniform, broad, interlacing fascicles that are spaced in a clear or mucinous matrix. There is no palisading arrangement of cells.

**Pautrier’s microabscess**
Collection of atypical lymphocytes found within the epidermis of lesions of cutaneous T cell lymphoma. An abscess characteristically refers to a collection of neutrophils or alternatively to a cavity formed by liquefaction necrosis within a solid tissue.

**Peruvian wart/verruga Peruana (Oraya fever, Carrion’s disease)**
Oraya fever is caused by Bartonella bacilliformis, and characterized by erythematous papules, which become pedunculated. Some lesions may become very large. They are not viral infection as suggested.

**Piezogenic pedal papules**
The term piezogenic means ‘producing pressure,” when in fact these lesions are produced by pressure. It is postulated that pressure induces herniation of fat through connective tissue in the dermis of the heels.

**Porokeratosis**
The cornoid lamella corresponds to ostia of eccrine glands or hair follicles occasionally, which led to the term “porokeratosis;” however, this finding is fortuitous, since the peripheral border of porokeratotic lesions is moving centrifugally and, therefore, it cannot be permanently bound to epidermal adnexae (that are definite structures).

**Pyoderma gangrenosum**
Bacterial infection of the skin is pyoderma. Pyoderma gangrenosum is a rare, non-infectious neutrophilic dermatosis.

**Pyogenic granuloma (Lobular capillary hemangioma)**
Neither infectious nor granulomatous.

**Ringworm/Tinea**
Tinea is commonly referred to as ringworm, which stems from the annular appearance of the lesions. It is a superficial fungal infection of keratinized tissue by dermatophytes. The literal meaning of tinea is larva as Romans mistakenly thought the infection to be due to insects.

**Scleredema**
In scleredema, the collagen bundles are thickened without being hyalinized and stain normally with eosin and the trichrome stain. There is neither sclerosis nor edema.

**Sebaceous cyst (Epidermoid cyst)**
Most epidermoid cysts are acquired and appear to be derived from the infundibular portion of a hair follicle. These cysts are not of sebaceous origin.

**Seborrheic dermatitis**
Malassezia yeasts have been associated with seborrheic dermatitis. Abnormal or inflammatory immune system reactions to these yeasts may be related to the development of seborrheic dermatitis. Seborrhea plays no part in the pathogenesis of the rash in most patients.

**Seborrheic keratoses/Seborrheic wart/Senile warts**
Most common benign tumor in the elderly. The lesions are well defined and flat initially, later develop a velvety to verrucous surface. It is derived
from keratinocytes and not sebaceous glands, and neither are they viral in etiology.

**Tinea amiantacea (Pityriasis amiantacea)**
It represents a particular reaction pattern of the scalp to various inflammatory scalp diseases. It is not associated typically with dermatophyte infection.

**Transient acantholytic dermatosis (Grover’s disease)**
It a self limited disorder, characterized by pruritic, papular, or papulo vesicular eruption, most commonly occurring on the trunk that resolves over weeks to months, but it can also be persistent and may repeatedly recur for years. Thus, the term transient is not appropriate.

**Trichomycosis axillaris (Trichobacteriosis)**
Common tropical disease affecting the hair shafts in sweat gland-bearing areas like armpits and pubic region by Corynebacterium tenuis. It is not a fungal infection, which is limited to the axilla.

**Trumpeter’s wart**
A hyperkeratotic nodule that develops on the upper lip of trumpet players due to friction. It is not caused by any virus.

**Water wart (Molluscum contagiosum)**
Molluscum contagiosum is also called water wart, as the lesion looks pink or translucent.

**REFERENCES :**


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FROM THE DERMATOLOGIST’S LENS

Dr. Priyanka Karagaiah
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Hi Everyone,

This is for all the Post Graduate Residents who will be attempting their final exam within a few months. I wrote my MD Final exam in May 2019. Most people don’t know the extent of information that they need to assimilate when they join the Post Graduate course in Dermatology, Venereology and Leprosy. They often get dismayed when they look at the size of the books that they have to read, in order to complete their degree. Following are the few points that I think would be useful for anyone who is preparing for the exams:

**E book Versus Textbook?**

Either one is fine as long as you highlight and repeatedly revise the main points. All your information should be concised into one book. I did study from textbooks but I found it inconvenient to carry around.

**Which book to Select:**

A lot of time gets wasted in trying to find the most suitable book for oneself. For General Dermatology any book is fine like Rook’s, Fitzpatrick’s and Bologna. For STD’s Bhushan Kumar, V K Sharma are good. King and Nicole is good for syphilis. For Basics and Histopathology I studied from Moschella and Levers. For Leprosy, I studied from IAL, Jopling and Hastings. For Aesthetics I referred to the ACSI Textbook.

**How to Study the topics:**

I preferred to take points from IADVL, as it had information regarding Indian patients as well as Rooks, as it had most of the extra points that I needed. I added points from Journals based on guidance given by my staff and seniors. I also looked at old question papers. Ultimately, I had almost all the information I needed, concised in one book. This was helpful for me as I didn’t waste time towards the end shifting from one book to another. For those topics that were difficult to understand from both the books, I also had a stack of notes. Both Moschella and Levers were good for basic topics and so I took points and made notes from both the books.

**Studying During The last Few Months:**

The last few months leading up to the exams can be especially stressful. Most people experience repeated bouts of self doubt and worry that one may not be able to complete everything in time. Due to more work load in the department, many colleges don’t give adequate study leave. Planning...
how you will be able to cover the important topics is important. As soon as I was finished with my thesis submission, I studied all the topics which I had not read previously during my Residency. This was probably around December. I started making concise notes on difficult topics like Mastocytosis, Deep fungal Infections, Epidermolysis Bullosa, Histopathology and Basics in dermatology. I kept a separate notebook for this. I also added diagrams which I needed to practice in this book.

After attending Dermadvance in January I had a better understanding of what to study and how much I needed to study. I got guidance from the Professors who conducted the CME. I went through the recent advances and the Biologics released in the past 2 years. I also studied the latest CDC Guidelines for the STDs.

Getting those Extra Marks:

This can only be achieved through repeated revision of the oft asked questions. I planned what I wanted to revise. I started from the toughest topics, which would require more time, to the easy topics, which could be completed in a short time. I studied up to 10-12 hours a day. I divided my time between all the topics. Presentation of your answers in an organised fashion and underlining the main points is important. Ultimately, all examiners would like to read a well written paper. Most of us have already been advised on how to write a paper. Including diagram, tables and flowcharts are important.

On the Day of the Exam:

Most people burn the midnight oil just before the exams. I preferred to sleep for at least 6 hours. I knew I had to prepare for the next exam and I would need adequate rest for the same. Having a nutritious breakfast and being adequately hydrated helped me to recollect everything that I had read. Like most other students, I felt that during the study leave I was not able to revise everything that I wanted to for the exam. In the three years of my course, my regular studying habits, helped me to answer a couple of questions without revision in the last few months.

To conclude, I studied mainly because I wanted to know enough to practice with confidence. At the end of the day, this exam is just like any other exam. The results show how sincerely you worked for the three years of your Post Graduate course. Passing is a stepping stone to the next level in your career. The results don’t necessarily indicate whether you will be successful or not in your practice. That is up to you!
itch that rashes!!!

Most of the conditions in dermatology present with rashes which is associated with itching. Here we list few conditions where itching precedes the rash, which may be primary lesions or secondary lesions due to scratching like excoriations.

Mnemonic: “ABCDEF PLUS T”

- Atopic dermatitis
- Bullous pemphigoid
- Chronic liver failure
- Cholestatic liver disease
- Debilitated skin
- Elderly skin
- Fibreglass dermatitis
- Polycythemiathe
- Prurigonodularis
- Lymphoma
- Urticaria
- Systemic pruritogens
- Thyroid disorders

Dr Thabassum
PG - 3
MIMS, Mandya
**AT THE BACK OF THE NECK!**

There is an arena of lesions which present at the back of the neck. Here is a collection of common and rare dermatological conditions which present to the OPD.

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**INFECTIONS:**
- **FOLLICULITIS** - infection of the hair follicle
- **CARBUNCLE** - pyoderma commonly seen in diabetics
- **TINEA CORPORIS** - lesions seen on

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**CONGENITAL ANOMALIES:**
- **Dermoid Cyst** - congenital benign tumors with mesodermal and ectodermal components usually seen along the midline.
- **Meningocele** - is a devastating congenital malformation characterized by open neural tube defect.

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**CONDITIONS WITH POSSIBLE ENDOCRINOLOGICAL ASSOCIATION:**
- **ACROCHORDONS** - pedunculated or sessile papules around the neck, axillae.
- **ACANTHOSIS NIGRICANS** - velvety hyperpigmented plaques.

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**OTHER COMMON CONDITIONS:**
- **POLYMORPHIC LIGHT ERUPTION** - Chronic recurrent photodermatitis.
- **ACNE KELOIDALIS NUCHAE** - chronic scarring folliculitis leading to keloidal lesions on the nape of neck.
- **CUTIS RHOMBOIDALIS NUCHAE** - deep furrowing in geometrical pattern at the posterior part of neck.

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**DR. SHRUTI APPANNAVAR**
PG - 3
BELAGAVI INSTITUTE OF MEDICAL SCIENCES
VULGARIS IN DERMATOLOGY

The term ‘vulgaris’ is a latin adjective meaning common, or something derived from the masses of common people. In dermatology it refers to the normal, classic or most common of a given disease.

1. Acne vulgaris: A very common malady of adolescents defined as a chronic, self-limiting, inflammatory disease of pilosebaceous unit, manifesting with pleomorphic lesions like comedones, papules, pustules, nodules and cysts which may lead to scarring.

2. Icthyosis vulgaris: (greek: icthys meaning fish) (syn: common ichthyosis, ichthyosis simplex)
   It is the least severe & most common type of ichthyosis. Ichthyosis are disorders of cornification in which abnormal differentiation and desquamation of epidermis result in a defective epidermal barrier.

3. Impetigo vulgaris: (latin: Impetere-to attack) (syn: school sores)
   It is a superficial highly communicable infection, noted predominantly in preschool aged children most commonly caused by staphylococcus aureus and group A beta haemolytic streptococcus.

4. Lupus vulgaris: (syn- tuberculosis luposa)
   It is the most common cutaneous mycobacterium tuberculosis infection characterised by red brown plaques revealing typical apple jelly colour on diascopy

5. Pemphigus vulgaris: (greek- pemphix meaning blister or bubble)
   It is an autoimmune, intraepithelial, blistering disease affecting the skin and mucous membranes mediated by circulating autoantibodies against keratinocyte cell surfaces.
6. Psoriasis vulgaris: It is a chronic, relapsing, multifactorial disease with both genetic & environmental factors playing a role in its development characterised on the cellular level by hyperproliferaton, and abnormal differentiation of epidermal keratinocytes.

7. Sycosis vulgaris:
(syn - sycosis barbae or barbers itch)
Deep seated folliculitis barbae is called sycosis barbae affecting the beard area due to infection with staphylococcus aureus.

8. Verruca vulgaris: (syn: common warts) It is caused by human papilloma virus type 2 but others like 1,4,7,27 & 57 are also associated and are characterised by dome shaped papules, nodules with finger like projections, giving cauliflower like appearance.

9. Vitiligo vulgaris: (Latin - vitelius meaning vale i.e pale pink flesh of calf) It is an acquired, often progressive disorder of hypopigmentation characterised by circumscribed achromic macules often associated with leukotrichia.

10. Polaris vulgaris (bullous frostbite): Seen in snowmobilers because of the frigid conditions in which they ride and the velocity they sustain. Bullous lesions may develop on unprotected neck after exposure to high wind velocity and extreme cold blisters contain clear yellow exudate.

Dr. Bhagya Shree S R
PG - 2
MIMS, Mandya
“DON’T JUDGE ME BY MY NAME: Misnomers in Dermatology”

<table>
<thead>
<tr>
<th>ACROSS</th>
<th>DOWN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I am not associated with tuberculosis or cold exposure. I develop as violaceous plaques over face in sarcoidosis.</td>
<td>1. Few called me elephantiasis, few as tsaraat, finally by this name which also doesn’t mean who I am</td>
</tr>
<tr>
<td>2. You see me in pregnancy; even though my name says green I am actually brown.</td>
<td>2. I’m neither wheals nor a disease of pilosebaceous unit, rather a chronic eruption seen in association with polycythaemia vera</td>
</tr>
<tr>
<td>3. I am a disorder of keratinisation but there are no holes as my name suggests. I have an active border represented by cornoid lamella.</td>
<td>3. My name says I am twisted; you can see me in many conditions but never in the disease that shares my name.</td>
</tr>
<tr>
<td>4. I am not a parasite but a dermatological condition caused by fungus.</td>
<td>4. I’m a chronic granulomatous infection; I may sound like a fungal infection but I’m not.</td>
</tr>
<tr>
<td>5. I’m an opportunistic infection, and not just a stage of ascomycetes as my name means.</td>
<td>5. My name means itch, but I don’t always itch and winter is my favourite season of the year.</td>
</tr>
<tr>
<td>6. I am not associated with HPV; I present as a soft pedunculated papule but with a firm fibrous core.</td>
<td>6. I am not a malignancy of mesenchyma; I rather form naked granuloma.</td>
</tr>
</tbody>
</table>
Psoriasis Poster competition : 1st Place

Psoriasis (ಸೋರಿಯಸ್) अನಾನುಭವವಿರುವ ಸಾಮಾನ್ಯವಾಗಿ ಬಹುಮಟ್ಟಿನ ಹಾರ್ಪೆಟ್ಟಿನಲ್ಲಿ ದೃಢವಾಗಿರುವ (ಸಹಿತ ರೋಗವು) ರೋಗ.

* ಸುಧೆ ಎರಡು ವರ್ಷಗಳ ಹದಿನ
  * ಬೀದ ಸ್ಮೃತಿ
  * ಬೀಜ
  * ಹೂದು ಸ್ಮೃತಿ

2019

* ಸಂಯೋಗಾಂಬೆಯ ಉಪಯೋಗ
* ಬೀಜ ಸ್ಮೃತಿ
* ಬೀಜ
* ಹೂದು ಸ್ಮೃತಿ

* ಸಂಯೋಗಾಂಬೆಯ (ನಿವೃತ್ತಿ ಕೇಂದ್ರ)

* ಬೀಜ ಸ್ಮೃತಿ

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* ಬೀಜ ಸ್ಮೃತಿ

* ಬೀಜ ಸ್ಮೃತಿ
"ಪಾಠ್ಯಪ್ರದಾಯ ಮನೆ"
ಅಂಗಾಂಶ 29
ಪಾಠಪದಪತ್ನಿಯ 125 ಪ್ರದರ್ಶನ (3) ಎಂಧ್ಯಾಗಿ ಪ್ರಪಂಚಕ್ಕೆ ಎರಡು

1. ರೋಗದ ಪ್ರತ್ಯೇಕಿತ ಚಿಕಿತ್ಸೆ.
2. ರೋಗದ ಪ್ರತ್ಯೇಕಿತ ಚಿಕಿತ್ಸೆ.
3. ರೋಗದ ಪ್ರತ್ಯೇಕಿತ ಚಿಕಿತ್ಸೆ.

ಚಿಕಿತ್ಸೆಯ ಪ್ರತಿಪಾದನೆಗಳು:
1. ರೋಗವಿಜ್ಞಾನ (54%)
2. ರೋಗವಿಜ್ಞಾನ (54%)
3. ರೋಗವಿಜ್ಞಾನ (54%)

ಚಿಕಿತ್ಸೆಯ ಪ್ರತಿಪಾದನೆಗಳು:
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4. ರೋಗವಿಜ್ಞಾನ (54%)
5. ರೋಗವಿಜ್ಞಾನ (54%)

ಯೋಜನೆಯ ಪ್ರತಿಪಾದನೆಗಳು:
1. ರೋಗವಿಜ್ಞಾನ (54%)
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5. ರೋಗವಿಜ್ಞಾನ (54%)

“ಪಾಠ್ಯಪ್ರದಾಯ ಮನೆ”
ಪಾಠ್ಯಪದಪತ್ನಿಯ 125 ಪ್ರದರ್ಶನ (3) ಎಂಧ್ಯಾಗಿ ಪ್ರಪಂಚಕ್ಕೆ ಎರಡು

YUVADERMA
Other poster participants
Other poster participants
Other poster participants
ANSWER KEY FOR CROSSWORDS

ACROSS:
1. Lupus pernio
2. Chloasma
3. Porokeratosis
4. Ringworm
5. Moniliasis
6. Soft wart

DOWN:
1. Leprosy
2. Acne urticata
3. Kinky hair
4. Botryomycosis
5. Psoriasis
6. Sarcoidosis

DR. BHANUTEJ K.L.
PG -2
MIMS, Mandya.

DR. JAYASHREE NAYAK
PG -2
MIMS, Mandya.
We hope you have liked this effort of ours. 
Mail us your feedback, queries and articles at 
iadvlkn.ebulletin@gmail.com

Regards,
Editorial Team