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ACAD EXPERT SESSION

“Chemical Peeling: The Art and Science of It”

"MELASMA"

Dr. Rashmi Sarkar

Dr. Bikash Ranjan Kar

Dr. Geraldine Jain

(27/02/2017 - 14/03/2017)

MELASMA

1. What role do the AGOUTI proteins play in the pathogenesis of Melasma?

   Agouti signalling protein is a soluble protein of 131 amino acids, apparently secreted by dermal papilla cells in hair bulbs. It competitively antagonizes ?MSH at the MC1R and inhibits the eumelanogenic signal, down regulating melanogenic enzymes and leading to pheomelanin synthesis.

2. What would be the topical agent(s) of choice for Melasma?

   For a treatment naïve patient the first drug would probably be Hydroquinone or a triple combo for a short duration of 2 -3 weeks of course under the coverage of daytime broad spectrum sunscreen .Hydroquinone at 4% can be irritating to some in which case triple combo may be of help. Maximum duration of hydroquinone use is 2-3 months beyond which chances of exogenous ochronosis increases.

3. Does modified Kligman's regimen still find a place in today's practice?

   Yes. This has a definite place in treatment naïve patients. This combination induces a rapid clearance in a short period and patient gets a confidence in the treating dermatologist.

4. Which oral agent do you prefer in Melasma management?

   Antioxidants

   Oral Iron supplementation if anaemic

   Tranexamic acid

5. Which peel (or peel combos) would you recommend for Melasma?

   Epidermal Melasma: Glycolic peel (35%-50%), tretinoin peel, SA peel, Glycolic-Kojic peel

   Dermoepidermal: TCA 35%

   Dermal: No peel (though theoretically we can offer a phenol peel)

6. Why do you think Melasma is not as common in males as in females?

   Various studies evaluating the hormonal profile in patients with melasma have found significantly increased levels of luteinizing hormone and lower levels of serum estradiol suggesting subclinical evidence of a mild ovarian dysfunction. Males with melasma tend to have low circulating testosterone .Melasma occurs in 10-15 percent of pregnant women and in 10-25 percent of women taking oral contraceptives. For decades, melasma was known as 'the mask of pregnancy," with the assumption that it must be caused by increases in female hormones due to pregnancy or the Pill. The reality is that we still do not clearly understand the hormonal link to melasma. Melasma skin is more estrogen-responsive than non-melasma skin. However other hormones involved in a woman's menstrual cycle and pregnancy, including progesterone and ?-MSH, can

also stimulate pigmentation. And just as melasma skin is more estrogen-responsive, it's also been shown to be more progesterone-responsive than normal skin.

7. How do you envisage melasma management 15-20 years from now?

The way cosmetology and lasers are making giant strides in management of various conditions probably they will be the frontrunners .We have a few new theoretical models of etiopathogenesis of melasma in the form of vascular theory, inflammation theory, genetic markers in melasma etc. Probably few new medical topical therapies would be unveiled by that time with mechanism specific to these pathways.

8. Can peels work standalone in a patient of melasma?

Superficial peels won't as they address only epidermal pigmentation and basically cause rejuvenation of skin due to exfoliation. Medium depth peels could work but as you know for dark skinned patients like ours, the recommendation is using very superficial and superficial peels. At any rate, you have to use topical agents for priming, so even if they work, it is better to give a combination. Peels are.

ACAD EXPERT SESSION

"Platelate Rich Plasma in Dermatology: Hair Disorders and Beyond"

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Dr. Pradeep Kumari

Dr. T. Salim.

(19/03/2017-03/04/2017)

Platelet Rich Plasma

1. Which is the cheapest method of PRP preparation?

   The cheapest method of preparation of PRP is byvacutainer tubes containing ACD. The disadvantage of this method is only if want more than 1-1.5 ml of PRP than you have to use more tubes as by 1 tube you can have only 1-1.5 ml of PRP The cost of each vacutainer tube is approx rupees 26/ only.

   Use double spin method for making PRP. First spin is 1200 rpm for 8 minutes. Then take out all plasma taking care of keeping minimal RBC. Second spin of this plasma at 2400 rpm for 8 minutes. Remove approximately 2/3 of upper portion and discard it. What remains is PRP.

   Alternatively, PRP KIT costing Rs. 200 having two 15ml tubes, two pipette and one anticoagulant vial can be used.

   Use 20G scalp vein set to bleed the patient.Under all aseptic precautions, take 30ml of venous blood in two 15ml centrifuge tubes. These tubes are pre filled with 1ml of ACD solution.Now centrifuge tubes at 1500rpm for 10 minutes to separate the blood in two components, bottom one of RBC and upper of plasma containing leucocytes and platelet.Now pipette out upper plasma till upper limit of red blood cell column in a plain centrifuge tube.This tube is then centrifuged at 3000rpm for ten minutes to give three distinct zones of upper plasma, middle layer of Buffy coat and few rbcs at bottom.Discard upper two third of plasma and remaining plasma is gently agitated to give a homogenous mixture of plasma containing platelet and leucocytes. Some amount of red blood cell are allowed in final L PRP.First soft spin is for 10 minutes at 1500 rpm, second hard spin is at 3000rpm for 10 minutes. By this method we get 5-6cc of L-PRP.

2. How does PRP differ from platelet concentrate?

   PRP should be freshly prepared. Platelet packs are days old. The most important is platelet yield prepared in a blood bank bag is not more than 6 lac per ml. Blood banks do not prepare concentrated PRP what we prepare in our clinic

3. Can allogenic platelets be used?

   PRP is exclusively autologous

4. What is the difference between vampire face lift and PRP?

   Technically both are same. When labelled as vampire facelift some people use additional modalities such as PRP with hyaluronic acid; threads; dermaroller; deep peel. Nothing is standard though; each doctor uses their own cocktail.

5. Is PRP worthwhile trying in acne scars?

It shows better efficacy in fresh erythematous acne scars when compared to old scars. Same goes for varicella erythematous scars. In old scars fractional co2 laser has better efficacy.

6. What's the role of photo activated PRP?

Studies have reported that photo-activation decreases proinflammatory cytokines (interleukin 2 and 6) and increases the concentration of leucocyte-derived anti-inflammatory factors (interleukin 1 receptor antagonist). However there are no comparative studies on PA PRP VS NON PA PRP.

7. How can we assess viability of platelets?

A simple way to find out if platelets have become dysfunctional is simply keeping aside 0.2-0.5 ml of PRP and then adding activator. IF the PRP clots then the platelets were viable. Growth factors are only released during clotting.

8. Is cold centrifuge required while preparing PRP?

There is no literature saying that any particular cold temperature/ centrifuge is better or in fact recommended to be used. (Only one Indian company promotes cold centrifuge for prp preparation) Known facts as of now are:

1. Hypothermia- even inside human body adversely affects platelet physiology and coagulopathies develop
2. Outside body almost all across world PRP is prepared at room temperature
3. For any live tissue- Process of reducing below 22 degree temperature and then taking it back to human body has to be done in a controlled manner otherwise subcellular damage is significant
4. Platelet concentrate is stored at room temperature unlike whole blood.
5. Cold stored platelets retain their functionality only for a day as opposed to room temperature/ temperature controlled to above 27 degree storage when post reperfusion functionality is up to 4 days.
6. In laboratory usage cold centrifuges are used for separation of subcellular organelles and not for whole cells.

9. What is activated PRP?

Activation of PRP is mostly by calcium chloride. It changes the pH to more acidic and is more painful to the patient. Activation converts prp to gel over next 5- 10 min and it is practically impossible to inject after 7 min post activation. So injector has to fast and still requires more force.If you choose to do activation do it in syringe, then take your chosen activator in the syringe and then take requisite amount of PRP in the same syringe. Mix and inject fast.

For all practical purposes, dermal collagen III activates platelets as soon as injected in skin so there is no need for activation.

ACAD EXPERT SESSION

"Genodermatoses"

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Co-moderator: Dr Tanumay Raichaudhary

(02/04/2017-18/04/2017)

Genodermatoses

1. What is the role of oral retinoids in the management of Xeroderma Pigmentosum?

   Oral retinoids definitely improve the texture of the skin, improve the morale of the patient and to a little degree decrease the incidence of cancers. However they do not serve as an alternative to photo protection, but something that augments synergistically your therapy. You have to be a very good counsellor when treating patients of XP, as even a slight improvement in skin texture due to retinoids, leads them to believe the disease is improving and they tend to experiment with going out in the sunlight.

2. What are the most important measures to be taken to prevent occurrence and recurrences of cutaneous malignancies in the patients of XP?

   Photo protection, period. Nothing else works and you have to tell them that photo protection does not only mean protection against the sun, but also fluorescent, mercury and xenon bulbs. They should get dark films installed in the windows of their rooms and vehicles. Wear large brimmed hats when going out, tight knitted clothes, gloves and wide dark glasses.

3. What is the role of genetic counseling in genodermatoses?

   Genetic counselling has an important role in any genodermatoses. It is common for patients to present with multiple children suffering from genetic skin defects. On telling them about the genetic nature of the disease and chances of recurrence, they always wonder why they were not counseled. And prevention via prenatal diagnosis and abortion if necessary is the only definitive treatment you can offer such families.

   Other than prenatal diagnosis, genetic diagnosis helps in cases like EB, PPK, XP and unknown disorders where knowing the genetic mutation can guide you towards a better understanding of the disease.

4. Is there Registry of Genodermatoses in India?? Do we do it?? If yes, where and how to approach??

   Definitely need to. Epidemiological data is the base for effective healthcare delivery. Without knowing what genetic defects are common in which region it is impossible to prioritize the genes which we need to standardize and offer for free/at reduced cost and/or go out in the field and counsel regarding consanguineous marriages etc. World over there is an increased focus on rare diseases, because they are not rare anymore and personalized medicine is on the anvil. We probably have the maximum number of cases in our country, how we utilize that clinical data depends on forming an effective registry with a large network of doctors and scientists collaborating with each other.

   IADVL can take the lead, a good software is not very expensive, and we can offer incentives at least initially, as to free genetic tests, names in publications etc.

   The other way would be that individual doctors and centers start specialized registries and then expand to more centers over a period of time.Any effort to start a registry would be immensely

beneficial, eventually helping us to be leaders in genodermatoses due to the sheer amount of data we will be able to generate.

The centers working on genodermatoses are:

CHG, Bangalore - EB, with outreach centers and teledermatology

PGI-chandigarh - Ichthyosis and EB - with special surgeries for ichthyosis done free of cost.

SGPGI-Lucknow - Muskuloskeletaldysplasias

National Institute of Research in Reproductive Health - Mumbai

Dr. DY Patil, Pune- Tuberous sclerosis and Neurofibromatosis.

Private companies which sequence genomes areCentogene and medgenome.

5. A child comes with a set of anomalies that I can see but I can't fit them into a syndrome I know. How should I approach?

First of all, when should be suspect GD

" Onset of skin lesions at Birth or at Early Age

" History of Similar illness in Family (Parents,Siblings, first or Second degree Relatives)

" Unexplained Skin, Hair, Nail, Teeth changes associated with or without systemic complaints

" Once we suspect GD, we should ask few important questions like

" skin lesions (onset, duration, evolution, progression, distribution and arrangement)

" Does it follow Blaschko's Lines?

" Erythroderma, Photosensitivity, Xerosis and about Hair, Nail, Teeth, Palms/ Soles

" Systemic features such as delay in Developmental milestones, Convulsion, Vision, Speech, Hearing, H/o musculoskeletal complaints, Infections, Dysphagia, Dyspnea etc.

Second important part is to know about OMIM (Online Mendelian Inheritance in Man). It is a continuously updated catalogue of human genes and genetic disorders, with particular focus on the molecular relationship between genetic variation and phenotypic expression.

One has to go to PUBMED and there is a dropdown box with OMIM as an option.

Please write key features in the search bar without adding comma (,) and with every addition of the feature, one keeps narrowing the diagnosis.

6. Parents present with history of two children who have already expired in the first three months of life. Now they have a third with blistering skin disease. What should I tell them?

I will presume a fatal disease to be recessive one; may be DEB or JEB.

It is ideal to do genetic counselling and it's a process that

1. Starts with making correct diagnosis
2. Confirming the diagnosis
3. Informing and educating family, importantly parents
4. Spelling out Rx and future plans
5. Educating about risk for recurrence
6. Last but not the least offering preventive measures wherever possible

It is vital to go for the gene testing of the affected baby and if possible of the parents.

Counsel parents to go for prenatal diagnosis during next pregnancy. It is easy and more economical if the gene mutation has been identified beforehand from the earlier baby or parents.

7. Are there options for fetal radiography like high frequency ultrasound which would be non-invasive but contributory?

The major benefit of ultrasound is its ability to detect abnormalities in the absence of any family history. However it still remains non confirmatory and most features can be visualized beyond 20 weeks of gestation, the current legal limit for termination of pregnancy.

In the case of harlequin ichthyosis, routine prenatal ultrasound may reveal echogenic amniotic fluid, large joint contractures, digital contractures, and facial dysmorphism.

TSC is another excellent example where cardiac rhabdomyomas can be seen in upto 2/3rds of newborns.

USG features of other syndromes described include ectodermal dysplasia, Noonan syndrome, Mandibuloskeletal dysplasia and other syndromes associated with structural abnormalities like CHILD syndrome etc.

Chorionic Villous Sampling (CVS) can be done at 11-15 weeks and Amniocentesis during 16-20 weeks may be undertaken. If the disorder is AR, clinical examination of parent generally doesn't yield any information about the disease so one of the parents usually mother can go for mutation analysis too.

If the disorder is AD, a good clinical examination of both parents may reveal some clue to the GD.

In addition, one can also keep in mind the possibility of spontaneous mutation. A clinical geneticist should be involved.

Coming to high frequency USG, though non-invasive it doesn't give significant information even in most expert hands till late 2nd trimester and thus may not be amenable to termination.

GD's that can be picked up/ suspected for the first time includes;

EEC- by making out ectrodactyly, cleft palate

Aplasia Cutis congenita

EB (DEB) is suspected in patients with pyloric atresia (hyper-distended stomach with polyhydroamnios)

Harlequin fetus- by everted lip and persistently open mouth, eclabiumetc

In nutshell; CVS and Amniocentesis are indispensable tools and should be utilized in association with clinical geneticist.

8. Please elaborate about genetic counselling. Should we do it or refer the patient to counsellor?

If the services of a counsellor are available, then you can always refer the patient to them. However, at present, trained genetic counsellors are not readily available.

In general, a genetic counseling session aims to:

" Increase the family's understanding of a genetic condition

" Discuss options regarding disease management and the risks and benefits of further testing and other options

" Reduce the family's anxiety

At a minimum, it should include a pre-testing and post-testing session

For genetic counselling, you have to understand the genetic disorder first whether the genetic disorder is chromosomal, microdeletion, single gene or polygenic. The most important step for genetic diagnosis is pedigree drawing (preferably 3 generations)

This gives a fairly good idea of the type of inheritance and once that is known, the most important question of the family, that is what are the chances of a child being born with a genodermatoses running in a family or chances of recurrence.

For autosomal recessive disorders where both parents are carriers - 25% chance of affected baby each pregnancy

For autosomal dominant disorders with single parent affected - 50% chance of affected baby each pregnancy

For autosomal dominant disorders with both parents affected - 75% chance of affected baby each pregnancy.

X linked disorders - females usually are carriers, as affected ones normally expire in utero

Mitochondrial disorders - pass from females to females.

Polygenic disorders (the most common query being vitiligo): It is currently not possible to do prenatal diagnosis of any such disorders.

The second step is to discuss with the family the course of the disorder, the fact that it is genetic and it is incurable at present. Patient friendly and disease specific handbooks really help, as do patient groups.

The third step entails laying down all current treatment options of varying costs and if possible consulting with multiple specialists to form a plan of action. It is imperative to not get tests like karyotyping done for clearly monogenic disorders. Major companies do offer large discounts for patients suffering from rare diseases as they require a lifetime supply of it.

Then you can offer services like next gen sequencing, panel testing or other available testing procedures and discuss each test's limitations. Reproductive options and various options of prenatal diagnosis available along with their costs involved should also be discussed. If the genetic test is positive, testing may be considered for additional relatives of the individual. Genetic counseling referrals for other family members for risk assessment may be discussed

9. Which tests should be done for prenatal diagnosis?

The techniques available for prenatal diagnosis include:

" Fetal skin biopsy at 15 - 27 weeks of gestation. It is only used when the causative gene not yet elucidated or when specific mutation in the causative gene not identified. However it needs to be performed late in gestation and an abortion may not be possible after 20 weeks without legal recourse. It is also not definitive and can lead to fetal scarring or fetal loss.

" Amniocentesis at 15-20 weeks and Chorionic Villous sampling at 10-14 weeks are widely practiced and can give a definite molecular diagnosis if the specific mutation in the family has already been identified. This is important, as Indian data is not yet robust enough, direct panel testing of the fetus will not be foolproof and Next gen sequencing will probably be limited by the time constraint.

" Pre-implantation genetic diagnosis is done prior to implantation of blastocyst. It is extremely costly, however it eliminates the need terminate an affected fetus and is advantageous for couples having trouble conceiving.

" Ultrasonography, better visualised after 18- 20 weeks which serves as its major limitation.

" Maternal serum screening can detect X-linked ichthyosis associated with low levels of unconjugated estriol, however further confirmatory prenatal diagnosis suggested.

10. What are the practical problems in trying to create a registry? Is my patient confidentiality maintained and when I include a case in the registry, what are my rights when there is use of that data? Who all can access the data with the registry? What are the other benefits to our patients if we have a registry?

Firstly, India is a huge country with a large population. So it is almost impossible to cover every part of the country. Also we are so diverse in our genetic constitution and hence it is important to cover as many parts of the country as possible. So with these 2 limiting factors, whatever data we get is somewhere close to establishing a registry if it not completely.

Second, sharing of data and end use of data- We need strict guidelines on how the data is share and used by dermatologists across the country.

Third- Patient confidentiality- This is of utmost importance as we usher into the era of consumer rights. It is not just discussion between professional on various media but also to keep information confidential from insurance companies and pharmaceutical industry who would be more interested in such information.

Costs- Since the land area and population is huge to get meaningful data of both clinical and molecular information, costs are huge.

Regarding data access, information has to be shared so that patients are benefited but at the same time guidelines have to be laid and non-disclosure agreement has to be signed before sharing any data of patients.

Benefits- This gives a wealth of information into understanding the disease manifestations in our population. Registry will help to define the extent of problem related to particular genodermatoses in our region so that in addition to providing an appropriate diagnosis, identification of candidate gene, genetic counselling, it will help in future to form Support Groups for these patients & objectives of these Support Groups then can be tailored according to our patient's needs.

11. Would standardising genes help in management of a disease like Tuberous sclerosis or neurofibromatosis in any manner?

There are a number of scenarios where standardizing genes for diseases like TSC or Nf will help. The most important being prenatal diagnosis. If done for a large enough sample, the standardized genes can jump start the work of finding the specific mutation in the affected parent, without going through the lengthy and costly process of whole exome sequencing. If the mutation is found with your standardized test, then the prenatal diagnosis becomes easy. The second scenario is when a child presents with isolated cafe au lait macules, or confetti lesions and you want to be sure whether the child will go on to develop nf or tsc. The third advantage could be genotype phenotype correlation. If are able to pinpoint specific mutations with specific manifestations of the disease, then we can start looking for them early which immensely helps in the management of the disorders.

What additional benefits our patients will get from registry, I will like to add: This will also help to provide them an updated insight into their genodermatoses based on recent literature, available treatment options & future outlook like gene therapy.

ACAD EXPERT SESSION

"Contact Dermatitis and Patch Testing: Relevance in Indian scenario"

Dr A. K. Bajaj

Dr Hema Jerajani

Dr Sanjeev Handa

Dr Iffat Hassan

(24/04/2017-04/05/2017)

Contact Dermatitis & Patch Testing

1. What is the clinical significance/relevance/usefulness of atopic patch test in routine dermatology?

   The role of allergy in eliciting and maintaining an eczematous skin lesion is controversial. However there is another line of evidence for the role of aeroallergens and food allergens (in children) found to be relevant in atopic dermatitis. An epicutaneous patch test with allergens known to elicit IgE mediated reactions and the evaluation of eczematous skin lesions after 48-72 hrs (Atopy patch test) can be used as a diagnostic tool in characterizing patients with aeroallergen triggered atopic dermatitis. In patients with an air exposed eczema distribution pattern, positive Atopic patch test reactions occur at lower doses compared with other patients with Atopic Dermatitis.

2. As AD is a Th2 mediated disease and ACD is a Th1 mediated phenomenon, the presence of either should practically decrease the possibility of other. Is it practically so?

   Atopy and contact allergy seem independent, while there is insufficient data to state upon the relationship between Atopy and allergic contact dermatitis. Data from available studies are insufficient to state upon the relationship between Atopy and allergic contact dermatitis, however, it is apparent that both conditions frequently coexist.

3. Is there a possibility of inducing sensitization with new allergens while patch testing in a predisposed individual?

   Yes there is a possibility of inducing sensitization with new allergens while patch testing in a predisposed individual. Sensitization is commonly produced by p-phenylenediamine, thiuram mix, epoxy resin, sesquiterpene lactone mix, primula extract and in recent years isothiazolinones or acrylates. The risk however is uncommon when testing is performed according to internationally accepted guidelines.

4. Is there any age criteria above which patch testing can be safely carried out? What special points need to be considered while patch testing in pediatric population?

   Contact testing seems to be as important in children (even those aged under 3 years) as in adolescents, since contact sensitization occurs in every age group, though sensitization frequencies vary according to the specific age group.

   Patch testing in children is safe, the only problem being technical because of small patch test surface. It is usually advised to use Finn chamber. Reinforcement of patch test units is suitable due to hypermobility of children. There is a general consensus of using the same concentrations as in adults. Irritant reactions are common in children but when in doubt the clinician is advised to retest with a lower concentration.

5. I take my readings at 48-72 hours usually. Is there a quicker way, like stripping the epidermis with adhesive tape since in humid months which is almost 7-8 months in a year, patients find it difficult to keep the patch strips on the back?

Keep the patches in place by using extra tape as needed, if the patches fall off completely, leave them. An effective way is to reinforce strips of plasters on the edges of the patch test tapes. The conventional skin marker does not remain on the skin due to perspiration. The silver nitrate skin marker is a useful marker for identifying patch test sites.

The following instructions may be given to patients:

Patients will be allowed to continue to take light showers or bathes to clean their face, chest, limbs and lower torso. They should avoid washing the back with water.

The back where the patch test tapes are placed will be allowed to be cleaned daily with light moist towels, avoiding the test strip area.

Patients should avoid outdoor activities and remain in a cool air-conditioned environment whenever possible.

6. Which are the kits available in with their costs and availability?

INDIAN STANDARD BATTERY (20 Antigens) 4,800.00

FOOT WEAR SERIES (15 Antigens) 4425.00

COSMETIC SERIES (32 Antigens) 8,000.00

available from Systopic Laboratories Pvt. Ltd.

7. When is the situation that you cannot afford not doing patch tests?

1) For patients with a diagnostic hypothesis of CD,

2) Patients with other skin conditions that may be aggravated by CD (atopic dermatitis, seborrheic dermatitis and stasis, nummular eczema, psoriasis, and dyshidrosis),

3) Patients which chronic eczema without an established etiology,

4) Suspected cases of occupational contact dermatitis

5) Chronic eczema of hands and feet

6) Eczematous disorders failing to respond to treatment as expected.

8. Few so called allergy specialists inject some costly vaccines in the suspected patients of ABCD/ ACD/ Parthenium Dermatitis. Is it some scientific therapy of some form of quackery?? Patients are charged heavily for these so called vaccines but none improves.

Sublingual Immunotherapy (SLIT) has been tried in parthenium dermatitis with limited cases in the literature - Jerath V P, Jerath P, Sood M, Nishchal R. Immunotherapy in parthenium dermatitis. Indian J Allergy Asthma Immunol 2014; 28:83-5.

There are also two reports in the literature about hyposensitization being carried out in patients of ABCD - Handa S, Sahoo B, Sharma VK. Oral hyposensitization in patients with contact dermatitis from partheniumhysterophorus. Contact Dermatitis 2001;44:279-82, Srinivas CR, Krupashankar DS, Singh KK, Balachandran C, Shenoi SD. Oral hyposensitization in parthenium

dermatitis. Contact Dermatitis 1998;18:242-3. Srinivasan et al tried oral hyposensitization in a patient with parthenium dermatitis using crushed leaves; however symptoms of the patient recurred on discontinuation of the therapy. In the second study by Handa et al, 50% patients had good clinical improvement and some had exacerbation as immunotherapy was stopped.

However, there are also studies on vaccine induced allergy - Lidholm AG, Bergfors E, Inerot, Blomgren AU, Gillstedt M, Trollfors B. Unexpected loss of contact allergy to aluminium induced by vaccine. Contact Dermatitis. 2013; 68(5): 286-292.

9. What is the opinion of experts in using Aluminum Hydroxide topically to alter the immunity of the skin in the hands of the patients of chronic hand eczema?

It has been reported that Aluminium magnesium hydroxide stearate barrier cream applied 2 - 3 times daily relieves the burning and itching experienced with various types of dermatoses, including atopic dermatitis, allergic contact dermatitis, and irritant contact dermatitis - Del Rosso JQ1, Bhambri S, Michaels B. An Aluminum Magnesium Hydroxide Stearate-based Skin Barrier Protection Cream Used for the Management of Eczematous Dermatitis: A Summary of Completed Studies. J ClinAesthetDermatol. 2008 ;1(4):18-21.

10. Are there some effective barrier creams to prevent the exposure to allergens and also are there any particular instructions for patients to help avoid exposure other than avoiding that work as it is difficult for laborer?

Barrier cream and ointment are available by praise pharma.

Barrier creams are many types depending upon the purpose you are looking for.

Even a white soft paraffin/ liquid paraffin applied before a mild contact can work protectively. It may help a house wife against cutting vegetables.

Regarding Industrial contact allergens, the barrier creams provide protection to some extent by water resistant creams (w/o emulsions). They also give protection to water soluble substances such as mild acids, alkalies and metal working fluids. The water repellent cream leaves film containing ethyl cellulose, lanolin, wax, and silicone on the skin

The oil resistant creams are oil-in-water emulsions and protect the skin against irritants which are hydrophobic greases, solvents and, paints & varnishes.

The barrier creams are somewhat protective towards epoxy resins, metals, paints and cutting oils. But it also depends on the duration for which you may to have to apply. Short contacts are manageable but long contact is difficult to manage and needs frequent application of barrier cream

The ingredients in barrier cream are tanning agents, Aluminum Chlorhydrate, perfluorpolyethers, zinc oxide, chelating agents, tartaric acid and glycine. The most popular Indian cream is Rebask from Dr Reddy's laboratories which contains ceramides, cholesterol, sqalene, dimethicone, glycerine, sodium hyaluronate, and white soft paraffin. You have to remember that you will have to clean the hands after the work with vegetable oils to dissolve the product.

Overall, the shorter the work better is the efficiency of the protective cream. Many authors are disappointed with overall efficacy of barrier cream for long term protection

Gloves can be protective but when used for periods, increase the chances of sensitization

A good thing is some laborers get hardening effect and tolerate the allergens well. It must be said, the protective measure are for all times and always.

11. How can cement related contact dermatitis be mitigated?

The best way to prevent cement-related skin problems is to minimize skin contact with cement. Compliance with good skin hygiene and work practices listed below, will protect against hazardous contact with cement.

Provide the proper gloves for employees who may come in contact with cement. Butyl or nitrile gloves (rather than cotton or leather gloves) are frequently recommended for caustic materials such as cement.

Use only well-fitting gloves. Loose-fitting gloves let cement in. Often the use of gloves and clothing makes exposure worse when cement gets inside or soaks through the garment. Use glove liners for added comfort. Wash hands before putting on gloves. Wash hands every time after removal of gloves. Dry hands with a clean cloth or paper towel before putting on gloves. Protect arms and hands by wearing a long sleeve shirt with the sleeves duct-taped to gloves to prevent wet cement from getting inside the gloves. Follow proper procedures for removing gloves, whether reusing or disposing them.

Steps for safe glove removal:

Wash off the outside of gloves while still wearing them.

Loosen gloves on both hands, holding arms down to prevent water from dripping onto the skin.

Holding arms downward, pull the first glove down to remove only the glove fingers. The cuff should still be covering the palm of hand.

Remove the second glove by grabbing it with the first glove.

Slip off the first glove.

12. How to treat chronic and recurrent cases of Parthenium Induced ABCD? How to prevent relapse?Which is the agent of choice for long term maintenance therapy, MTX or AZA?

i) Azathioprine is effective in the treatment of Parthenium dermatitis at the dose of 1-2 mg/kg/day. Various regimes have been tried such as daily doses of 50-150 mg with/without 300 mg monthly bolus doses. Weekly azathioprine therapy (WAP therapy) 300 mg/week was found to be as effective as daily treatment with azathioprine with better compliance and reduced cost of therapy. The treatment may be continued for 6-12 months after subsidence of disease. Its major limitation is that azathioprine takes 4-6 weeks to exert its action. It is preferred for the treatment of chronic stage, and it should be supplemented with corticosteroids in the beginning during the management of acute stage.

ii) Combination/sequential therapies have been suggested, which consists of giving (cyclosporine + azathioprine) × 4-6 weeks followed by daily azathioprine × 6 weeks and then weekly azathioprine as maintenance for 2-5 years.

iii) Oral hypo sensitization means that an antigen is introduced into the body by a route different from natural one to induce such a change in the immune system that the body does not develop clinical manifestations when antigen is introduced into the body through normal route. It is thought to act by causing depletion of memory T-cells. Oral hypo sensitization was demonstrated to be effective in the 1950s for ragweed dermatitis, but has not been widely accepted because it carries considerable risk of provoking and worsening eczema. The results of oral hypo sensitization with Parthenium leaf are not consistent, and continued therapy appears to be necessary. Handa et al evaluated the effect of oral hypo sensitization as an alternative therapeutic modality in 24 patients of Parthenium dermatitis. In 70% of those patients who completed the study, there was a gradual improvement in their clinical status and 30% of patients had an exacerbation during the course of the study. Patients tolerated therapy well and no significant side-effects were seen, except for abdominal pain, 'heartburn,' and cheilitis. Handa S, Sahoo B and Sharma VK. Oral hypo sensitization in patients with contact dermatitis from Parthenium hysterophorus. Contact Dermatitis 2001;44:279-82.

iv) Immunotherapy with recombinant protein is administered in cases where patients are co-sensitized with several unrelated pollen allergens. Based on frequent co-sensitization patterns, some of the hybrid proteins have been evolved with the polymerase chain reaction. These hybrids contain all the epitopes from the different allergen in a single protein. These have been used for vaccination against pollen allergy. It has been reported useful in hay fever and allergic rhinitis and is under trial for use in ABCD.

v) Prevention of relapses -Azathioprine or other adjuvants like methotrexate or cyclosporine can be used in maintenance doses. Protection can be given by covering the exposed parts, removal of the agent from the environment, or removal of the patient from the contaminated environment and desensitization methods.

De et al in their study comparing the efficacy of methotrexate vs azathioprine in the treatment of Parthenium Dermatitis -(De D, Sarangal R, Handa S. The comparative efficacy and safety of azathioprin v/s methotrexate as steroid-sparing agent in the treatment of airborne-contact dermatitis due to Parthenium. Indian J Dermatol Venereol Leprol 2013;79:240) concluded that both azathioprine and methotrexate are effective and safe steroid-sparing agents in the treatment of parthenium-induced ABCD, methotrexate helps in achieving an earlier control of the dermatitis and is a cheaper alternative to azathioprine.

13. What is"Angry back" syndrome or "excited skin" syndrome?

It is defined as false-positive patch test (PT) reactions usually adjacent to large true-positive reactions that induce contiguous skin inflammation and irritability. If a patient has a large number of positive patch test reactions, retesting the patient sequentially to a small series of these allergens may be necessary to exclude nonspecific false-positive reactions. The syndrome most likely

occurs in individuals who have active dermatitis at the time of patch testing or who have a strong positive patch test reaction, both of which may induce local skin hyperreactivity in the area where patches were applied.

14. What is Baboon Syndrome?

It is an allergic skin condition which results in redness of the buttocks, upper inner thighs and armpits. The condition can occur as a hypersensitivity reaction to ampicillin, nickel and mercury. It is also called as symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) and is known as baboon syndrome because the rash on the patient's buttocks resembles the red hindquarters of some monkeys. Baboon syndrome typically appears a few hours to two days after exposure. The syndrome rarely affects small children, but cases have been reported in an 18-month-old baby and a 5-year-old child. Recovery can sometimes take up to three weeks. Although exposure to penicillin, nickel or mercury are the most common causes of the syndrome, but it has also been linked to certain proton pump inhibitors, biological agents and chemotherapy.

ACAD EXPERT SESSION

"Leprosy, Reactions in leprosy, Resistance and Management"

Dr . P. Narsimha Rao

Dr H. K. Kar

Dr P.V.S. Prasad

Dr V. V. Pai

Dr Sunil Kumar Gupta

(21/05/2017 - 07/06/2017)

Leprosy, Reactions in leprosy, Resistance and Management

1. Leprosy in year 2017- The claims of eradication apart, where are we as of today??

   According to official reports received from 138 countries from all WHO regions, the global registered prevalence of leprosy at the end of 2015 was 176 176 cases (0.18 cases per 10 000 people). The number of new cases reported globally in 2015 was 211 973 (0.21 new cases per 10 000 people). In 2014 the number of new cases reported was 213 899, and in 2013 the number of new cases reported was 215 656.

   The number of new cases indicates the degree of continued transmission of infection. Global statistics show that 203 600 (96%) of new leprosy cases were reported from 22 priority countries.

   In India if we see the total number of reported cases of leprosy from year 2010 to 2015, it is increased from 126800 to 127326(60% of the global new cases). But in fact the leprosy prevalence in India is just tip of iceberg on record.

2. Drug Resistance in Leprosy : claims v/s facts

Ans. The recent mapping of the M. leprae genome has identified sites at which mutations occur, conferring resistance to dapsone, rifampicin and the quinolones. Rifampicin binds the beta-subunit (coded by the rpoB gene) of the RNA polymerase and certain mutations in the rpoB gene lead to rifampicin resistance in M. leprae and M. tuberculosis. Missense mutations leading to the substitution of any one of the following positions (positions 407, 410, 416, 420, 425 and 427) or an insertion of amino acids between position 408 and 409 confers rifampicin resistance to M. leprae. Missense mutations in the sulphone resistance determining region of the folP gene (codes dihydropteroate synthase), resulting in alterations of amino acids at positions 53 and 55, confer dapsone resistance to M. leprae. Missense mutations in the gyr A gene at position 89, 91, 92 and 95 are correlated with ofloxacin resistance but there is no cross resistance so the fixed dose MDT is helpful always.

3. Management of ENL. Thalidomide v/s other immunosuppressant??

   ENL is a very serious reccurring reaction in lepromatous leprosy even after completion of MBMDT therapy. No doubt thalidomide is the miracle drug when used in this situation but this drug has its own limitation of high cost and teratogenecity in pregnant women. In such situation the judicial use of oral prednisolone and other immunosppressive drugs like methotrexate are used.

4. Management of Resistant cases of Leprosy

   All MB relapse cases included in the surveillance study should immediately be put on treatment with standard MB-MDT without waiting for the results from the reference laboratory on the status of drug resistance. If the result comes back as sensitive to rifampicin, MB-MDT treatment is to be continued accordingly. For patients who are reported to be resistant to dapsone only, standard MB-MDT can be safely continued. In case the reference laboratory reports that the

patient is harbouring rifampicin resistant M. leprae the following treatment should be given:- o administration of 50 mg of clofazimine, together with 400 mg of ofloxacin and 100 mg of minocycline, daily for six months; o administration of 50 mg of clofazimine, together with 100 mg of minocycline or 400 mg of ofloxacin daily for at least an additional 18 months. The above-mentioned treatment regimen is also to be used for patients reported to be harbouring both rifampicin and dapsone resistant M. leprae.

5. Drug Resistance in Leprosy: claims v/s facts:

The biggest hurdle in the management of leprosy is that of non-compliance. The single most contributory factor for development of resistance as Primary resistance in leprosy is rarer than Secondary Resistance.Acknowledging the growing concern of drug resistance in leprosy, the WHO issued guidelines for the global surveillance of drug resistance in leprosy using PCR-direct sequencing of M. leprae from patients with characterized relapse from MDT.

These guidelines included:

1) DNA isolation from skin biopsy of MB relapse patients using DNeasy Kit (Qiagen, Germantown, MD);

2) PCR amplification of the appropriate target DNA fragments containing DRDRs of M. leprae using specific primers

3) Automated DNA sequencing of these fragments with both forward and reverse primers; and

4) Alignment of generated sequences to that of reference DRDR sequences in the M. leprae TN strain (NC002677GenBank) to determine the presence of drug-resistant mutations.

Facts about Drug Resistance:

Global and Indian magnitude remains: unknown (few rare published case reports)

How to improve the current scenario:

1. Proper case documentation

2. Proper documentation of clinical features and neural involvement and sensory loss at baseline to avoid confusion in future between Relapse v/s Resistance.

6. What are the standardized regimens of drugs other than classical MDT for both PB and MB leprosy(quinolones and macrolides): especially needed when drug reactions occur to the classical MDT.

For PB- o administration of 50 mg of clofazimine, together with 400 mg of ofloxacin and 100 mg of minocycline, daily for six months;

For MBo administration of 50 mg of clofazimine, together with 100 mg of minocycline or 400 mg of ofloxacin daily for at least an additional 18 months.

The above-mentioned treatment regimen is also to be used for patients reported to be harbouring both rifampicin and dapsone resistant M. leprae.

Above mentioned regimens are only used in specific conditions only. Theoretically the chance lepra reactions are less as these drugs are not as efficacious as first line drugs and second the macrolides and minocyclin both have very good anti-inflammatory effect.

7. How can we replace Dapsone from MDT if someone is sulpha sensitive or G6PD Deficient???

Dapsone can be replaced with ofloxacin/minocyclin in sulpha sensitivity (though there is no cross sensitivity to other sulpha drugs but do not take risk) and in G6PD deficient patients.

8. ART and MDT - How should one proceed?

Leprosy has now been reported presenting as immune reconstitution disease among patients commencing HART so MBMDT must start simultaneously. Remember that cell mediated immune response to M leprae are preserved at the site of disease despite evidence that these responses are abrogated systemically-a paradox. So HIV infection and ART does not affect leprosy or its management.

9. Pregnancy(both early and late) and Leprosy?

The pregnancy status is associated with a relative decrease in cellular immunity which allows M. leprae to proliferate. This results into a high rate of relapse of the disease and reactional states during pregnancy. Type 1 reactions maximally occur post partum when cell mediated immunity returns to pre-pregnancy levels and Type 2 reactions may occur throughout pregnancy and lactation. Cases of silent neuritis are also seen predominantly 6-9 months after delivery, especially in patient with borderline leprosy. MDT is the treatment of choice. Systemic steroid is given to manage reaction and silent neuritis. Leprosy has been associated with increased fetal mortality, prematurity and low birth weight and risks proportional to the bacterial load. Approximately 20% of children born to mothers with leprosy will develop the disease by puberty.

10. Why LL (MB) patients are now more in India?

It could related to the epidemiology of leprosy as a disease. While the disease was waning in parts of the world it was noted that as the number of patients starts decreasing the percentage of multibacillary forms increase. In India, (in contrast to Africa), the PB percentage was higher (almost 70% before 2000). As the leprosy load decreased, the MB patient's percentage increased. (From 30% in 1980s to > 53% now). It also probably demonstrates that there is a population sub-group which is selectively unable to clear lepra bacilli which needs attention.

11. What is the present status of 12 pulses of ROM vs 12 months of MB-MDT?

The regimen of single dose ROM for single lesion paucibacillary leprosy was a recommendation of the WHO 7th Expert Committee on Leprosy of 1998. For some time ROM was available in blister packs in India, Bangladesh, Nepal and Brazil and used quite widely, particularly in India where at the time, the 7th Expert Committee reported that more than 50% of newly detected cases were classified as single lesion PB leprosy. However it was quietly withdrawn by the year 2004. In WHO Global Strategy 2006-2010 and the WHO Enhanced Global Strategy 2011-2015,

no recommendation is made regarding the use of ROM and it is not included in the recommended regimens in the operational guidelines of both those strategies.

Now, regarding ROM use in MB leprosy there are few studies, mostly from South America, which found it to be as effective or superior to MB MDT. But note that they are just isolated studies and the observations still to be validated / accepted by leprosy authorities. It is not a recommendation. http://journal.fk.unpad.ac.id/index.php/amj/article/viewFile/955/899. The drawback is that there was no followups beyond 12 months. .

12. Before stopping MDT after one year, in a teaching institute, should we do MI/BI and stop therapy on the basis of the results or stop therapy irrespective?

We must assess the BI/MI in all patients before initiation of MDT in teaching institute. Over 99% live bacilli get killed from MDT. Thereafter BI reflects only dead bacilli. As dead bacilli can be cleared from the body by natural mechanisms of the host, BI in skin smears start falling after one year of MDT as 0.6-1.0 log per year and continue even after stopping the treatment. On the contrary MI falls to 0 within 5-6 weeks following MDT. So if the patient is improving after the initiation of therapy till end of therapy then there is no need of further assessment of BI/MI during RFT. But we can do this if there is no clinical improvement due therapeutic failure, in relapse/resistant and in non-compliance. There are also some other method to assess the activity of disease/viability of M. leprae like ACE level, ATP content in infected tissue and Fluorescein diacetate-ethidium bromide(FDA-EB) staining.

13. What is the last line of therapy in resistant ENL cases who have failed thalidomide, azathioprine, methotrexate, colchicine, clofazimine and dcp in various combinations?

Non response to so many combinations you mentioned is rather rare, unless the therapy principles were not properly applied. Having said that, in rare such cases the following areas have to checked and attended again.

1. Check for intercurrent infection focus as it is usually hidden in patients on steroid therapy. (Usually in lungs kidneys, skin). Once found, treat with full course of therapy.
2. Restart MB MDT, if not already on. Increase dose of clofazimine to 200 and later to 300 mg/day. Note that it takes 3-4 weeks for this dose to be effective for reactions.
3. Start Thalidomide if not contraindicated. Always add analgesic in max tolerable doses.
4. Provide Zn and vitamin B-complex supplements.
5. If ulcers are present, dress and treat them as any infection can stimulate/keep the ENL going.
6. If patients is already on oral steroids, try to taper them over 6 weeks to a low dose 15-20 mg/day. Later one may try to stop them completely.

ENL breakouts will happen while on therapy which need minor/major adjustments. Inform patient that control may take about 6-9 months.

14. What is the reason for hypopigmentation in leprosy?

It is due to block in the transfer of melanin from melanosomes, direct destruction of melanocyte by phenolic compound, decreased melanocyte activity by o-diphenoloxidase enzyme, ischemic changes due to vascular inflammation and utilization of DO PA substrate by Mycobacterialeprae enzyme system have been speculated as basic factors for the cause of hypopigmented macular lesions in leprosy. Post inflammatory hypopigmentation also contributes.

15. How can IADVL contribute in acquiring authentic data on epidemiology of leprosy in India?

SIG Leprosy is coming up with a DermLep Study to find out the actual incidence of leprosy in India. Further details can be sought or you can confirm your participation in survey by email on sigleprosysurvey@gmail.com. This will be possible if all the dermatologists associate themselves in this endeavour to help in gathering the required data and information.

16. How are nerve conduction studies helpful in pure neuritic form? How to correlate them in the prognosis of the disease and can they replace biopsy?

There is diffuse neuropathy (neuritis) in early stage (even in pure neuritic form when it cannot be detected by routine clinical testing. Nerve conduction velocity decreased before any sensory deficit appears. Nerve conduction studies are reliable diagnostic and prognostic indicators useful in leprosy especially in areas that are endemic for the disease like ours. But when we compare it with nerve biopsy then nerve biopsy is the ideal for the diagnosis of clinically manifested pure neuritic Hansen disease.

17. Ideally thalidomide should be started only after tapering down steroids. How is it practically possible?

When we give oral steroid or thalidomide there is increased risk of thromboembolic phenomena in susceptible persons like bed ridden, obese and in concomitant comorbidity as both drugs are thrombogenic. Regarding management of ENL first we classify the patients in different categories like:

a) Can afford thalidomide and there is no contraindication to drug- give Thalidomide in standard dose as it is the best one in terms of response.

b) Cannot afford thalidomide/contraindication to thalidomide- Such patients are put on oral steroid and other immunosuppressive drug therapy.

c) If patient is on oral steroid from beginning and not responding to it but afford thalidomide and there is no absolute contraindication to thalidomide- In such situation there should be fast tapering of oral steroid with addition of standard dose of thalidomide done but in the same time add oral aspirin and careful watch for coagulation profile and early sign/symptom of DVT and try for early mobilization of patient in ward.

18. What is the practical utility of ultrasound examination of nerves? How does it guide the treatment process?

Imaging of peripheral nerves directly observes structural changes in the nerves objectively using a special superficial probe of frequency of 10-14 MHz for accurate assessment. Normal peripheral nerves in US are seen as markedly echogenic tubular structures with parallel linear internal

echoes on longitudinal sonograms with round to oval cross section on transverse scans with occasional internal punctuate echoes. High resolution US allows complete analysis of median, ulnar, posterior tibial nerve and studies have shown that the site of maximum involvement was limited to 6-10 cm above the flexor retinaculum for median nerves, 8-12 cm proximal to the cubital groove for ulnar nerves, and 4-10 cm proximato the medial malleolus for posterior tibial nerve.

In addition, Color Doppler (CD) imaging of each nerve can be performed to look for absence or presence of blood flow signals in the peri-neural plexus and inter-fascicular vessels of nerve trunks. The increased blood flow signals seen in CD in thickened and tender peripheral nerves of leprosy supports the theory that they reflect the edematous and hyperemic changes secondary to the inflammation leading to alteration of an effective blood-nerve barrier during reversal reactions.

Clinical applications of HRUS in leprosy are:

1. In leprosy, routine HRUS scanning of commonly involved nerves in patients of leprosy provides useful information on their state and extent of involvement. This is an objective assessment of nerve involvement in a suspected case of leprosy. It is of medico-legal value as well.

2. Significant correlation was observed in published studies between clinical parameters of grade of thickening, sensory loss and muscle weakness and US abnormalities of nerve echo texture, endoneural flow and CSA of nerves.

3. Follow-up USG imaging of nerves helps to know the changes in the structural integrity which could be correlated directly to treatment efficacy and clinical improvement.

4. HRUS provides documentable proof of nerve pathology which would be a welcome addition to the existing tests for nerve function impairment.

5. Besides enlargement, structural abnormalities such as integrity of fascicular structure, type of enlargement, edema and state of neural vascularity of nerves in leprosy patients can be studied by USG.

6. Increased blood flow signals indicative of active inflammation in HRUG in nerves can be correlated to severity of neuritis, especially during reactions.

19. What are the indications for nasal scrapping in a case of Leprosy?

Initially nasal smear was used for the diagnosis of leprosy but after several meta-analysis it is clear that it is not very useful so is rarely practiced now. It rarely provides early evidence of leprosy infection, and sometimes may lead to perforation of the septum by ungentle manipulation during procedure. However, it has value in confirming the diagnosis, in assessing the response to treatment (as nasal mucosa harbors highest MI and BI in comparison to other sites) and indicating freedom from infectivity (as nasal mucosa is the site for entry and exit).

ACAD EXPERT SESSION

"Photography and Archiving Of Clinical Images in Dermatology"

Dr. Shyam B. Verma

Dr. Sanjeev Goyal

Dr. Sanjeev Aurangabadkar

Dr. Rashmi Mittal

(11/06/2017 - 23/06/2017)

"Photography and Archiving Of Clinical Images in Dermatology"

1. Which is the best Modality to shoot a clinical Image: SLR, DSLR, Mobile Camera, small and Handy cameras?

   As far as clinical images are concerned,smartphone cameras are no less than other costly cameras.

2. What is the role of good back ground and angle of shooting in a clinical image for publication?

   For any image to look good,subject must be in focus and highlighted and the background must be blurred.This is known as BOKEH in photography language.This effect is seen in DSLR pics at low aperture (f number) but one can make the surrounding blurred with help of photo editing software. If that is not possible,pictures must be taken with plain,contrast colored cloth in background that does not distract.

3. Optimal colour reproduction of skin tone in 'before and after' images. What needs to be done to ensure that these are most accurately repeatable?

   This can be done by clicking the images in RAW mode (facility available in DSLRs and now even a few point and shoot cams also).RAW image is like the negative of old world pics.RAW file is to be processed and you can add color, contrast, brightness, sharpness etc. as per your choice to look it natural and it gets converted in JPEG format. If you click in JPEG format, that can also be edited but not to that extent. Lightroom CC, Picasa, fastone are little software to edit pictures.

4. Should it be a routine to use an artificial light source for clinical images of excellent quality?

   If one is using artificial light,it must not directly fall on the subject.If you are using DSLR or point and shoot, it is advisable to use a light diffuser(very cheap are available online).Diffusers are the invertedumbrellas covering the light source,which we usually see in weddings.Similar covers are available that can be fixed on cam flashes.

5. What are pixels?

   The word "pixel" means a picture element. Every photograph, in digital form, is made up of pixels. They are the smallest unit of information that makes up a picture. The number of pixels in an image is sometimes called the resolution. Camera manufacturers are always trying to sell you on the number of megapixels. The fact is, from a strictly megapixel point of view, most camera phones have "enough" for the average home user.The answer to how many pixels are "enough" depends on what you want to do with the image, and how big you want to enlarge it.For excellent quality prints, you'd ideally like a minimum of 240 pixels per inch in each dimension. This means for a 4"x6" print, you need 240x4 pixels in the width, and 240 x 6 pixels in the height. That's 960px wide x 1440px high. Multiplied together, that's 1,382,400 pixels, or approximately 1.4 megapixels. By the same token, to make decent 8"x10" print, you'd need a 4.6 megapixel camera.

   Lower resolution comes with a few advantages, as well as disadvantages.

Advantages are:

1. It results in smaller files, which are easier and faster to post-process.
2. Smaller files also reduce the overall need for storage - you can use smaller capacity memory cards and smaller backup storage (whether local or online).
3. Lower resolution images typically do not need to be resized / down-sampled - they look pretty clean "as is", so if you need to quickly process and provide them to someone, you can do it with ease.
4. Smaller files also make it possible for cameras to push higher frame rates, allowing them to be used for such needs as sports and wildlife photography, where fast frame needs are often needed to capture the right moment
5. Lower resolution cameras are more forgiving on focusing errors and lens resolution - slight focus errors will rarely be noticed in images and if a lens is not capable of resolving a lot of detail, you will not see it in images anyway.

Disadvantages are: Lower resolution cameras translate to lower image dimensions in pixels, which means that you do not have a lot of pixels to start with. So if you need to crop an image heavily, you will lose a lot of resolution and the images will become much smaller while doing so.

High resolution photography

Advantages:

The biggest advantage of a high resolution camera and why people choose them is larger output size - when you want to make a huge print, or display all the intricate details of an image on a high resolution. Another advantage of high resolution cameras is cropping options. Since you start off with a lot of pixels, you could crop images (sometimes aggressively) and still end up with plenty of resolution for high-quality print / output.

Disadvantages:

1. The higher the camera resolution, the bigger the image dimensions in pixels and hence overall file size. This not only puts a strain on storage, requiring larger memory cards, hard drives and backup storage, but also on processing power - you will need a computer that can actually handle such large files at acceptable speeds.
2. High resolution cameras require high-quality lenses capable of resolving a lot of detail. If you want to be able to get superb pixel-level details when looking at images at 100% zoom, you will need to pay much more attention to your focusing techniques - even a slight focus error will show.
3. You will need to pay more attention to camera shake As a result, you might find yourself using a tripod more often than you would like.

6. Can you elaborate on the pros and cons of using a flash for clinical photography?

Fortunately, clinical photography involves a subject that is generally within the photographer's field of control; unlike wildlife photography where the subject may appear and disappear in a fraction of a second. Lighting is often a tricky issue in dermatological photography.

The following aspects should be carefully considered to capture a clear image: adequate lighting in the room and position of light, patient's position, background and distance of camera from the patient.

Ideally broad daylight or a naturally lit room, if available would be the best; however, many times we have to take our photographs indoors with the use of flashes or other accessory light sources. The patient must face the slanting sun rays, so as to get a shadow less picture.

All compact digital cameras have inbuilt flash units. Unfortunately most of the compact units do not have options for controlling the intensity of the flash. The most important thing while using an inbuilt flash is to avoid getting too close as the distinctive features of the lesion may get washed off. One should keep in mind that distance of the flash from the subject can also alter the saturation of the colors of your subject. Even high intensity of flash tends to reduce the saturation of the subject colors. With point and shoot cameras, especially when shooting indoors without a tripod, it is better to keep the flash on. This prevents image blurring that happens due to minor camera shakes. Also, it would be advisable to vary the 'white balance' on the camera depending on the primary lighting (e.g. adjust the white balance to fluorescent if shooting under predominantly fluorescent light).

Ring flash surrounds the lens and provides light from all around the lens. The subject tends to get lit from all directions. This eliminates shadows. However, this too may reflect from the surface of the skin lesion being photographed, particularly if the distance between the camera and the lesion is small, and wash away all the surface details.

7. What is the best method of taking close-ups of mucosal (oral/genital) lesions with both a DSLR and a smartphone camera?

As there is mucus covering on oral mucosa,flash light is going to be reflected leading to highlight issue. Therefore, use of diffuser is advised even with ring flash for clinical pics in general and oral mucosa in particular.

8. Which is the best photo editing software for beginners?

Microsoft office picture manager is the basic and easy to use photo editingsoftware, where you can compress the pics to thedesired size.Picasa is also easy to use with some advance options.Lightroom CC by Adobe is best for pics shot in RAW mode.

ACAD EXPERT SESSION

"Hemangioma, Keratoderma and Alopecia Aerata in Children"

Dr. S. Criton

Dr. Jayakar Thomas

Dr. Rajeev Shrama

Dr. Dinesh Kumar

(26/06/2017-10/07/2017)

Pediatric Dermatology: - Hemangioma, Keratoderma and Alopecia Areata in Children

1. Is minoxidil safe to use in children?

   It can be used inchildren above 5 years with localized resistant patches with a strict instruction to the parents not to let children play in heat as the drug may trickle down the face due to sweating and result in hypertrichosis. No side effects if used for localized patches, not absorbed much.

2. Which immunosuppressive agent is preferred in pediatric alopecia areata while tapering OMP?

   None of the immunosuppressant is FDA approved in pediatric AA. For localized disease, while tapering off OMP, topical steroids, topical immunomodulators, local irritants or I/L steroids or topical minoxidil alone or in combination depending upon the response, if multiple AA patches but less than 40% affection, anthralin or minoxidil or earlier mentioned alone or in combination depending upon the response. For AA > 40%, alopecia totalis or universalis, phototherapy or DPCP or tofacitinib can be used. If last three options not available then cyclosporine or MTX. But please keep informed that for DPCP, phototherapy, JAK inhibitors etc. when decided to use, use of OMP is not a must. They alone are sufficient for extensive disease. OMP steroids should ideally be reserved for rapidly progressive disease or resistant to above therapies alone or if above mentioned treatment modalities are not readily available or cost is an issue. Despite all precautions, rebound is most common after steroids therapy, although can happen after all therapies.

3. What would be the standard set of investigations for every pediatric patient who comes with a significantly large hemangioma over face?

   Hemangiomas can be diagnosed clinically, however when a large significant hemangiomas over the face needs further evaluation by imaging studies(MRI, Doppler). MRI with angiography is usually indicated. Large segmental hemangiomas on the face can be indicative for PHACES syndrome (Posterior fossa anomalies, Haemangiomas, Arterial anomalies, Coarctation of aorta and Cardiac defects, Eye abnormalities, Sternal clefting and Supra-umbilical raphe). In such a situation a thorough ophthalmological, cardiac and neurological evaluation is needed.

4. What are the investigations required before starting oral beta blockers in a child?

   Routine investigations like CBC, RFT, LFT, blood sugar, Chest X-ray, routine urine and a cardiopulmonary assessment including pulse rate, blood pressure, baseline ECG needs to be done. Echo is indicated if suggestive of any structural or functional heart problems. Height, weight of the child, along with measurement of dimensions of the lesion and photographs need to be done.

   The following contraindications should be ruled out, bronchial asthma, heart failure, sinus bradycardia, hypoglycemia, hypotension, heart block & known allergy to propranolol.

5. There are a variety lesions mimicking various keratodermas in pediatric age group. How do you differentiate between various types of seasonal keratodermas in the pediatric age group? Is biopsy and patch test a must in every such case?

Biospy & HPE in keratodermas show few unique HP changes such as perinuclear vacuolization and keratohyaline granules (of varied in size and shape, located in the cell periphery). EM and molecular level study may throw more light in such cases. HPE would be useful in Epidermolytic PPK, Palmoplantar psoriasis, Norwegian scabies, Tinea mannum.

6. What are the bad prognosis indicators in alopecia areata?

The bad prognostic indicators are: early age of onset, past history of AA, Down syndrome, extensive involvement, ophiasis and/or onychodystrophy, atopy (or first-degree family history), other auto immune disorder associations.

7. Why do few patients do not respond to intralesional steroid? Is there any specific reason?

The possible reasons could be:

- ❖ Decreased expression of thioredoxinreductase 1 in the outer root sheath resulting in glucocorticoid resistance in some AA cases.
- ❖ ILS is more effective in limited patch disease and in those with a shorter duration of presentation. It is less effective in those with rapidly progressing disease, extensive disease and in those with an onset of more than 8-12 months.
- ❖ AA is an inflammatory condition with dermal infiltrate, so if the injection happens to be placed a few millimeters deep than the dermis, I guess there would a less than adequate response. Also a deeper injection will result in atrophy.
- ❖ ILS will be more effective in patients who will show active inflammatory infiltrate on HPE. ILS response is not great in an inactive patch.

8. How is tofacitinib used in alopecia areata? Is it available in India?

Tofacitinib is a JAK 1 inhibitor, only oral biologic available. So far it is used starting from age 12 yrs. It is indicated for extensive AA (>40%), alopecia totalis or universalis. So far we use it when other lines of therapies including DPCP or PUVA have failed. As cost is a real issue. Like any other biologic, initial screening for hepatitis, tuberculosis, or other significant underlying focus of infection, LFTs, etc. is a must. Recommended dose is 10mg/ day. Minimum trial period should be 6 months before we call it a failure.

Yes, tofacitinib is available in India.Pfizer markets it. Dose is 5 mg twice daily. The monthly cost is approximately 30k.

9. How long can we continue acitretin in palmoplantarkeratoderma?

Acitretin works well in some cases of keratodermas and not so well on others. In kids, can be given in dose of 0.5-1mg/kg/day with a maximum of 25mg/day.Generally response is seen by 3-6months, thereafter dose can be titrated as per response & reduced.

ACAD EXPERT SESSION

"Leg Ulcers and Role of a Dermatologist In Management"

Dr. Rajesh Verma

Dr. Reena Rai

Dr. Manjunath Shenoy

Dr Brijesh Nair

(27/07/2017-14/08/2017)

Leg Ulcers and Role of a Dermatologist in Management

1. Many times the venous ulcer heals but the skin changes in the form of pigmentation or thickening persists for a long time. What are the best treatment modalities for this?

   Stanazolol gets rid of the thickening (lipodermatosclerosis) and in the process might reduce pigmentation. But in the healing stage of an ulcer, using a specific depigmenting cream carries risk of sensitization.

2. When do we decide to do PRP for an ulcer?

   Leg Ulcers persisting for more than six weeks and showing no tendency to heal after three or more months.

3. Topical antibiotics- which one to be preferred?

   None. Only saline cleaning/ tap water cleaning. Antibiotics will sensitize and delay healing in many.

4. How do you determine the end points of the treatment of leg ulcers?

   Difficult to determine endpoints but periodic assessmentof size is important. Data suggests that a venous leg ulcer that fails to decrease in size by 30% (percentage area reduction) of its initial size over the first 4 weeks of treatment has a 68% probability of failing to heal within 24 weeks. Treatment of underlying cause, good wound care, adequate nutrition, and long standing ulcers should heal within 4 weeks. PRF can be used as an adjunct treatment with excellent results.

5. How exactly do you define a Leg Ulcer?

   It is a full thickness skin loss on the legs. Chronic leg ulcer is defined as a defect in the skin below the level of knee persistingfor more than six weeks and shows no tendency to heal after three or more months. Chronic lower limb ulcer is a chronic wound of the leg that shows no tendency to heal after 3 months of appropriate treatment or is still not fully healed at 12 month

4. Is it a dermatologist alone domain or we should incorporate a plastic/vascular or general surgeon along with since beginning??

   A dermatologist cannot go solo in many cases. So different leg ulcer scenarios demand liaison with various other specialties based on the underlying etiology. Leg ulcers demand collaborative care among dermatologist, vascular, plastic, general surgeon, diabetologist, podiatrist, physician,rheumatologist, physiotherapist, counsellor/psychologist and nutritionist.

4. How do you determine the end points of the treatment of leg ulcers?

   Complete re-epithelialisation of the wounds with adequate control of the underlying co-morbidities which predispose to the ulcer would be a reasonable end point.

5. What is the utility of ABPI in venous ulcer?

Ankle Brachial pressure Index is one 'not to be neglected' index for differentiating Venous Vs Mixed Venous arterial. Compression if delivered to a mixed AV Leg ulcer will aggravate arterial compromise and will lead to disastrous consequences. The majority of patients diagnosed with so-called 'mixed ulcers' in fact have ulcers of venous etiology and develop arterial insufficiency over time. All patients with an ABPI of less than 0.8 should be referred to vascular surgeon/ surgical specialist for further evaluation. If ABPI is between 0.5-0.8, then compression stockings/ bandages can be administered after specialist approval and careful monitoring. ABPI < 0.5 is an impending vascular emergency and is outside the purview of our specialty. The best method to assess ABPI is by handheld Doppler.

6. What are the drug options for CVI and how you chose a drug?

Pentoxyfylline (400mg twice to thrice a day) and Cilostazole are the drugs of choice.

Micronized Purified Flavonoid Fraction (DAFLON, 500mg twice a day) has a body of evidence going for it and is the vascular surgeon's favorite. It can be combined it with Trental.

Lipodermatosclerosis responds well to Stanozolol, keeping in the mind the anabolic steroid related adverse effects and potential hepatotoxicity.

Zinc has very low levels of evidence but if tolerated well, it can be administered in high doses. (Zinc - 1 tab ASCAZIN BD)

Calcium Dobesilate (500mg twice a day) & Rutoside-Bromelain are anecdotally helpful.

7. What is the role of lymphatic insufficiency in chronic venous incompetence and venous ulcer?

Lymphatic insufficiency is almost always accompanied by venous insufficiency. Compression is the key to management. There is microangiopathy of the lymphatic network in CVI.The resulting lymphatic dysfunction leads to chronic edema which complicates the clinical picture. Optimum management requires measures to improve lymphatic drainage as well as attending to the venous disease. Exercise, elevation, and compression are principles of treatment effective in both pathologies.

The inability to pinch a fold of skin at the base of the second toe (Kaposi-Stemmer's sign), hyperkeratosis, and increased skin creases are the signs of lymphatic insufficiency. Lymphedema equates with delayed wound healing. It increases exudation & maceration.

8. Kindly elaborate detailed method for compression therapy, that is, what exactly we need to do and for how long? How to determine n apply suitable pressure bandage?

Any form of compression, even crepe bandage is better than no compression at all. Readymade hosiery stockings are graded into 4 classes. The highest pressures are delivered by class 4 stockings. But as pressure increases, discomfort increases. In venous leg ulcers, class 3 stockings are recommended. But practically a few patients drop out due to discomfort. Hence even class 2 stocking may be practical. 4 layer bandaging is a more complicated affair. We have absorbent wool initially, then comes second layer of cotton crepe bandage. The cotton is to level the inherent anatomical unevenness. Then an elastic compression bandage layer and finally covered by a cohesive compression bandage is the 4 layer bandage. Highly technique intensive.

The method of application of compression is to hold the ankle at 90 degrees. Start from base of toes. Start in figure of 8 pattern of wrapping around ankle and start going upwards up to knee. Both spiral and figure of 8 pattern can be followed on leg but figure of 8 delivers better SBP. Overlap of one turn over next of the bandage should be 50 %. These are the essentials of any compression bandaging. The number of components only change with each bandage type. Bandaging Vs hosiery is a controversy. Less resting pressures in bandage makes it more comfortable at rest. But changing it needs nursing care. So hosiery scores in convenience over bandages

9. Leg Ulcers in 2040

    1. Integration of all concerned subspecialties into a dedicated 'LEG ULCEROLOGY' Specialty Hospital.

    2. Robotic 3 D printing after automatic assessment of ulcer depth. The 3 D printer will have all skin components loaded into it and the printing occurs directly on the breach.

    3. Safer biocompatible bioengineered dressings

    4. Intense research on cytokine profile in Venous Leg Ulcers makes us home on to the master cytokine controlling wound healing and appropriate adjuvant biological therapy against the said cytokine

    5. Robotic venous Surgery to tackle incompetent perforators which is the often neglected part of phlebosurgery.

    6. AI takes over High quality real time MRI based Duplex Ultrasound substitute. Nanobots released into each nooks and crannies of the vascular system. And they keep giving real time inputs to a master bot overseeing the imaging. So real time imaging from both within and without and collation of the data by AI. Then the surgeons/ robotic perforator collaborate to set things right.

10. Prevention of trophic leg ulcer

    Prophylaxis is of utmost importance. Trophic ulcers are seen mostly secondary to diabetes, hansens disease and rheumatoid arthritis. Gouty tophi might also ulcerate.

    - ❖ Daily night the diabetic must inspect the foot in detail
    - ❖ Any calluses must be tackled with appropriate keratolytics under occlusion and paring.
    - ❖ Look for rise in local skin temperature in the affected area indicating impending ulceration
    - ❖ Lifestyle changes for weight control
    - ❖ Regular moisturization.
    - ❖ Disseminating information about the concept of podiatry which many are unaware of.
    - ❖ Adequate nutrition and correction of hypoalbuminemia, good glycemic control

- ❖ Albuminuria must be checked for as it is the albuminuric diabetic who is more prone to trophic ulcers

11. What are the indications of debridement?

Presence of non-viable necrotic non bleeding slough which delays healing of wound needs to be debrided.The aim is to convert chronic wound into an acute wound so as to promote healing.

12. Management pearls in an established case of classical lipodermatosclerosis.

- ❖ General measures - Weight reduction (It makes huge difference), Pain reduction - antibiotics/ pain killers if frank infection is seen
- ❖ Rheologics of your choice
- ❖ Fibrinolysis - Stanozolol
- ❖ Inflammation - Topical steroids, IL steroids in worst cases
- ❖ 4- Regular management of venous incompetence - Elevation, compression, exercise

13. What are the common causes of leg ulcers in Systemic Sclerosis?

The etiology is multifactorial, including microangiopathy, macrovasculopathy, bacterial infection, fibrosis, and calcinosis

- ❖ Calcinosis - Treat systemic sclerosis, surgery
- ❖ Vasculitis - steroids, immune suppressants
- ❖ Vasculopathy - pentoxyphyllin, Ca-channel blockers etc
- ❖ For digital ulcers tadalafil, topical nitrates help in healing and relieving pain
- ❖ Apart from treatment of infection, local wound care low molecular weight heparin followed by warfarin is effective.
- ❖ Investigate for procoagulant state especially Antiphospholipid antibody.

ACAD EXPERT SESSION

"Topical Steroid Dependent Face - Facts and Management"

Dr Arijit Coondoo

Dr Kaushik Lahiri

Dr Abir Saraswat

Dr Rajetha Damisetty

Dr Deepika Pandhi

(17/08/2017-03/09/2017)

Topical Steroid Dependent Face - Facts and Management

1. Topical steroid dependent or damaged face? How safe we ourselves are, as dermatologists when we ourselves use this term and prescribe steroids on the face??

   As far as the question regarding Damaged or Dependent is concerned my take is that both are relevant. It is the dependence which brings on the damage. Sometimes of course damage may occur after short term use without any dependence. On the other hand a person may be dependent on the TS without much damage to the so n. In this case the amount of TS absorbed with resultant HPA axis suppression which causes concern.

   Both terms describe the entity-there is dependence as well as damaged skin. Topical steroids are well known (with adequate experimental data) to have a compromised barrier function

   If we want to discuss all types of TS abuse-related problems on the face, 'damaged' is good enough. It can refer to atrophy, redness, telangiectasia, acne, hirsutism, acne, hypo pigmentation etc.

   "Topical steroid 'dependent' face" is certainly not a misnomer. But it does not apply to all patients who misuse topical steroids (TS). There are some who develop severe withdrawal symptoms even after using mid or low potency TS while some get away with the usage of super-potent ones without too much of obvious damage or dependence.'Dependence' however is very specific and should only be used in patients who get redness and burning/itching on stopping the steroids.

2. As an association of learned members and for a social cause, if we want to eradicate TSDF, what would be the best target to do so?

   Given the sheer volume of the number of people that are currently diagnosed with TSDF-the target for education about this entity and prevention should be multipronged- Public media awareness, general practitioners,nurses as a focus topic for undergraduates would be a good initial area to focus on. For beauticians commonest cause is their recommendation of betamethasone as a moisturizer and triple combination for melasma/ pigmentation. However, targeting RMPs, pharmacists and beauty parlours would require more logistic inputs and planning.

3. Is there a role for ivermectin cream in steroid induced rosacea?

   Theoretically, yes as Demodex is increased in Steroid rosacea too. Ivermectin 1% (Soolantra) is well tolerated, fast in onset of action, anti-inflammatory and decreases relapse.

4. What is the role of tacrolimus and metronidiazole in TSDF?

   Tacrolimus 0.03% over face reduces the erythema as it is anti-inflammatory but has minimal effect on papules and pustules.Interestingly that has been a criticism for use of tacrolimus for TSDF as it may increase signs and symptoms due to flare of Demodex.

   Ancillary treatment is important- sunscreen, moisturizer and mild cleanser to ensure tolerance

Metronidazole cream/ gel we were using when other options were not available as it is much cheaper. At least the generic and some brands available in India are not well tolerated and no - minimal response is there. It is important to stress on photo protection to ensure tolerance when metrogyl is used.

5. How do you suggest management for vellus hair induced by topical steroids?

   Vellus hair is almost always temporary and you don't really need any intervention.

6. What is the safest maximum duration of steroid application on the face according to the potency category?

   Any steroid stronger than class 5 (National psoriasis foundation classification) for more than two - three weeks is known to cause side effects. Unless the indication is DLE or severe actinic dermatitis or airborne CD in which case you'll have to use more potent TS and can use them without adverse events. Vitiligo patients seem to do well with class 5-6 steroids for many months, especially if drug holidays are given.

7. Is there any role of oral steroids in the TSDF management?

   None

8. What can be the ideal management of the patients who has steroid dependent face??

   That can be summed up in the following steps.

   a. Diagnosis of underlying condition and initiation of appropriate systemic therapy for the same.

      Eg. Oral tranexamic acid for melasma, oral isotretinoin for recalcitrant acne, combined oral contraceptive and spironolactone and isotretinoin for hormonal acne after assessing risk-benefit ratio, oral antifungals to reduce Malassezia load in seborrheicdermatitis (itraconazole), tetracyclines, permethrin, metronidazole for rosacea.

   b. Gentle cleanser (cetyl-steryl alcohol based)

   c. Emollients (filaggrin and ceramide containing ones may have a role). Ichyol pale's anti-inflammatory effects may also come in handy.

   d. A pure physical sunscreen

   e. Pimecrolimus for the symptomatic ones. Those having severe stinging and burning may need a mild steroid like hydrocortisone.

   f. Tricyclic antidepressants with anti-histamine like effects like doxepin help in relieving symptoms.

9. Does the treatment vary with the duration of the steroid application?

   Not really. Symptoms of withdrawal are not directly proportional to the duration of misuse or the potency of TS used. There are patients who get away with going cold turkey after years of

clobetasol application and there are others who are miserable after stopping mometasone after a few weeks of using mometasone.

It is important to treat only symptoms of burning or itching with a milder steroid. For all other concerns like flare of acne and melasma and diffuse darkening of skin, patients have to be repeatedly told that their skin would get worse before it gets better. Those with underlying rosacea or an already compromised barrier probably develop more symptoms than those who had normal skin prior to TS misuse. It is best to go by the symptoms that surface after withdrawal than to have a rigid approach.

10. What is the current legal position of the steroid combinations available in India??

The legal position of TS containing fixed dose combinations (FDCs), in a nut shell, is 'anything goes'. The Kokate committee which was appointed by the central govt to scrutinize irrational FDCs banned around 350 combinations including 22 TS containing ones in March 2016. The highest selling one, Panderm plus (clobetasol-ofloxacin-ornidazole-terbinafine aka COOT combination) was one of them. The pharmaceutical industry went to court promptly and managed to get the case thrown out by the Delhi high court on grounds that the way the ban was carried out was faulty. The case is right now pending in the Supreme Court and is expected to come for hearing in the next few weeks.

During this time, the ever innovative Indian Pharma companies prepared for the ban by introducing new combinations to cash in on the brand value of their best sellers. A case in point is Panderm-NM (Clobetasol-neomycin-miconazole combination) and Panderm super (Clobetasol propionate 0.05 %W/W+Clotrimazole 1 %W/W+Fusidic Acid 2 %W/W) that were launched within six months of the ban and made a neat 100 crores, even while Panderm + was re-launched and garnered 270 crore INR in the financial year.

ACAD EXPERT SESSION

"Dermatopathology as A Speciality In India"

Dr M. Ramam

Dr Sujay Khandpur

Dr Sherina Laskar

(14/09/2017-24/09/2017)

"Dermatopathology as a Specialty in India"

1. Ideally who should practice dermatopathology- a dermatologist or a pathologist?

   This specialty can be practiced by both dermatologists and pathologists with adequate training and exposure in the respective field. It is important for a dermatologist to undergo some sort of preliminary training in basic pathology and dermatopathology in his own institute and then undertake a fellowship in dermatopathology. Similarly it is important for a pathologist, not only to undertake training in dermatopathology but also undergo clinical training in dermatology. Hence both the dermatologist and pathologist must be well equipped with in clinical and pathology skills to practice dermatopathology. It is essential for dermatology trainees to learn as much dermatopathology as they possibly can during their two or three years, not to report their slides, but in order to understand the pathogenesis of skin disease better. The dermatologists should make better dermatopathologist as most of the issues in dermatology are resolved by clinic pathological correlation & good clinical skills help in that.

2. What are the various trainings available in India for an aspiring dermatopathologist?

   Currently, training is through IADVL fellowships at CMC Vellore and Mumbai, AIIMS fellowship for which one applies directly to AIIMS academic section and Department of Dermatology, IADVL International fellowships being given each year to go abroad for training, ICMR International fellowships to Biomedical Scientists in which one can go for dermatopathology training. IADVL Academy proposes to start in near future more fellowships/observer ships in this subject. KEM Hospital is an excellent setup for acquiring dermatopathology training. The only program in India providing a year-long training is Khopkar Sir's Fellowship course in Diagnostic Dermatopathology under the MUHS that takes place at the Seth G.S. Medical College and KEM Hospital.

3. What is the investment required to run an Individual dermatopathology clinic in private setup?

   The investment would be similar to any other histopathology service. The investment depends on how ambitious one is. The minimum requirement for a laboratory is a microtome and a fairly good one should cost approximately 5 lakhs. For a starter, tissue processing and staining could be done manually, instead of using expensive auto processors and auto stainers. However, there are other expenses involving an embedding station, water bath, moulds, reagents etc. and this involves a total expense of 15-20 lakhs to project a very modest estimate. The most important investment is a technically sound lab technician!

4. What's the optimum training period required for a dermatologist to be trained as a dermatopathologist?

   A dermatologist before undertaking a fellowship in this subject, must gain some sort of personal experience/training in his own center in collaboration with his pathology department and senior dermatology colleagues who are trained in dermatopathology, by looking at slides regularly, attending classes, doing some research work etc. This equips him to undergo more advanced

training at higher centers within the country or abroad. A minimum required training period is of a year, and ideally two. That should include for a dermatologist, a few months learning basic lab techniques in histopathology beginning from handling specimen accessions to grossing and processing of tissues, in order to not feel handicapped while having a lab of their own. This is the reason why most dermpath programs in the US and elsewhere require dermatologists to have 6 months experience in Pathology. The best training programs also involve dermatologists spending a few months of their training period in General Pathology. If possible it should be further garnished with short courses in immuno-dermatology to have familiarity with DIF, immunohistochemical staining relevant to dermatology & immunofluorescent antigen mapping for EB.

5. What is the global scenario and future perspectives for a budding dermatopathologist in India??

   Internationally, dermatopathology is a full time specialty and function as independent departments, consisting of dermatopathologists with basic training in dermatology or pathology. They are the leaders in medicine in their country. In India, prospects of this sub-specialty in both government and private sector is immense.

6. Can a general dermatologist sign out reports as dermatopathology?

   Dr. Ackerman's article says: "Dermatologist (not equal to) dermatopathogist: No place in a profession for pretenders."

7. Immunohistochemical markers are very costly with SRL or Metropolis, are there any centers where we can get it done at a cheaper cost especially for the needy in private practice?

   We have a similar problem here at our medical college, since the majority of our parents ill-afford these investigations. I've struck a deal with a lesser known lab called Onquest (headquartered in Delhi); they do provide discounts for poor patients. I recently got CD1a, CD 68 and S100 at a total of Rs. 2400 for an adult (suspected) LCH patient (they provided brilliant images too), when SRL asked for 5K for the panel or 2500/marker. You could look for something similar in Mumbai too. They do IHC at CMC, Vellore, St. John's, Bangalore and a number of other institutions too; we could collate a list of these centers for the benefit of our patients and work out a system of referral.

8. Can confocal microscopy replace classical histopathology?

   Given that confocal microscopy requires special training, is expensive and most importantly, provides optical imaging up to a depth of 200microm (upper dermis), it is unlikely to replace routine histopathology as the gold standard in the near future.

9. What is the role of telepathology?

   Telepathology would be an important aspect of dermatopathology reporting. Currently due to inconsistency in quality of photomicrographs from different centers it is not successful. Now scanners are available that can automatically scan many slides together and the virtual slide image obtained on screen just looks like one under the microscope. One can move this image and see every detail from any portion of the specimen easily. But these scanners are expensive

costing between 50 lakh to a crore and can be only procured by bigger set ups. But they make telepathology a wonderful experience.

10. What are the common errors you see in the technique of taking biopsy (size/site/etc) which makes the job of a dermatopathologist harder?

A standard 4 or 5mm punch suffices in most inflammatory dermatoses and neoplastic conditions, however, in case of suspected panniculitis: a deep incisional biopsy is preferable for adequate depth. Vesiculobullous: a punch or incisional from a fresh blister

Suspected melanoma/pigmented neoplasm: an excision biopsy with a 2mm margin as far as possible, to assess the depth and lateral margins.

Inflammatory/infective conditions with associated ulceration: avoid the ulcerated area, if possible, as the intense inflammation and vasculitic changes (non-specific change) beneath the ulcer distracts from the actual pathology of the condition.

Erythroderma: repeated biopsies may be required, but to get lucky, do look for a papule or plaque that may have constituted an early, but well-formed lesion in the disease process.

Alopecias: two 4 or 5mm punches are desirable. Non-scarring: one from centre of area of hair loss, and the other from the occiput (non-androgen-dependent area) that serves as a control/ comparison for evaluation of AGA (we must be aware that the alopecia may be multi-factorial). Scarring: both from the active edge of the patch, especially if erythema is discernible. Type of sectioning: there are several different protocols (please see attachment), but generally for non-scarring, both specimens are bisected and embedded face down and sectioned horizontally. In scarring, one specimen is bisected and sectioned horizontally; the other is bisected vertically, one half is reserved for DIF if needed, and the other serially sectioned vertically.

11. What is the standard way of taking biopsy for patients of epidermolysis bullosa to prevent epidermis sloughing off?

Adequate biopsy size, depth, sometimes need of multiple biopsies, time in evolution of disease at which biopsy taken, site of biopsy are important considerations.

Not just EB desloughing of epidermis is a problem in all vesicobullous biopsies. Taking a new small and intact vesicle. If there is epidermal removal making sure that the roof and rest of specimen are put in the same bottle before sending it to pathology may circumvent this problem.

The sample should be taken from the normal skin from anywhere in the skin (in minor variants may be from vicinity of the lesions). After gentle rubbing of the skin (to induce a split), a sterile needle can be placed in the skin & a shave biopsy can be performed on top of that. The aim is to include epidermis with dermo-epidermal junction with little of dermis, deeper biopsy is not indicated. Light microscopy has no role except for acral peeling skin syndrome or may be in few acantholytic variants with desmosomal protein mutations. Ideal to look for immunofluorescent antigen mapping, the gold standard & Scanning EM if available as it is still of help & directs in some cases.

Problems can be caused by small biopsies, shallow biopsies, biopsies not taken from well-developed lesions, biopsies injured during the process (forceps/needle artefacts), biopsies not put in formalin right away, or put in formalin that is not properly buffered and of the right concentration or is old and exposed to the atmosphere, biopsies put in formalin and then put in the freezer leading to freeze artefacts, biopsies taken with electrocautery/RFA, and biopsies cut into 2-3 pieces to send to different labs (it is better to take multiple biopsies and send complete biopsies to each place).

Problems can occur in the lab if the section is not properly processed, if thick sections are cut, if staining is not balanced, if sections tear or are folded, if the DPX develops bubbles or if stains fade quickly.

Reporting is facilitated if the clinical history is provided adequate detail, differential diagnoses have been considered and listed, and clinical images are available. Though sometimes difficult to do, taking 2-3 biopsies when the diagnosis is in doubt increases the chances of reaching a conclusion. Both the pathologist and clinician should recognise that discussing the case with an open mind after viewing the slides can lead to a better evaluation; this should be a collaborative relationship not an adversarial one.

12. How to choose horizontal v/s transverse sectioning in alopecia?

Horizontal sections are required in non cicatricial while for cicatricial both are needed.

13. Are IIF and serological tests really going to challenge DIF as thegold standard for immunobullous disorders?

IIF is not done routinely as it has low sensitivity and animal substrates are not available in most labs due to government policy of animal protection. Few labs may use healthy human skin biopsy as substrate if client insists for the test. Elisa test is very sensitive and can differentiate pemphigus from other vesicobullous diseases if confusion occurs especially between different causes of oral ulcers as diagnostic yield of mucosa biopsy and DIF is low or in a situation where we are not getting anything specific on these tests.

Ideally, all three, DIF, IIF (with salt splitting if preliminary observations of DIF warrant it) and an ELISA for a quantitative estimation of the antibodies should be done. It is hard to imagine that IIF and ELISA or other serological tests will replace DIF as the gold standard.

In immunobullous conditions, it's best to send a lesional biopsy in formalin for routine HPE and a perilesional in Michel's medium for DIF at one go, because both would be required to arrive at a diagnosis and it is easier for the patient and the doctor taking the samples too.

IHC should never be interpreted in the absence of H&E findings. Since IHC can be carried out on the sections embedded in the paraffin block made from the original specimen, one could wait for the H&E findings in order to decide what immunomarkers to ask for. You have the option of asking for a panel of immunomarkers (it's cheaper when several are required) or select a few based on the H&E findings.

14. A lot of conferences these days prefer workshops on dermatosurgery, cosmetology etc, but hardly dermatopathology- your views on how can we change this scenario?

Ans- Through SIG Dermpath we held 7 workshops in 2016 and 4 so far in 2017. Dermatopathology Society of India,has been doing a lot. and holds high quality conferences and CMEs in this field, the next one being at JIPMER Puducherry from Dec 1-3. There is the Indian Journal of Dermatopathology and Diagnostic Dermatology that is publishing articles in this field. A lot of interest is getting generated in this subject. The Dermatopathology Society of India runs a monthly quiz with digital whole slide images that can also be helpful for someone wanting to test themselves. It is open to both members and non-members and can be accessed most easily thorough the Society's Facebook page at https://www.facebook.com/Dermatopathology-Society-of-India-143668552438932/

15. Can you suggest good resource material- websites, books to increase the knowledge in this subject?

www.pathpresenter.com provides free access to whole slide images with teaching information. www.dermpathpro.com is similarly helpful.

Dr Jag Bhawan's online atlas can also be accessed for free at http://www.dermpathatlas.com/

The page is also a good social media destination for people interested in dermatopathology. Other societies and groups are also on Facebook and there are some useful YouTube videos, too.

16. Dermatopathology in 2040

Molecular diagnostics aiding histopathology

High quality teledermpath teaching

All institutes of country having highly trained dermpath faculty

Indian dermpath journals competing with world's best

Significant improvement in softwares for quantitative assessment of various histological parameters

Noninvasive diagnostic methods comparable to histopathology.

17. When not to believe dermatopathologists report?

Since biopsies are often performed to resolve diagnostic uncertainty, it may be difficult to decide that the biopsy report is incorrect when we are ourselves unsure of the clinical diagnosis. However, uncertainty is not limitless. When faced with a perplexing clinical problem, our differential diagnoses include a set of conditions that look somewhat like each other and does not include all the diseases described in the book. If the biopsy report offers a diagnosis that is clearly inappropriate, e.g., mycosis fungoides for a single, hypopigmented macule on the arm of a young man, it is better to ignore the report than to search for reports of single patch mycosis

fungoides and see if it fits the patient's clinical presentation. (Or even worse, refer the patient to a cancer hospital).

In fact, it may be a good idea to speak to the dermatopathologist and work out a way for him to communicate such thoughts without putting them down as a histopathological diagnosis that many people consider the final truth. Some strategies include speaking to the clinician on the phone and discussing the situation before signing out the report, or wording the diagnosis in a way that indicates uncertainty, e.g., "some histopathological findings in this biopsy suggest the possibility of mycosis fungoides. Is this clinically relevant?"

Paradoxically, biopsy reports that agree with the clinical diagnosis should also be viewed with suspicion if the diagnosed condition does not have a distinctive histopathological appearance e.g., biopsy reports of drug eruptions, pityriasisrosea, atopic dermatitis, pigmented cosmetic dermatitis, ichthyosiformerythroderma, early vitiligo, among others.

"Consistent with clinical diagnosis" is the path of least resistance and one that is often taken in situations where the biopsy findings do not add up to a particular diagnosis. An extreme example was a report of "consistent with scleredema" in a biopsy that showed nothing. The pathologist defended the report stating that early scleredema may show no changes! Biopsies sent with a diagnosis of leprosy may be reported as consistent in the absence of clear cut features because the pathologist does not want to miss this important diagnosis and knows that some cases may show a non-specific picture.

A more subtle error occurs when the diagnosis is not clear and the clinical differential diagnoses include histopathologically disparate conditions. For example, a biopsy from an indurated papulo-nodule on the face may be sent with differential diagnoses of appendageal tumour, nodular amyloidosis, sarcoidosis, nodular acne, granuloma faciale. If the pathologist sees granulomas on biopsy, she may be happy to sign it out as sarcoidosis, the only granulomatous disease on the list. However, the true diagnosis may be cutaneous leishmaniasis which the clinician had not thought of. In these challenging situations, the pathologist would do better to indicate that she has seen a granulomatous tissue reaction and ask the clinician to reassess the patient in the light of this.

Honest errors are made by dermatopathologists, as frequently or infrequently as anyone else, because they are as human as anyone else. Problems arise because it is generally believed that a lab test is superior to clinical evaluation but this is not the case. When there is discordance between the diagnoses and how the patient behaves with time or treatment, both clinician and dermatopathologist should be willing to revisit the case, think again and come up with a fresh assessment.

ACAD EXPERT SESSION

"Chemical Peeling: The Art and Science of It"

Dr Niti Khunger

Dr Maya Vedamurthy

Dr Malvika Kohli

(02/10/2017 - 20/10/2017)

"Chemical Peeling: The Art and Science of It"

1. What is punctuated phenol peel?

   Phenol is an important peeling agent in the treatment of facial rejuvenation, but there are limitations to its use because of the potential for side effects. Therefore a new protocol was developed known as punctuated phenol peel which is a modification of conventional phenol peel to reduce the adverse effects and down time. In this protocol 88% phenol peel is applied with a toothpick soaked in phenol solution with 3 mm between dots along the pathways of fine facial wrinkles. Although it is less potent and seems to require multiple applications with little or no downtime it was found to be useful in facial rhytides. Thus this new therapeutic protocol results in less downtime than traditional peels with the same agent and is a safe, low-cost procedure but has to be reserved for fairer skin types. It is the same principle as a fractional laser. But for all pure phenol peels (88%) on the face do a small test area behind the ear. It can cause depigmentation and there is a downtime of 7-10 days. The combination phenol peels like Nomelan are safer as they contain only 8%.

2. During healing phase after chemical peeling, patients often experience Pruritus. How can that be managed?

   Pruritus is more common in medium and deep chemical peels during the healing phase and lasts up to several weeks. In superficial peels it is not so common but can occur due to the following reasons

   - During the healing phase Pruritus may be accompanied by erythema
   - Peels in sensitive skin individuals
   - Dryness which follows the peel may induce Pruritus.
   - Pruritus may signal a possible contact allergy to the wound care or post care products used.

   Generally a bland emollient like soft paraffin and antihistamine is sufficient. Avoid use of drying agents like tretinoin which can worsen the itching. If it is not controlled with simple measures hydrocortisone cream can be tried. In severe cases a short course of oral steroids can be used. It is always safer to avoid fluorinated or stronger steroids for fear of atrophy and other side effects. Generally Pruritus subsides once re-epithelization is completed. Pimecrolimus may also be tried to alleviate Pruritus.

3. What is the place of chemical peels in Rosacea and which peeling agent is most appropriate to use?

   Chemical peels in rosacea can be safely used once the disease is brought under control with systemic agents. Better to avoid peels when the skin is acutely inflamed as it can worsen the erythema which can be mistaken for a disease flare up. It is generally safe to use superficial light peels like glycolic acid 20% to start with and gradually increase the concentration. Azelaic

acid peel is also beneficial in rosacea as it has anti-inflammatory action in addition to sebum control.

If there is an acute flare and severe erythema, wait for 5-7 days. You can the cautiously use a salicylic-mandelic acid gel based peel. SA takes care of the inflammation and mandelic is anti-inflammatory, reduces pigmentation, and antibacterial. It has an inhibitory action on Staph and also improves gram negative folliculitis. Since it is a larger molecule it is absorbed slowly and is less irritating.

4. Can chemical peels be used to treat pigmentation in stasis dermatitis due to hemosiderin deposition? Which agent is preferred?

Chemical peels can be done to treat pigmentation in stasis dermatitis after controlling the underlying cause. The choice of peeling agent depends on the cause of pigmentation. If pigmentation is secondary to PIH peels used to treat pigmentation can be used. If pigmentation is due to hemosiderin which is usually the case in stasis dermatitis, thioglycolic acid peels can be tried. Thioglycolic acid (TA) - or mercaptoacetic acid - belongs to the class of thioglycolates, which are substances that solubilize hemosiderotic deposits.

The daily use of a depigmentation formula (1.5% TA + 3% tranexamic acid) at night is associated with enhanced peeling treatment results.

5. Can you recommend good brands of TCA peels? Is it worthwhile to do peels in lichen planus pigmentosus?

Good brands of TCA peels are Easy TCA, mesoestetic (Glenmark/Spectra), Benev TCA (Zydus). Some TCA peels come as combinations with other peeling agents like lactic acid and is available with Sesderma.

Peels in lichen planus has to be done only when the disease is no longer active as peeling in active stage can worsen the disease due to Koebnerisation. You need to do peels which are superficial and light to avoid risk of PIH as it may darken the skin and appear as worsening of lichen planus. You can start with glycolic peels 20% and gradually increase to 50%.If you find the patient is able to tolerate glycolic peels well you may move on to TCA or phenol peels to achieve satisfactory results. However one would need many peels to see results. Nevertheless peels do help in faster resolution of pigmentation.

6. What is the utility of hand held fans during chemical peeling?

Hand held fans are quite useful during chemical peels in many ways.Some peeling agents produce stinging and burning during application which is quite normal.If it is not alleviated using a fan patient may perceive it as abnormal resulting in termination of the peel procedure. The patients hold the fan and invariably they direct it to the areas of maximum discomfort and this can act as a guide to your decision on application of further coats of peeling agent when you do procedures with multiple coats. Sometimes a burning sensation occurs during neutralization process as with glycolic acid as a result of exothermic reaction with sodium bicarbonate. This can create a lot of anxiety to nervous or anxious patients and those undergoing the treatment for the first time. Making them hold a fan not only decreases the symptoms but also serves as a distraction

technique. Remember these are desire dermatology patients and not disease dermatology patients and they would expect a lot of hand holding and careful handling.

7. Can LPP be treated with chemical peels?

LPP has dermal pigmentation, most of it in the upper dermis. A peel may be useful to hasten clearance by causing low grade inflammation and attracting macrophages. But it should be done only if the disease is not active. For LPP a medium depth peel is required like TCA 25-35%, Glycolic acid 70%. Sequential peel like 30% SA followed by TCA 25%, Jessner's peel followed by TCA 25-35%, GA 70% followed by TCA 25-35% (Cook's body peel) can be used?

The body peels are safer. Cooks peel can be applied for large body areas as it is not absorbed. Jessner's and SA should not be applied on large body surfaces.

8. What are spot peels?

Spot peels are done only an on affected area which needs to be treated with the peeling agent. They are recommended when the affected area is small and localized like freckles, age spots, FDE, Melasma. Here the surrounding normal skin is protected using Vaseline and the chemical is applied only on the lesion. The advantage is that the normal areas are spared and free from risk of any complication. Higher concentrations may be safely used in this technique and healing is also faster as the area treated is relatively smaller.

9. What are top up peels?

Top up peels is a technique of combining different classes of peeling agents to enhance the depth of the peel without using a higher concentration of the peeling agent.

i. Eg. Glycolic acid 70% combined with TCA 35% (Coleman's Peel) In darker skins, lower concentrations of TCA (10-25%) may be used .

ii. Jessner's solution with 35% TCA (Monheit's Peel)

These are also known as penetration enhancement combinations.

10. What properties do you look for when a company comes to you and shows you different peels? Specifically if you had three brands of Glycolic acid of the same percentage in front of you, what factors would help you decide the peel to buy?

If we take glycolic acid the first choice should be based on the manufacturer or brand as we can expect basic standards to be maintained with reputed companies, eg. Correct strength, stability etc. Next we must take the pH and pKa into consideration. An ideal peel solution must have pH similar to the pKa of the acid. The choice of formulation to suit your requirement.eg gel formulations are useful for under eye and small areas, lotions for larger surfaces and occlusive masks for specific areas. The last and best way to go is to ask them for samples (which they should if they are confident of their product) Do it on volunteers and use your own judgment.

11. How do you prime the patients before chemical peeling?

Skin priming is divided into 2 phases.

1) Pretreatment phase

2) Preparation phase

Both are different in timing and the agents used.

The pretreatment phase consists of topical agents applied days or weeks before the peel. The aim is multifactorial-to enhance uniform penetration of peeling agent, to accelerate wound healing, to thin down epidermal barrier and to minimize complications. Examples of priming agents in pretreatment phase are tretinoin in acne, hydroquinone in dyschromia, alpha hydroxyl acids in rejuvenation, Pimecrolimus in sensitive skin etc.

The preparation phase is one that comprises of products used just before the actual procedure.eg degreasing, cleansing agents, depth enhancing agents or even emollients applied on dry areas of skin to protect from excessive peeling.

Invariably most of the patients have undergone the pretreatment phase as a part of their regular treatment before they actually end up doing the peel. But the preparation phase is a must for safe and effective results. Priming agents need to be used at least 2-4 weeks prior to peel to be effective. Very light superficial peels like 20% GA, lactic acid and 20% mandelic peels do not need priming agents.

12. What precautions are needed for the patients on isotretinoin?

Patients on isotretinoin can undergo superficial peelings safely and studies done by IADVL members have confirmed this. We Indian dermatologists do not use as much dose of isotretinoin as in the West and this could be the cause for safety of these procedures in Indian patients. We should insist on use of sunscreens strictly in these patients as the drug itself can add to photosensitivity. In addition as a result of decreased sebum secretion there is likelihood of developing areas of dryness on the face where the peeling agent can penetrate faster leading to a deeper peel. These are also referred to as hot spots. This can be avoided by applying a moisturizer in these areas or by terminating the peel quickly in these spots. It is advisable to use lactic acid as it helps to moisturize dry skin of isotretinoin treated patients. However most of the peels can be used safely of necessary precautions is taken.

13. Are gel based peels more potent than their counterpart regular peels, eg glycolic gel 55% v/s glycolic solution based 55%.?

In general gel peels are supposed to be less potent than liquid peels and therefore considered safer in dark skin and can be used as starter peels. In addition it is less easy to spread uniformly and therefore will require a longer time to complete the peel as this is very important for peels like GA which are contact time dependent or time bound peels. I feel it is easier to observe the end point easily through liquids rather than gels especially in dark skin and that may be the reason for the apparent sudden end point. In fact gel peels can be left for a longer period of time as the free acid available is lesser and penetration rate of gel formulation is slower than an aqueous based solution.

14. What are the post peel precautions and medications you ask the patient to take?

Post peel precautions are less for superficial peels compared to medium depth peels. Avoid excessive sun exposure, scrubs, facials and sauna and advise them to avoid picking the skin during peeling phase.

Medications- Sunscreens, moisturizers or hyaluronic acid gels if skin is dry, mild steroid like hydrocortisone topically for Pruritus, Pimecrolimus if there is prolonged erythema. Antiviral if needed.

Post peel hyper pigmentation- Counseling is very important to avoid unhappy patients. They must be reassured that it is temporary. Hyper pigmentation can occur with any depth of peeling and can also occur in light skinned individuals. Sunscreens are to be used diligently. Skin lightening agents like hydroquinone to be started. Later tretinoin may be added to increase the bleaching effect. Avoid photo-sensitizing agents strictly. Tranexamic acid creams and vitamin C serum helps a great deal. Re-peeling with glycolic acid can be safely done every 2-4 weeks .If PIH persists QSW laser can be tried. Risk of hyper pigmentation can be assessed using Roberts hyper pigmentation scale which determines the propensity for pigmentation

15. Are there any new peeling agents (apart from the common ones) that are worth trying?

Ferulic peels, thioglycolic acid peels, glutathione peels

16. What is the reason that peels have not entered the mainstream guidelines in spite of being effective?

There are no worldwide multicentric placebo controlled trials published.

No large MNC sells peels in America. Hence funding for trials on chemical peels is lacking. The day Galderma or J & J start selling peels, they will fund trials, data will be published and then peels will enter into guidelines as guidelines are based on published literature. Even polidocanol for sclerotherapy got FDA approval recently in spite of long term usage because there were few trials.

In India now Cipla and Glenmark have started selling peels, but they are not initiating any trials as yet.

17. Which is the peeling agent of choice for periocular hyper melanosis?

Combination peels

Glycolic acid- will cause increased collagenisation, reduce fine wrinkling, and reduce vascular see through.

Yellow peel - increase collagenisation, reduce pigmentation

Lactic acid-moisturizing, reduce irritation due to peeling

Mandelic acid - gentle, will reduce pigmentation

TCA - potent, will reduce pigmentation

18. Which peels are safe in pregnancy?

Phenol and salicylic peel are absolutely contraindicated. Yellow peel or retinoic acid peel should also be avoided. Though even after application of yellow peel on the 30 % of the body surface area the peel has not been found to be absorbed but still better be avoided.

Another aspect of pregnancy and peel is the physiological changes that take place in the skin due to the hormonal surge. Peels can sensitize the skin and leave it susceptible for sun damage. Melasma which may occur normally in pregnancy can be blamed on the peels. So ideally peels should be avoided. Only peels that can be done after proper counseling are superficial peels like AHA and lactic acid peels.AHA are category B drug so can be safely used in pregnancy.

19. What should be the ideal interval between peels and gap between peels and other procedures?

Gap between 2 peels is generally kept at 2 to 3 weeks. But it also depends on the peel used and patient skin and indication for which we are doing a peel. If we are doing superficial peels like lactic or salicylic peels it is fine but for medium depth peels we can keep a 3 to 4 weeks gap.

Especially if the exfoliation with the peel is higher, sensitive skin patients or patient who develops acneiform eruptions post peels, we need to give a bigger gap for the skin to heal itself before we do the 2nd session. Peels usually done for acne like SA peel are best done every 10 days to 2 weeks.

Peels when done with ablative procedures like fractional laser, MNRF or DR need to kept 4week to 8 week apart. Quick sessions do not guarantee quick results. All these procedures cause controlled injury and healing of the skin before initiating a new procedure ensures better results, less adverse effects and are more cost effective treatment.

20. Cosmelan peel

Cosmelan is the peel for the experienced Peeler. A Cosmelan peel is a two-step treatment where the peel or a mask is applied, washed off at home after several hours. Mask can be kept from 8hours to14 hours, more for skin of color than Caucasian skin. Once the mask is removed, a secondary cream cosmelan 2 is applied to protect new skin growth for almost 8 to 10 months. Cosmelan 1 may sometime be repeated after 2 months. Actual ingredient of the cream like coca cola is not revealed by the company, mesoesthetic which makes the peel. Cosmelan peel is believed to contain Azelaic, kojic, ascorbic acid and phytic acid.

It gives good results for melasma and hyper pigmentation but hypo pigmentation, rebound pigmentation and over sensitization of skin is known. It costs around 18000, the rate before GST and without much bargaining. Doing a mandelic 30% prior to application of mask improves the results. It must be a planned session, where patient has to be off peels and lasers 14 day prior to the session and 2 months post the session also.

21. Contraindication to peels

Absolute CI will be like active infection like herpes labialis, open wounds, uncooperative and patients with unrealistic expectations.

Relative CI would be prior hypersensitivity to peels, photosensitive disorders like LE, other dermatological conditions like atopic dermatitis. Also, those patients who have received radiation therapy. Medium peels also should not be done in patients on isotretinoin.

22. PHENOL PEELS in Indian Skin -

One point needs to be clarified here. Phenol peels have been used in three different ways (over-and-above the standard Baker-Gordon (BG) formula, which is totally inappropriate for Skin of Color). Firstly, Modified Phenol-Croton Oil [MPCO] peels. the BG solution contains the highest conc. of Croton oil [2.1%], an epidermal vesicant which dramatically enhances the depth of phenol peels. MPCO peels contain not only lower percentage of phenol [30-45%], compared to 50% in BG solution, more importantly they employ very low conc. of Croton oil - 0.1-0.7%. These are available as Hetter's, Exoderm, and Stone 2 ranges abroad and still not suitable for SOC due to possibility of hypo pigmentation, PIH, as well as cardiac adverse effects if large amount used. Secondly, 88% phenol, which qualifies for medium depth peel, is also unsuitable for direct use as a peel for pigmentation/acne etc. The most established use of 88% phenol is as a therapeutic wounding agent for vitiligo, Alopecia Areata and IGH. The only other way 88% phenol can be used is, as CROSS technique, but that too carries risk. Now it is important to understand that phenol has a unique characteristic. Akin to higher dilutions of homeopathic remedy providing higher potency, phenol 45-55% is much stronger (depth wise) than 88%. Phenol 88% causes immediate epidermal coagulation that limits its penetration to upper reticular dermis making it a medium-depth peel. Diluted phenol 5-55%, on the other hand penetrates much deeper as it becomes a strong keratolytic, disrupts sulphur bonds and goes down till mid reticular dermis, qualifying for DEEP PEEL. The only approach suitable for SOC is the CFPC peels - Croton oil-Free Phenol Combination peels like No Melan Fenol of MedidermaIndia or FMC peel of Leaderma; these consist of 10-15% phenol, with 8-10% TCA, and 1-5% of multiple other AHA/BHA combos. They serve well, and the depth can be controlled by - 1) number of coats, and 2) Leave-on time (2-10 hours). A bronzing effect is very common with CFPC peels, which must be foretold to the patient.

Q. Use of EXPIRED PEELS??

Rightly mentioned - alcohol vaporization renders SA peel stronger and stronger, and phenol vaporization renders NMF peel weaker. So Dr Niti's idea of storing in smaller homeopathy bottles is very practical

Q. PEELS in MACULAR AMYLOIDOSIS?

DMSO, as a peel in MA apart from giving it for home use, Superficial peel with 4-5 minutes of GA 35-50%, followed by DMSO works well and then home use of incremental DMSO 50-75-100% solution can be continued.

Q. PEELS IN ACANTHOSIS NIGRICANS?

TCA is a good peel because it ablates the hyperkeratotic epidermis. But, SA-based peels are better, as they tend to accumulate in the HK epidermis and provide prolonged effect.

Q. Documentation?

Take photos in THE SAME ANGLE and light - fussy patients would always find faults even if results are clinically visible. The best is to keep the last photograph in front of you, while clicking the result-photo, to ensure it is in nearly the same angle.

ACAD EXPERT SESSION

"P.C.O.S., Hirsuitism and Optimizing Outcomes of Laser Hair Reduction"

Dr. Apratim Goyal fromMumbai

Dr. Rajetha Damisetty fromHyderabad

Dr. Rekha Singh alsofromHyderabad

Dr. Sheena Pandey from Kanpur

Co-moderator

Dr. Rekha Singh

(18/10/2017)

1. PCOD or PCOS: Do the semantics matter?

The semantics certainly matter! The pathology of the syndrome in question is not limited to the ovaries. Young women with PCOS have a significantly higher risk of type 2 diabetes, cardiovascular disease and endometrial carcinoma. Using the word "ovarian disease" makes it easy for many clinicians to ignore the consequences of insulin resistance and metabolic syndrome that frequently co-exist in this condition.

It's been three centuries (1721) since Vallisneri, an Italian scientist, described a married, infertile woman with shiny ovaries with a white surface, and the size of pigeon eggs. It's time to think beyond the ovaries.

There is also a strong argument about getting rid of the word "ovarian" in the nomenclature. "We believe the name "PCOS" is a distraction and an impediment to progress. It causes confusion and is a barrier to effective education of clinicians and communication with the public and research funders. The name focuses on a criterion—polycystic ovarian morphology—which is neither necessary nor sufficient to diagnose the syndrome. We believe it is time to recognize the advances that have been made since the description of the syndrome by Irving F. Stein, Sr., and Michael L. Leventhal. It is time to expeditiously assign a name that reflects the complex metabolic, hypothalamic, pituitary, ovarian, and adrenal interactions that characterize the syndrome and their reproductive implications. The right name will enhance recognition of this major public health issue for women, educational outreach, "branding," and public relations and will assist in expanding research support."

2. How do you differentiate between generalised hypertrichosis and hirsuitism?

The key to this is the distribution of hair. If the distribution of excess hair is limited to that seen in a male, you'd call it hirsutism. Excess terminal hair on the chin and the lower abdomen is the most sensitive predictor of hirsutism.Also, a change in the rate and form of growth in those areas mentioned in the modified FerrimanGallway scoring is diagnostic of hirsutism. So a woman who was comfortable threading her upper lip once a month needs to do it every fortnight can be labelled as hirsute even if the density and coarseness upper lip hair 'seems normal' to the clinician.Hirsutism is rare in pre-pubertal years. If it begins in early childhood, one should think of an androgen secreting tumor.

3. Insulin Resistance: Association with PCOS and Hirsuitism?

Excess insulin and IGF 1 in the circulation

a. stimulates the ovarian theca to produce more testosterone and worsens hirsutism.

b. Has a permissive role in enabling the binding of androgen to the androgen receptor.

c. If insulin resistance isn't controlled, the full benefit of androgen control wont be experienced.

This is an over simplified explanation.

4. Hirsuitism: how should we look at it as a dermatologist- A localised trouble Or an Alarm ringing just before a big trouble?

   Hirsutism is a sign of androgen excess. The excess could be in the circulation or at the level of the androgen receptor.In patients with hirsutism prior to menopause, almost 90% are due to underlying PCOS. 5% each are due to non-classic congential adrenal hyperplasia and idiopathic hirsutism. Less than 1% are due to tumors (ovarian, adrenal mostly), secondary PCOS or secondary to drugs.Alam bells should ring if hirsutsim is rapid (6-12 months) and severe or accompanied by signs of virilisation and aggressive search done for a tumor. Almost 50% of these tumors are malignant.

5. What should be the ideal, minimum and maximum gap between LHR sessions?

   Ideal gap- Depends on the hair cycle of the area being treated. Ideally you want maximum no of hairs in anagen phase for optimum effect. So it can be 4 wks for areas like face, neck, u/a and around 6-8wks for chest, abdomen etc during intial 2-3 sessions. Post few sessions this gap would increase and a good effective first intense phase would have left at a frequency of around once in 5-6 months for face.

   Minimum gap- in any circumstances you would not want to do before 3 wks. For daily shavers as an exception may be first session at 15 days but not beyond second session.

   Maximum gap- once a good reduction has happened, patients can walk in as late as five yrs from last session to tackle few odd strands remaining in the earlier treated area.

6. How can white hair remaining post LHR be managed?

   · Electrolysis

   · FUE

7. Can LHR be done during pregnancy? Is it safe for pregnant dermatologist to do laser on her patients?

   By logic it can be continued all through the pregnancy but as a blanket contraindication it is usually avoided. If you are being brave and allowing the patient to continue make sure you, patient and obstetrician are on the same page regarding this and patient has signed an additional informed consent.

   A pregnant dermatologist can do it till last day of her working if she is comfortable.

8. Which is the best laser for LHR?

   There is no such thing as best laser which would work on everyone and in every scenario. Remember that sometimes hairs can be technology insensitive meaning a subset of patient require change of technology like not responding at all on diode but excellent response with a single session of IPL / NdYag. So if by 3-4th session if you see absolutely no response be open to using a different type of wavelength. At the end of the day all machines work well in good knowledgeable hands and our results are many a times combination of art and science of it.

9. How relevant are LH and FSH ratio and AMH levels in PCOS? What is the sermatologist's consensus owing to new developments in investigational modalities?

Role of LH and FSH are of very little relevance in PCOS as they are mainly for ovarian reserve and ovulation induction which may be more of relevance to Gynaecologists

AMH is important as it can play as a surrogate marker in patients with PCOS for diagnosis. This is helpful whenever USG pelvis is normal and testosterone levels are well in lower limits. However right assays are not available currently.It is let to the comfort of the dermatologist to treat or refer the patient to an endocrinologist.

10. How to diagnose PCOS in adolescent girls? Should polycystic morphology of ovaries in adolescent girlsalways be considered as PCOS?

Adolescent PCOS merits special attention. In an adolescent, USG can only be done transabdominally. The emerging imaging technique for all PCOS, especially adolescents is MRI pelvis, but the ovarian volume cut off by MRI has been suggested to be 14 ml, instead of 10 ml by USG. Serum AMH is helpful too.

Menstrual irregularities with anovulatory cycles and varied cycle length are common due to the immaturity of the hypothalamic-pituitary-ovarian axis in the 2- to 3-year time period post-menarche. Persistent oligomenorrhea 2 to 3 years beyond menarche predicts ongoing menstrual irregularities and greater likelihood of underlying ovarian or adrenal dysfunction. In adolescent girls, large, multicystic ovaries are a common finding, so ultrasound is not a first-line investigation in women <17 years of age. Ovarian dysfunction in adolescents should be based on oligomenorrhea and/or biochemical evidence of oligo/anovulation, but there are major limitations to the sensitivity of testoterone assays in ranges applicable to young girls. Metformin is commonly used in young girls and adolescents with PCOS as first-line monotherapy or in combination with OCPs and anti-androgen medications.

In lean adolescent girls, a dose as low as 850 mg daily may be effective at reducing PCOS symptoms; in overweight and obese adolescents, dose escalation to 1.5 to 2.5 g daily is likely required.

Anti-androgen therapy in adolescents could affect bone mass, although available short-term data suggest no effect on bone loss.

11. What is secondary PCOS?

When features suggestive of PCOS are there secondary to other endocrine or metabolic disorders, adrenal or ovarian androgen secreting tumours, or because of medications like anti- epileptics, it is called secondary PCOS. Since these can be fatal, it's important to diagnose it early and treat.

The concept of secondary PCOS implies that there is a primary well-defined cause leading to the PCOS phenotype with underlying androgen overproduction, regardless of the origin. In all these conditions, the potential complete recovery of the hyperandrogenemic state as well as the remission of the PCOS phenotype should follow the removal of the cause.

12. Are there some advanced hormonal therapies besides OCP, Spironolactone and finasteride/ dutasteride and n acetyl cysteine for hirsutism in general and PCOS associated hirsuitism?

There are at least three drug classes which have effect on reduction of hyperandrogenemia, but only the latter two provide clinical benefit:

1) STATINS - they do result in reduction of testosterone levels, but do not impact the cyclical abnormality. But yes, they are helpful in metabolic syndrome - reduction of TG, non-HDL cholesterol and hsCRP.

2) BICALUTAMIDE - the next-generation congener of flutamide. Liver safety better. Off label use as 25 mg/day X 3-6 months has shown reduction in hirsutism. Indian brands - ANDROBLOK 50, BIPROSTA 50 mg. Worth adding onto aldactone.

3) EXINATIDE - An incretin mimicker, it acts via glucagon receptors. Typically approved for diabetes, off label add in use in combination with metformin for those not responding to high dose metformin alone. Improves metabolic and hormonal profile. Issues - has to be administered S/C in doses of 5-10 mcg BD. Available as BYETTA. A 250 mcg pre-filled pen costs around 7,500-8,000 INR. It'scongener, lirunatide is also available now.

PHQ-4 score must be recorded to know about anxiety-depressive disorder.

The results of LHR are better if metformin has been started, but that should not delay the first session for a patient whose primary concern is hirsutism. Prioritization as per patient's needs is prudent.

Metformin has well defined insulin independent benefits too. So it should be offered to every patient of PCOS, irrespective of insulin levels.

Although mild hyperprolactinemia is common in PCOS patients, Cabergoline 0.5 mg/week X 3 months should be added on in any PCOS patient with hyperprolactinemia - it helps in reducing metabolic syndrome too.

13. What is the role of eflornithine along with Laser hair reduction? Should it be used or not?

Eflornithine and laser for hair reduction are contradictory in their functions.

Eflornithine slows down hair growth making either the laser less effective as lesser hair would be in anagen for targetting or will give pseudo satisfaction where hair growth will increase again after stopping application as laser could not target hairs properly.

Only valid indication would be the case where patients who due to some reason can't do laser or continue their already on going laser sessions.

14. What age would one consider as ideal for starting a laser hair reduction especially in young girls or rather what age should one use as a cut off for doing laser hair reduction?

Minimum age of starting laser- ideally one should wait at least 2 years post menarche to allow for the maturation of growth to be targeted and also the menstrual irregularities to settle down.

However, it is better to start after the age of 18 years. If laser is being started <18years, informed consent should be taken from the parent/guardian.

15. What is the role of stromal thickness in pathophysiology of PCOS?

Ultrasound measurement of ovarian stroma is a predicting factor of hyperandrogenism degree, prothrombotic factors and cardiovascular risk in patients with PCOS.The evaluation of the S/A ratio (stromal volume/total area) can differentiate between PCOS and control or multifollicular women with both a sensitivity and a specificity of 100%. Furthermore, this ultrasound parameter is strictly related to hormonal milieu and to anthropometric characteristics.

16. What is the role of dairy and high glycemic food in PCOD?

Diet reduces serum AMH in association with decreased androgen levels in obese women with PCOS. Increased serum AMH may be used as a marker of ovulatory dysfunction and hyperandrogenism but not as a marker of insulin resistance.

17. What is the role of medical therapy in management of hirsutism- indications, contraindications, drugs, dose, duration and adverse effects?

Lifestyle therapy constitutes the first step in the management, especially when excess body weight is associated with hirsutism. Pharmacotherapy is frequently used to treat the most predominant manifestations in each age group, such as irregular menses and hirsutism in adolescence, fertility problems in adulthood, and metabolic problems and risk of cancer in old age.Oral contraceptives (OCPs) can effectively lower androgens and block the effect of androgens via suppression of ovarian androgen production and by increasing sex hormone-binding globulin.

Anti-androgens can be used to block the effects of androgen in the pilosebaceous unit or in the hair follicle. Anti-androgen therapy works through competitive antagonism of the androgen receptor (spironolactone, cyproterone acetate, flutamide) or inhibition of 5á-reductase (finasteride) to prevent the conversion of Testosterone to its more potent form, 5á-dihydrotestosterone. The choice of antiandrogen therapy is guided by response and symptoms.

Metformin is commonly used in young girls and adolescents with PCOS as first-line monotherapy or in combination with OCPs and anti-androgen medications. In lean adolescent girls, a dose as low as 850 mg daily may be effective at reducing PCOS symptoms; in overweight and obese adolescents, dose escalation to 1.5 to 2.5 g daily is likely required. Anti-androgen therapy in adolescents could affect bone mass, although available short-term data suggest no effect on bone loss.

Duration of therapy should be continued for at least for the first 6 sessions of laser hair reduction for optimum results

Rule out thromboembolic disorder history before starting OCPs. Better to check serum electrolytes and serum creatinine when higher doses of spironolactone are chosen (>100mg/day). Advice on refraining from high potassium rich food to be given.

18. Is there any use of herbal phyto products used by gynaecologists in PCOS?

There are articles on interventions including herbal extracts of Vitexagnus-castus, Cimicifugaracemosa, Tribulusterrestris, Glycyrrhiza spp., Paeonialactiflora and Cinnamomum cassia. Endocrine outcomes included reduced luteinising hormone (LH), prolactin, fasting insulin and testosterone. There was evidence for the regulation of ovulation, improved metabolic hormone profile and improved fertility outcomes in PCOS. There was evidence for an equivalent effect of two herbal medicines and the pharmaceutical agents bromocriptine (and Vitexagnus-castus) and clomiphene citrate (and Cimicifugaracemosa). There was less robust evidence for the complementary combination of spirinolactone and Glycyrrhiza spp. for hyperandrogenism.

Preclinical and clinical studies provide evidence that six herbal medicines may have beneficial effects for women with oligo/amenorrhea, hyperandrogenism and PCOS. However the quantity of pre-clinical data was limited, and the quality of clinical evidence was variable. Further pre-clinical studies are needed to explain the effects of herbal medicines not included in this review with current clinical evidence but an absence of pre-clinical data.

ACAD EXPERT SESSION

"Hair Transplantation: - Fears, Technology and Advantages"

Dr. Venkatram Mysore

Dr. Venkatram Mysore

Dr. Kavish Chauhan

Co - Moderator :- Dr Ganesh Avhad

(02/11/2017 - 20/10/2017)

"Hair Transplantation: - Fears, Technology and Advantages"

1. What is the role of PRP in HT?

   PRP is mainly used as a holding solution. There is strong logic - but evidence is still low. Main limitation is that how long growth factors will act as PRP is supposed to be prepared fresh. It is true that most hair transplant surgeons including me have observed an odd case in which growth of grafts is faster and patients result in 4-5 months. Since the procedure is without side effects, almost every hair transplant surgeon is doing it. Hope full more evidence will soon emerge

2. Who can perform HT?

   A registered MBBS doctor with adequate surgical skills can do hair transplant according to present MCI rules (that's my reading). On curriculum I do not think it's mentioned on any particular specialty curriculum- These two are possibly the most problematic parts about the interpretation too.

   Personally I feel that dermatologists understand all aspects of hair well. If the required surgical skill is acquired 6 months dedicated training is what I advocate), we are best equipped to do FUE than any other specialty. For FUT, more surgical skill is required. The surgeons will definitely be more adept at taking FUT donor strips, but the skill can be learnt with good training.

   As per MCI curriculum Hair transplant is only included in curriculum of dermatology (MD/ DNB).

   Plastic surgery is yet to have any defined curriculum by MCI.

   Hair transplant was invented by dermatologist and most advances in field have been done by dermatologist.

   Current stand of IADVL SIG trichology & HT is that only "qualified" dermatologist and plastic surgeons should be allowed to do hair transplantation.

3. Should the patient be continued on minoxidil and/or finasteride post hair transplant?

   Yes, unless he was in stage 6 or 7 at the time of surgery.

4. What grade of hair loss is right for hair transplant?

   At least stage 3, but there can be exceptions in special cases with professional requirements. In some cases it can be done in grade 2 if expectations are realistic and family history is not of aggressive/extensive baldness.

5. Scar, morphea, burnt out DLE, excised nevus sebaceous, old fibrotic alopecia areata- Would you do HT in such cases?

   Yes, after a test patch, with low density of around 20 followed by another session after 6 months.

If it's a secondary cicatricial alopecia (like burn, electrocution scars) then response is excellent (one result is attached) done in 2 sitting). Transplant needs to be done in 2 or more sittings at low density (20-25 fu/cm2).

Avoid adrenaline in such cases. Primary cicatricial alopecia cases should be stable for >1year .

6. Can a patient who has undergone HT once, can undergo second HT session?

Yes. But no FUT after a FUE session. FUE after a previous FUT is perfectly fine. A second FUE after a previous FUE will surely not be yielding as the first. Duration preferred is after 9-12 months at least. Keep a minimum gap of 6 months between 2 sittings. If the 2nd sitting is done in the same area to increase density then preferably a gap of 12 months should be kept.

7. How much minimum time does one need to spend in a team practice before starting HT independently?

There are practitioners who start independent practice immediately after training. A team practice of at least 6 months will definitely add value. Hair transplantation can be a team practice throughout too. One needs minimum 6 month training at a high volume HT clinic to be confident and even after that he should first do some surgeries independently but under supervision of a senior hair transplant surgeon

8. What makes an ideal setting and a team for HT- Our own clinic or a multi-specialty hospital?

If an own clinic has the facilities of an OT which can have sterility maintained, good OT table, lighting, equipments required for hair transplantation and a good team- it is as good as a multispecialty hospital. Individual dedicated hair transplant clinics are most successful worldwide. Multi-specialty hospitals keep the major chunk of surgical income so most hair transplant surgeon will find themselves in more profit if they do their own practice. The clinic should be equipped to handle emergencies and all doctors and staff should be at least trained in basic life support.

9. Robotic hair transplantation machines: opinion on them and investment advice.

Robotic HT machine as of now is mostly a marketing gimmick and their performance is way inferior to expert hair transplant surgeons.

e.g. The graft transaction rate with ARTAS varies between 8-23% as per there publications despite using punch size >1mm while most expert FUE surgeons can maintain TR <5% with punch size of 0.8 or 0.9mm. The extraction speed is also way slower than human hands.

The only advantage with robot is non fatigability and help in marketing.

10. Complications of hair transplant post procedure- immediate, after 15days, after 1 month and after 6 months?

Immediate- Post op edema- eyelids, upper face, Pain, skin ooze, syncope, bleeding

Short term- Folliculitis, Necrosis, Scalp infection, cyst formation, telogen effluvium

Longer term- Hypertrophic scars, Change in texture and color and elevation of skin, poor growth, paresthesia, patient dissatisfaction

The technicians should only be used for graft separation and graft plantation after proper training that too should be under supervision.

11. What is the ideal holding solution for grafts Normal saline v/s ringer lactate v/s hypothermosol?

Both Normal saline and ringer lactate are equally fine. Though hypothermosol is best, availability and cost wise NS or RL are fine.

Best is hypothermosol if out of body time is >2hrs. IF <2hrs then NS/RL also are equally good

12. What is the ideal temperature we need to maintain for maximum graft survival?

4 degree C

13. Which kind of punch is best Blunt Vs Sharp?

Sharp serrated punches are better and have least transaction rate.

14. Which method is good for extraction Manual -Motorized (PCID, CID), robotic or Neograft?

Motorized FUE is best one can achieve graft extraction speed of 1500 FU/hour with <5% TR with experience.

Manual is way slower.

Robotic (ARTAS) has scoring speed of ~800 FU/hour (actual extraction would be <500FU/hour) with TR 8-23%. So as of now its way inferior then expert FUE surgeons.

Suction assisted machines like neograft gives inferior results due to risk of graft desiccation and also are very slow.

15. What are the maximum grafts we can remove from safe donor area?

In 1 sitting mostly 2500-3000 FU if one follows 25% rule.

16. The most common complication after HT is either edema forehead or eyes and Sterile folliculitis

How to reduce these complications?

Use prednisolone 40mg ABF for 3 days. Use tight dressing on forehead to prevent edema coming onto eyes. Grafts should not be buried should be left slightly above skin.

For edema, use Elastoplasts 3 cm above eyebrows in 2cm width all around. This and steroids (Oral prednisolone 40mg X 5 days) prevents eyelid edema and also forehead edema. Proper implantation techniques reduce chances of sterile folliculitis.

17. What are the best magnifying loupes available in India?

Best is Carl-Zeiss other good ones is Heine.

18. What is best age to undergo hair transplant?

It is usually believed that before age of 25 medical treatments is more effective than after.

The general rule is to wait till 24-25

However, age is one of the factors- not the sole factor- depends on extent of baldness, psychological factors, profession etc.

One should not do hair loss at young age for very early baldness, vertex.

The disadvantage of doing early HT is that he will continue to lose hairs, and will need repeat session- particularly as they don't take medicines regularly.

19. Does hair transplant sometimes cause an impact on the surrounding hairs resulting in increased alopecia?

Yes telogen effluvium or shock loss can happen in surrounding area. It is more common in females around 20-30% in males <5% cases have it. It resolves on its own in 3-6 months. Hair fibers can be used to temporarily camoflouage it.

20. As androgenetic alopecia is generally progressive, should we not strongly recommend the strict continual use of minoxidil in the patients undergoing HT?

It is strongly recommend to start minoxidil 15days post-transplant and to be continued to protect existing hairs except in grade 7 cases.

21. How many months before his wedding should a bridegroom who wants a HT undergo the procedure?

Ideally, 1 year or more. Minimum period should be 6 months.

22. What is the downtime for the patient before he/she can resume work?

2-3 days in FUE. It may take 2-3 weeks for crusts to fall off so cosmetic impact may be there till that time but functionally patient is fine just on next day. In FUT more physical restrictions are required for a month to protect stretching on sutures.

ACAD EXPERT SESSION

"Recalcitrant Dermatophyte Infection"

Dr. Ameet Valia, Mumbai

Dr. Shital Pujari Amin, Mumbai

Dr. Abir Saraswat, Lucknow

Dr. Ankur Talwar, Lucknow

(23/11/2017-04/12/2017)

"Recalcitrant Dermatophyte Infection"

1. How much minimum time does one need now a day, to treat a case of Tinea to cure clinically?

   There is no fixed duration but oral antifungal need to be given for at least 4 to 6 weeks duration. A static drug like itraconazole requires a longer duration of therapy when compared to a cidal drug like terbinafine. Continue the patients on topical anti fungal even after clearance of lesions. Follow the rule of 2: Topical antifungal two times a day, till two cm beyond the boundary of lesion, till two weeks after the clinical clearance.

2. Why is it that there is a sudden surge in the cases of Tinea out of proportion? What are we seeing at present? Is it a different disease then the one we use to see 10 years before?

   Possible causes are:

   - Inadequate therapeutic levels of drugs: either due to inadequate compliance or questionable quality of drugs or inadequate prescription dosage as well as duration: It is a proven fact that repeated exposure to low levels of antifungal results in emergence of resistant strains. Dermatophytosis is now less confined to hot, humid flexural areas as used to be earlier. It can infect any part easily and spread easily among family members inspire of strict traditional precautions.
   - Some changes in strains, needs microbiological studies
   - Incomplete treatment might be an important factor
   - Misuse of steroid containing combination creams
   - Change in lifestyle, food habits (More of tight fitting clothes, increasing obesity)

3. How do you manage the cases of Tinea in the current difficult recalcitrant case scenario?

   Counselling regarding not wearing tight clothes, reduction of weight, compliance of antifungal drugs

   Dosage of drugs: ITZ: AT least 100mg BD(5mg/kg), TRB: 250mg BD (Adult dose) , Micronized Griseofulvin 500mg BD (20mg/kg)

   In recalcitrant cases or very extensive Tinea: ITZ 100Mg 2 cap BD

4. Do you think there is a difference in the scenario of Tinea in India and elsewhere on the globe?

   Yes, there are no reports of such epidemics from other countries.

5. When can we combine two systemic antifungal drugs and for how long the combination can be used ?

   These combinations are unscientific. There is so much wrong about this combo:

   - Increased risk of hepatic derangement
   - Itraconazole is in a tablet form

- Higher side effect profile
- Improper dose equivalence for eg. For Itra 200mg per day, we will have to give 500 mg terbinafine alongside according to this combo.

6. When to change antifungal if there is no clinical response?

   Give a fair trial of at least 4 weeks in appropriate doses before calling an infection resistant. Ideally wait for at-least 4 weeks before deciding to change. However if absolutely no response in 2 weeks, one can switch over especially if the first drug given is TRB.

7. What about antifungal powder and soap?

   These are at best adjuvant. No specific curative role, May be prescribed according to the needs of the situation. Some advocate their avoidance as this may cause exposure to subclinical doses of antifungal.

8. What about LFT monitoring with systemic antifungal therapy?

   The monitoring may be done in cases where patients report some abnormal symptoms like loss of appetite, weakness and also when patient has been given higher than standard doses. A LFT done at 6 weeks is usually sufficient. Ideally while combining antifungal especially ITZ and GRF, we should do a baseline LFT and repeat after 6 weeks. But do make sure to ask about history of alcohol intake and prior history of jaundice.

   There is an article in BJD wherein the authors searched all literature to assess whether repeat LFTs are essential. The background is that while both US FDA and British National Formulary (BNF) recommend a baseline LFT prior to starting Terbinafine, FDA has done away with the repeat testing recommendations at 4-6weeks, while BNF still recommends it. The authors of this article concluded that terbinafine induced liver injury would always be symptomatic and hence the BNF recommendation is unjustified. However, we wrote a correspondence in response to this (would be online on the journal site soon) citing our experience of detecting highly deranged LFTs on routine monitoring, which we now diligently do, when using the higher dose of 250mg BD beyond 4 weeks. Although the liver injury by terbinafine is known to be idiosyncratic, we think it is still prudent to repeat LFT once at 4-6 weeks, especially with the up dosing so commonly followed now. We also suggested that in fact the recommendation of a baseline LFT may be omitted in an otherwise healthy young adult with no prior history of any liver disease, to make it more economically feasible.

9. Is there any difference in good brands and local ones?

   Yes. There is a huge variability between the pricing of different brands of itraconazole. But products from standard companies deliver predictable and consistent results.

10. What is the ideal topical oral treatment in first trimester of pregnancy?

    All topical antifungal except selenium sulphide, are safe in pregnancy. The choice is entirely yours.

11. What is minimum age to start itraconazole?

Itraconazole has safety studies from the age of 6 months onwards while terbinafine has been approved from the age of 2 years onwards. The problem is in administration to small children who do not swallow the capsule and itraconazole doesn't work if the capsule is opened and given with honey etc. So practically itraconazole can be given only once the child starts swallowing the intact capsule.

12. Should we avoid itraconazole in every cardiac patient?

Itraconazole has a negative inotropic effect. It needs to be avoided in cardiac patients with a low ejection fraction. There is no blanket ban in all cardiac patients. Further drug interactions of itraconazole with certain drugs need to be kept in mind as these may lead to ventricular arrhythmias. Itraconazole may also prolong the QT interval, and therefore co-administration with other drugs that also increase the QT interval is contraindicated.

13. Why we should know separate about Dermatophytosis in children - is the triggering factors are different , is the clinical manifestation are different, is the diagnosis is different or is the treatment and prognosis is different?

It has been observed that clinical clearance is faster in children and recurrences are less in present scenario. Clinical manifestations may be limited and prognosis is good. Nowadays we are seeing chronic and recalcitrant T. corporis and T cruris in infants and children. Fluconazole works at higher doses; like 150 mg alternate days preferably when combined with a new generation topical. It works in pediatric age group and is quite safe. Terbinafine is all or none; when it works, it works well

14. What are the five general lifestyle/ finite instructions that you tell your patients with tough to treat tinea corporis/ cruris?

- Take bath twice daily with any standard soap.
- Wash garments and undergarments in hot (preferably boiling) water and dry in sun.
- Take itraconazole after food and preferably with lime juice.
- Do not share towels / undergarments / socks.
- Change socks daily.
- Iron your undergarments from inside.
- No tight clothing (Easy to recommend but very difficult to implement especially in youngsters)
- No sharing of clothes/separate washing of clothes
- Hot ironing of clothes
- No hot water baths (It is a common practice in lay public to take hot water baths for any kind of skin infections)

One quotation from a recent review in IJD "T. rubrum survived for <12 weeks on a towel while T. mentagrophytes survived for >25 weeks on towel. This fact highlights the importance of disinfection of clothes which could be best done by washing in hot water at 60°C and drying in sunlight, as sunlight is considered to be the most effective disinfectant for dermatophytes.'

15. How important is the biofilms in this scenario? Is it just jargon or something we must tackle clinically?

As of now, there is no evidence supporting the formation of biofilms by fungi. This is just a hypothesis that we propose to explain the treatment failure. Not yet on skin per se, but confocal scanning electron microscopy at > 1000 X has been used to see and characterize biofilms on medical paraphernalia like ET tubes.

16. What is the utility of microbiological study of fungus?

Unfortunately there is a massive gap between the in vivo and in vitro results as far as fungal studies are concerned. The in vitro results do not correlate well with clinical outcome. Mere measurement of MIC does not ensure drug efficacy and doesn't take into account host factors, pharmacokinetics of the drug and drug levels achieved in stratum corneum. A greater standardization is needed before these tests become of greater value. We need to define the cut off levels/ breakpoints of MIC of antifungal in India.

17. Company claims that bio avaibility increases due to addition of beta-cyclodextrinin tablet. Is it true?

Cyclodextrins are actually sugar molecules that are obtained from starch. These molecules have a hydrophilic and a hydrophobic end. These are very useful in delivery of hydrophobic drugs like terbinafine. They can form complexes with hydrophobic compounds. Thus they can enhance the solubility and bioavailability of such compounds. Cyclodextrins can also enhance drug permeability through mucosal tissues. But the formulation of ITZ in a tablet formulation itself is questionable.

18. What about applying emollients in tinea? Will it not increase messiness and moisture?

Emollients do help to restore the skin barrier which may be having been damaged due to indiscriminate use of steroids. This in turn may help in clearing of fungal infections. Reduction of dryness leads to alleviation of pruritus as well.

19. Can itraconazole be given to lactating mothers?

Pregnancy

ALL topical antifungal agents are considered safe in pregnancy since there is very little systemic absorption.

Amongst oral antifungals, Terbinafine is category B and is considered safe after the first trimester. All oral antifungals to be avoided in the first trimester. Itraconazole is category C and is to be avoided at all stages of pregnancy. Itraconazole has been associated with birth defects in animal as well as human studies.

Lactation.

Once again, all topical are safe as very little is secreted in breast milk.

As for oral antifungal, there is little data. Terbinafine is secreted in breast milk but has not been associated with any infant toxicity (categorized as L2 - relatively safe) hence can be given albeit for short duration only (current recommendations limit long term use). Itraconazole, although classified as L2 has been found to concentrate heavily in breast milk. It is a drug better avoided during lactation although there have been no reports of infant toxicity to my knowledge. The maximum safety data during lactation is with Fluconazole (again categorized as L2) hence should be preferred wherever feasible.

20. What is the place of Griseofulvin in today's era?

There has been a recent upsurge in the use and marketing of Griseofulvin, mainly due to the lack of other options. However, Griseofulvin is a very weak fungi static drug. The bioavailability after oral ingestion is very low (around 30 - 40%). Further the levels achieved in stratum corneum are also not adequate. The MIC values for this drug are also very high as compared to any other antifungal. It is best reserved for Tinea capitis where it is the drug of choice since it achieves much higher levels in the hair due to some yet unknown mechanism.

While Griseofulvin seems to show some effect in doses of 1000 mg/day, it has produced a headache in all the patients I have tried it in. Also, their disease has not resolved even after a month of treatment.

21. Levamisole as immunomodulators: any role?

No documented benefits, Non specific action. There are no reports at its entire role in management of Tinea.

22. Chemical peels for Tinea: - when, how much, which chemical, to do or not to do?

We have better modalities available for management of Tinea. A superficial peel removing the upper layers of stratum corneum will have marginal benefits at best. It is impractical and way too costly. Rather hazardous if done over large areas. Let us not promote this.

22. Do we need psoriasis like protocol for Tinea management now depending upon the BSA of the Tinea involvement?

It will be needed very soon. However, oral and topical antifungal combination has become a need for small and large, all types of Tinea, a protocol depending on BSA and agents of choice would add more hands to the treating doctor.

23. Any experience with Voriconazole in refractory cases?

There are very little studies as such about the efficacy of voriconazole in Dermatophytosis, However, the few that we have demonstrated good in vitro efficacy of voriconazole against dermatophytes. The MIC levels are higher than itraconazole. However, one study showed lesser percentage of resistance to voriconazole as compared to itraconazole. The problem lies in ascertaining whether the in vitro efficacy turns into clinical efficacy as well. The standard anti

fungal achieve much higher concentration in the skin as compared to serum levels, because they are secreted in the sebum. On the contrary, the skin levels of voriconazole are much lower than the serum levels because it doesn't follow the same pharmacokinetics. Hence it requires a loading dose followed by maintenance dosing. How it impacts the efficacy, is yet to be fully determined in clinical scenario.

24. What is your take on pulse anti fungal therapies and stat dosing patterns?

Pulse therapies should be restricted only for nail infections which have a longer reservoir effect. In case of cutaneous Dermatophytosis, the reservoir effect of itraconazole is only 7 to 10 days so pulse regimen doesn't hold well. But yes in the present epidemic, pulse therapy is not recommended

25. Existing causes of immunosuppression like DM/ HIV/Hep B/Hep C presenting with wide spread lesions of Tinea. Do you modify your treatment regimen in them with up dosing and drug interactions?

Rule out drug interactions. Nevirapine and efavirenz decrease efficacy of itraconazole. Most DM patients respond well to standard therapy. Most oral antifungal do not have any interactions with anti viral used for hepatitis infections. However, caution must be exercised.

DM: Oral hypoglycemic: Not much interaction. But monitor blood sugar levels

HIV: AKT in HIV patients: Rifampicin reduces azole levels.

Terbinafine: better option in these cases

HEP B and HEP C: Azoles, GRF and TRB , all are not recommended in liver dysfunction; this is a nightmare situation; one of the indications of only topical antifungal.

26. What is your take on usage of the superior antifungal for benign conditions like T. Versicolar and Candidal infections which are easy to treat with the topical alone and proper dosing with old molecules.

Fluconazole works well here. Cost effective too.

ACAD EXPERT SESSION

“Vitiligo - Surgery”

Dr Somesh Gupta

Dr Imran Majid,

Dr Umashankar

(13/12/2017 - 25/12/2017)

"Vitiligo Surgery"

1. What according to the experts is the current definition of a stable disease? Who according to you is an ideal patient to undergo vitiligo Surgery?

   As far as the 2nd part of this question goes, any patient with segmental vitiligo or a localized vitiligo on face, neck, trunk or proximal limbs, not responding to medical management is an ideal candidate for vitiligo grafting.

   Now, as for the first part about stability in vitiligo, there is still no absolute consensus as of now.

   There are two main debatable issues in vitiligo stability for grafting procedures

   a) The minimum period of stability that qualifies a patient for vitiligo grafting. Different authors have used and recommended different durations ranging from 6-months to 3 years. The IADVL recommendation is however to wait for at least 1 year stability of disease before undertaking surgical management in vitiligo.

   b) The relative importance of lesional v/s disease stability-- we come across patients in whom some vitiligo lesions have been stable for years but there are some fresh lesions occurring elsewhere. Should the stable patches in such cases be taken for grafting is again an issue without a definite consensus? We at ACSI conducted a multicentric study on this controversial topic and the results from this study were in favor of 'lesional stability' being as relevant as disease stability. This study was published in JCAS in 2016.

2. In what sites/scenarios should a dermatologist not undertake vitiligo surgery?

   A-While there are really difficult sites for vitiligo grafting; no site qualifies as an absolute contraindication. The most difficult site, bordering on being an impossible site, is the palm or sole. Rest, all sites can be managed by vitiligo grafting. Acral areas like fingers toes are the next most difficult sites to graft surgically after palms and soles. Genitals also are really challenging sites.

3. For non-culture or culture melanocytes transplantation, what is the method of choice for dermabrasion?

   The choice of dermabrasion in any vitiligo grafting procedure depends upon

   a) Site of vitiligo

   b) Facilities available with the dermatosurgeon

   One can use manual dermabraders in most of sites to prepare the recipient bed. However, acral areas pose a challenge and these sometimes need either a resurfacing laser like CO2 or Er:YAG to dermabrade the surface properly. Eyelids and genitals also pose challenges for dermabrasion. Motor dermabrasion is easy in most of the cases particularly while performing surgeries in larger areas. But the depth should be adequate. It should neither be too deep nor too superficial.

One of the causes for failure in ECS surgery is inadequate dermabrasion. Fine bleeding points should be the end point.

Cryo blister method can be followed to prepare recipient area on finger or toe.

As skin on eyelids and genitalia are thin, additional care has to be taken. Manual dermabrasion would be appropriate on these areas for those who are not familiar with motor.

For dermabrasion using a CO2 or ErYAG laser we normally use the ultrapulse mode

It is better to use the laser in a slightly defocused mode. The fluence usually ranges from 8 to 15mJ/cm2 depending upon the area to be derma braded. Use saline soaked gauze to wipe off the vaporized tissue and don't go too deep to avoid scarring.

4. What is the role of steroids/cyclosporine immediately post-surgery and how long do the experts continue them?

   Ideally, there should not be a need for immunosupressants in vitiligo grafting. If one is grafting a stable vitiligo patient whose disease has been stable for at least a year, we don't need any steroids or immunosppressants in such a case. And if we are not sure about stability we should ideally not graft such a patient.

5. It is difficult to transplant cells during vitiligo surgery, especially at finger tips, genitals etc. Do you use any special dressings for these areas as most of the regular dressings do not or cannot stay on these areas for a week or more?

   Please also elucidate the dressing technique you use after non culture or blister grafting of lesions of vitiligo on the lips.

   For the lips we can use either a stay suture for keeping the dressing in place or even cyanoacrylate glue at the edges of the graft.

   Dressing for the fingers is a bit challenging but can be accomplished by using the traditional pressure dressing like Leukoband keeping in mind that the hand movements are restricted by the dressing itself

   Regarding dressing following cellular graft on lip - most of the cells will be taken up on recipient area within first 2 hours. So we may avoid secondary dressing for 2 hours. Moreover, as dry collagen dressing is usually used, it retains the cells on the abraded site and thus minimizes failure chances.

6. What are the important precautions, does and don't to be followed to get uniform pigmentation especially in NCMT.

   Dermabrasion should be appropriate and uniform.

   1. Suspension should be spread out evenly on the recipient area..
   2. As per the studies, approximately 4 to 5 melanocytes in a drop of suspension is ideal to get uniform results which is practically difficult to monitor. Ideally measure donor to recipient ratio which has to be 1:8.

More than 90% repigmentation can be considered as excellent results. However, even with best care, avoiding perigraft hallow is challenging.

One very important aspect of this is to take very small pieces of dry collagen dressing, Neuskin-F. Cut them into 2-4 cm square pieces. As because of surface tension, the suspension tends to get collected at the periphery of the collagen dressing. If we take very large pieces, it is difficult to achieve uniform pigmentation.

Another very important aspect is resuspending the cell suspension into very small amount of PBS or Ringer Lectate or DMEM. This allows the suspension to be retained at the patch and it doesn't drain off and we get uniform pigmentation even on curved surfaces.

Simple sun exposue is good enough post-transplantation. Tacrolimus 0.1% can be started soon after complete healing along with sun exposure.

7. How to ensure than the melanocyte rich solution does not drain off while placing it over the recipient site?

Best is to add Hydroxy Propyl Methyl Cellulose (ophthalmic solution) in the concentration of 1:4 to the final suspension. Viscosity can also be increased by using hyaluronic acid. Recently we have been using patient`s own blood plasma in place of DMEM which also increases the viscosity of the suspension and give equally good results.

Use of dry collagen dressing will also ensure the retention of cells.

8. Should we start PUVASOL or NBUVB post procedure especially in outstation patients?

Yes. Please refer to nearby hospitals which have phototherapy and start NBUVB 2 weeks post operatively. Most of the patients in fact responds well within 6 to 12 weeks. PUVASOL can be an alternative if phototherapy unit is not available.

PUVAsol or NBUVB is not needed in every case of NCES or tissue grafting. It is better to wait for a few weeks after the surgical procedure and look for spontaneous repigmentation. This helps to avoid hyperpigmentation of the recipient area. If one feels that there is a delay in repigmenttaion one can start any form of phototherapy like NBUVB or PUVAsol. Personally I prefer to use Excimer light or Targeted UVB in such cases. One important advantage of using Targeted phototherapy is that the incidence of perigraft halo is minimized (personal observation, not published yet)

9. By what time do the experts expect that we will have readymade melanocytes to be transplanted on the derma braded skin like the various readymade and ready to use stem cell preparations?

Synthetic melanin is available. It used to be very expensive, but recently its price has dropped substantially and it can be made to match various shades of Indian skin.

However the scientists who have made it think its main indication to be in camouflage as due to the skin turnover any intra epidermal melanin will keep getting lost.

10. Any special tips to prepare recipient area over eyelid for NCMT?

Laser dermabrasion for recipient area

Use manual dermabraders preferably on eyelids

If one has to use motorized dermabraders, one should use cylindrical or conical burs.

Eyelids are very thin- need very superficial dermabrasion. The skin is pulled against the bony surface when we dermabraders. The dermabrasion should be minimal- barely bleeding or not bleeding at all.

The repigmentation on eyelids is generally excellent unless the vitiligo is unstable.

11. Laser dermabrasion for recipient area. Is it better than motor dermabrasion? What parameters to use to get the right depth?

Laser dermabrasion is as good as motor dermabrasion and there are no studies that have documented any superiority of one above another. However, laser dermabrasion is useful when one is grafting the thick acral areas where dermabrasion is the most difficult. As a rule don't over-dermabrade with laser. It is safer to be conservative and then after the initial dermabrasion with laser one can use a manual dermabraders to reach the required depth. There is no evidence that laser is superior; however it may be more convenient. Many vitiligo surgeons feel that dermabrasion is superior as it does not coagulate capillaries, which allows rapid healing and better uptake of melanocytes. However, there is no evidence for this.

12. Very often we are asked about the inheritance mode of vitiligo by the patient.

For example: The girl's father has vitiligo, girl doesn't have vitiligo. Boy wants to marry this girl and then what are the chances that their kid will get the Vitiligo?

How can this be explained?

Vitiligo is a disease with a polygenic inheritance and the presence of vitiligo in an affected parent increases the chances of vitiligo in the offspring marginally. The chances of having vitiligo in an offspring from an affected parent are in the range of 6-8%.

13. What dressings do you prefer after non culture melanocyte transplantation and in what order do you use them?

The first layer- collagen dressing like Neuskin-F.

The second layer- thin gauze pieces.

The third layer- Tegaderm

The fourth layer- Dynaplast- only in mobile areas to fix the movements.

We need to stretch the Dynaplast dressing full to ensure complete immobility which is crucial.

14. Is single cycle of trypsinization enough to provide adequate ORS melanocytes in follicular cell suspension? Why should it be done thrice?

The full thickness of outer root sheath is uniformly rich in melanocytes unlike epidermis, in which only basal and suprabasal layers contain melanocytes. So we proposed to do the trypsinization thrice, 30 minutes each, then pool the three solutions and centrifuge it. This helps improving yield.

No need of antitrypsin, though it improves viability if you use it.

15. What is your opinion about tattooing and camouflage for small patches on visible areas?

Tattoo has very limited indications as the colour changes with time and needs repeated touch up. Areola and nipple, gingiva, are few sites may be considered for tattooing.

For camouflage we have some brands available in India like Dermacolor or CTone. They come in 20 different shades and you can get a near perfect match in more than 90% cases.

These need to be applied each day in the morning and usually last for the whole day. Many of my patients including some VIPs use these camouflage creams regularly and have been using them since years. We have to get a constant supply for these patients.

There are also some brands available internationally which claim to remain for a few days after a single application. These are available as marker pens (a brand from US) or sprays and creams.