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Quarterderm

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INDIAN ASSOCIATION OF DERMATOLOGISTS
VENEREOLOGISTS & LEPROLOGISTS
GUJARAT STATE BRANCH**



Indian Association of Dermatologists, Venereologists & Leprologists,
Gujarat State Branch

Volume - 1, 2013 (Issue 88)

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FROM THE DESK OF CHIEF-EDITOR

New journeys are always challenging as well as interesting. After a successful & highly acclaimed DERMACON2013, our spirit to take new challenges is very high.

When I decided to take over as the editor of our own Quarterderm, fundamental idea was sharing common interest and imbibing knowledge. A smooth path has been already paved by our esteem previous editors; we have the responsibility of maintaining a legacy and plant some beautiful flowers on the way. Quarterderm entering into its young dynamic age of 26th year may witness many changes in this journey. Myself, along with my associate editor Dr. Amit Mistry, Co-editors Dr. Ketuman Joshi and Dr. Santosh Rathod are in a constant process to get our journal indexed from day one.

To begin our association; we present our own Quarterderm issue packed with articles of common interest, regular feature like photo quiz and many more. It goes without saying that zeal and fervour you may feel across the pages is a mere reflection of our spirit as a proud IADVL –GSB member compiling our own Quarterderm. Inside the issue you will discover many new concepts from the depth of basics to breakthrough in medicine, regular photo quiz will be accompanied by some more challenges like crossword or MCQs. Prizes will be given to first two correct entries in each category to stimulate zest for reading and fanatic internet searching which I did while arranging the crossword for this first issue. I am sure our young readers will find this activity interesting.

I hope you will enjoy reading the issue as much as we have enjoyed putting it to gather. Your suggestions are most welcome and are valuable for us. Due respect will be given to best suggestions also. We welcome your articles in all categories.

With pride putting forward the 88th issue of Quarterderm. Thank you all & Happy reading!

Dr. KRINABHARAT PATEL
Chief Editor

INSTRUCTIONS FOR AUTHORS

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- Focus subject for future issues will be displayed on website – www.iadvlgujarat.org for your reference. Articles may be prepared accordingly.

Readers are requested to check the dosages of drugs mentioned in the articles from standard textbooks.
All Precautions have been taken to avoid errors but authors/editors cannot be held responsible for any inadvertent error.

Notalgia Paresthetica

Dr. Krina Patel
Associate Professor & HOD
GMERS Medical College & Hospital,
Sola

Introduction:

"I will scratch your back if you will scratch mine"

Notalgia pareshtetica (NP) is common dermatological complaint characterized by pruritus medial or inferior to scapula in unilateral distribution. It is a sensory neuropathic syndrome of the mid back skin, classically seen located in unilateral infrascapular area. It is a localised pruritus disorder which may present with episodic itching or paresthesia or even pain in mid back area of skin past easy reach of patient.

NP was first described in literature by Astwazaturow in 1934 but the complain of chronically itchy back has likely plagued people since the beginning of time. History shows that for centuries people have complained about an itch between shoulder blades that is out of reach to scratch by themselves. Sometime so maddening is the itch that to find a way to scratch is all consuming. The back-scratcher is a primitive too that even apes have been observed making from tree branches. Elaborate back-scratchers have been fashioned from everything from whale bone to tortoise shell and have been seen to be prevalent throughout time in diverse cultures.⁽¹⁾

NP is typically confined to T2-T6 dermatomes and may have accompanied pain, paresthesia and numbness to hyperesthesia. A lichenified plaque or post-inflammatory hyperpigmentation due to chronic scratching may be evident. It is seen in all races. More prevalent in middle aged females as compared to males and it has not been reported in children. NP lasts for years.

Pathophysiology:

The cause of NP is still unclear. Several possible etiologies have been proposed. The most accepted is that NP is a sensory neuropathy. Others less accepted etiologies suggested are proliferation of cutaneous nerves in affected area, damage to cutaneous branches of the posterior divisions of the spinal nerves. This can occur by impingement from degenerative changes in the spine or spasm in the paraspinal musculature. Pain, paresthesia and numbness can be related to neurological findings but pruritus is an often unrecognised symptom of nerve damage. Muscle spasm or fibrous bands compressing cutaneous nerves have been suggested.⁽¹⁾ Electromyography study to detect paraspinal denervation at T2-T6 in NP patients suggest that because of their anatomical placement as the dorsal nerve roots exit the fascia of multifidus spinae at a right angle en route to epidermis; as a result they are more exposed and prone to injury. Another study by Savk et al showed radiographic findings of degenerative vertebral changes or herniated discs in more than half of study patients of NP in areas that correspond to dermatomal distribution of their symptoms.⁽²⁾ Several other studies have shown significant spinal pathology in NP patients.^(3,4) Additional studies are needed to further assess the relationship of notalgia paresthetica with cervical spinal disease to determine whether this is a casual or incidental finding.

The diagnosis is made clinically on the basis of history and clinical findings. Clinical signs may or may not be there as changes are generally secondary to chronic pruritus in long standing cases. In some cases localised hyperpigmentation, lichenification or macular amyloidosis may be seen. Spinal imaging studies in cases with neurological or musculoskeletal symptoms may be helpful.

Differential diagnosis include tinea versicolor, tinea corporis, contact dermatitis, neurodermatitis, delusional parasitosis, lichen simplex chronicus, and macular amyloidosis with which it has significant overlap.

Histopathology in early cases shows mild

hyperkeratosis and signs of post-inflammatory changes, and mild inflammatory infiltrate of the papillary dermis with variable dermal melanophages. In chronic cases amyloid deposits in dermal papillae may be seen; as a result of damage to keratinocytes from chronic scratching.⁽⁵⁾

Relationship may exist between NP and brachioradial pruritus as both are types of localised pruritus syndrome. Many studies have described association between brachioradial pruritus and cervical spine disease, with possible neuropathic etiology which also support neurogenic association with NP.^(6,7) Theory against this is NP is generally unilateral while brachioradial pruritus could be bilateral.

NP is seen worldwide. No particular racial variation is described. It is slightly more common in females. Generally affects adults of 40-80 year age group. NP is a chronic condition with periodic remissions and exacerbations. Although not a life threatening condition, this disorder may affect patient's quality of life significantly.

Clinical Presentation: [Figure 1]



NP present with hallmark symptom of localised pruritus of unilateral infrascapular area. Skin findings are described as ill-defined hyperpigmented, non-indurated patch on mid back of generally 3-10 CM diameter. Secondary skin changes like lichenification, macular or lichenoid amyloidosis, excoriations, eczematous changes, secondary infection etc. may be seen. Examination of spine may show normal result or may reveal tenderness, decreased range of motion in neck or associated cervical muscle spasm. MRI or radiographic studies may be warranted in such cases.

Treatment:⁽⁸⁻¹⁸⁾

Treatment of NP is often refractory and frustrating for both patient and practitioner.

Antihistamines, topical steroids do not relieve symptoms much as this is neuropathic itch.

Topical capsaicin, EMLA cream, botulinum toxin type A injections, local nerve block, gabapentin, oxcarbazepine and surgical decompression of the nerve have been employed with variable results. Oral NSAIDs and muscle relaxants like metaxalone or cyclobenzaprine may help in patients with spinal symptoms. Thalidomide has been tried occasionally.⁽¹⁸⁾

Several non-pharmacological, non-surgical treatment options have been suggested recently; which alleviate side-effects of above treatments. Transcutaneous nerve stimulation, exercises to strengthen postural muscles and extend the spine, acupuncture, osteopathic manipulative treatment are some of the modalities tried on small number patients but with promising results.

Surgical therapy like cervical block, steroid injections, discectomy with fusion or disk repair techniques may be employed as last resort in patients with significant associated cervical disease.

Patients often get relief by learning that pruritus they are experiencing has a biological cause. In persistent cases that interfere with quality of life even back scratchers that relieve patients is worth trying.

Physical activities that may exacerbate neck spasm like constant work on computers, activities that require excessive forward bending of the head should be avoided in susceptible individuals.

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Pruritus

Dr. Jigna Padhiyar (Senior Resident),
Dr. Nayan Patel (Assistant Professor)
GCS Medical College, Ahmedabad.

Definition:

Pruritus can be defined subjectively as a poorly localized, non-adapting, usually unpleasant sensation which elicits an immediate desire to scratch.¹

Classification:

1. Pruritoceptive (cutaneous, e.g. scabies, urticaria)
2. Neuropathic (due to lesions of afferent pathways of the nervous system, e.g. peripheral neuritis, nerve entrapment, brain tumours)
3. Neurogenic (due to centrally acting mediators which do not damage the central nervous system, e.g. morphine, opioid peptides of cholestasis)
4. Psychogenic, including delusional parasitosis.²

Introduction:

Itch and pain can readily be dissociated since the former elicits the scratch reflex, but the latter cause withdrawal. Although they broadly share similar overall molecular mechanisms and neurophysiological pathways, new evidence indicates the existence of separate dedicated neurones for itch in both the peripheral and central pathways^{3,4}. Chronic itch is multidimensional, the sensation being variably modified by emotional and cognitive components. In dry skin and skin of some atopics, mechanical stimulation may trigger a more persistent itching sensation (**alloknesis**)⁵. The quality of itch varies greatly, ranging from burning, through pricking, to sensations of insects crawling over the skin. The psychophysiological basis of these differences remains unclear.

Pathophysiology:

The sensation of pruritus is transmitted through slow-conducting unmyelinated C-polymodal and possibly type A delta nociceptive neurons with free nerve endings located near the dermoepidermal junction or in the epidermis. These neurons appear

to be located more superficially and are more sensitive to pruritogenic substances than pain receptors. Fibers most concentrated in wrists and ankles. Activators of these nerves include histamine, neuropeptide substance P⁶, serotonin, bradykinin, proteases (eg, mast cell tryptase), Prostaglandins, and endothelin (which stimulates the release of nitric oxide). Impulses are transmitted from the dorsal root ganglion to the spinothalamic tract.

Opioids are known to modulate the sensation of pruritus, both peripherally and centrally. Stimulation of opioid mu receptors accentuates pruritus, while stimulation of kappa receptors and blockage of mu receptors suppress pruritus. External mediators like Skin inflammation, Environmental heat or dryness, Psychological concerns etc also play role.

Mortality/Morbidity

Pruritus causes significant morbidity. Some conditions that cause systemic pruritus appear to be associated with an increased mortality rate. In patients receiving hemodialysis, renal pruritus is an independent marker for mortality at 3 years. Patients with severe, generalized pruritus associated with Hodgkin disease have significantly shorter survival than those with mild or no pruritus.

Causes:

Dermatologic Conditions

1. Dry Skin or Xerosis (most common cause)
2. Urticaria
3. Atopic Dermatitis
4. Allergic Contact Dermatitis
5. Bullous Pemphigoid
6. Dermatitis Herpetiformis
7. Folliculitis
8. Psoriasis
9. Lichen Planus
10. Mycosis Fungoides (Pruritus suggests worse prognosis)
11. Sunburn
12. Local Infection
 - a. Scabies
 - b. Pediculosis corporis (lice)

Systemic

1. Iron Deficiency Anemia
2. Severe Chronic Renal Failure (Uremic Pruritus)
3. Diabetes mellitus
4. Neurodermatitis or Delusions of Parasitosis

5. Polycythemia Rubra Vera (30-50%)
 1. Provoked by hot shower or bath
 2. Pricking type itch may persist for hours
6. Hodgkin's Lymphoma
7. Malignant Carcinoid
8. Multiple Myeloma
9. Scleroderma
10. Malignant Carcinoid
11. Rapid weight loss (e.g. Anorexia Nervosa)
12. Hyperthyroidism (4-11% long-standing Grave's Disease)
13. Cholestasis
 - Chronic Liver Disease
 - Pancreatic carcinoma
 - Primary biliary Cirrhosis (Pruritus in all cases)
 - Intrahepatic Cholestasis of Pregnancy
 - o Affects 0.5% of women in third trimester
14. Systemic infection
 1. HIV Infection (see Pruritus in HIV)
 2. Filariasis
 3. Schistosomiasis
 4. Onchocerciasis (river blindness)
 5. Ascariasis
 6. Hookworm
 7. Trichinosis
 8. Parvovirus B19

Localized causes:

1. Eye

1. Allergic Blepharitis
2. Allergic Conjunctivitis
3. Atopic Dermatitis
4. Contact Dermatitis

2. Ear

Otitis Externa
 Otomycosis
 Ear Canal Dermatitis
 Contact Dermatitis
 Neurodermatitis
 Seborrhea
 Psoriasis

3. Scalp

1. Pediculosis (Head Lice)
2. Psoriasis
3. Seborrheic Dermatitis
4. Allergic Contact Dermatitis

5. Pustule

4. Back

1. Notalgia Paresthetica
2. Xerotic Eczema
3. Psoriasis
4. Folliculitis
5. Cholestasis (Butterfly rash)

5. Arm

1. Brachioradial Pruritus
2. Xerotic Eczema
3. Eczematous Dermatitis (antecubital fossa)

6. Hands

1. Dyshidrotic Eczema
2. Eczematous Dermatitis
3. Contact Dermatitis
4. Scabies (interdigital web space involvement)

7. Groin or inguinal area

1. Pruritus Vulvae
2. Candidiasis
3. Tinea Cruris
4. Erythrasma
5. Contact Dermatitis
6. Extramammary Paget's Disease
7. Intertrigo
8. Lichen Sclerosis et Atrophicus (LS&A)
9. Pediculosis
10. Scabies

8. Rectum

1. Pruritus Ani
2. Anal Fissure
3. Condylomata acuminata
4. Pinworm

9. Legs

1. Xerotic Eczema (shin)
2. Neurodermatitis
3. Stasis Dermatitis
4. Atopic Dermatitis (popliteal fossa)
5. Lichen Simplex (lateral malleolus)
6. Dermatitis Herpetiformis (knee)
7. Cutaneous T-Cell Lymphoma (buttocks and thighs)

10. Feet

1. Tinea Pedis
2. Eczematous Dermatitis
3. Contact Dermatitis
4. Scabies (interdigital web space involvement)

Exposure related Pruritus

1. Water
 1. Aquagenic Pruritus -Intense distressing itch after water contact
 2. Cholinergic Urticaria
 3. Polycythemia Vera (follows warm bath)
 4. Swimmer's Itch
2. Pregnancy
 1. Pruritic Urticarial Papules and Plaques of Pregnancy
 2. Prurigo of Pregnancy
 3. Herpes Gestationis or Pemphigoid Gestationis
 4. Cholestasis associated Pruritus (Prurigo gravidarum)
 5. Pruritic Folliculitis of Pregnancy
 6. Atopic Dermatitis
 7. Contact Dermatitis
3. Medications
 1. Itraconazole, Fluconazole, Ketoconazole
 2. Niacinamide
 3. Vitamins
 4. Aspirin
 5. Quinidine
 6. Ointments with high concentrations of inert oil
 7. Narcotics (especially via spinal administration)
 8. Hypersensitivity Reaction
 1. Rifampin
 2. Vancomycin
4. Allergen or irritant exposure (e.g. Contact Dermatitis)
 1. Heat exposure: Miliaria rubra (Prickly heat)
 2. Cat exposure
 3. Fiberglass exposure (Fiberglass Dermatitis)

Age-related Pruritus Causes

1. Children
 1. Atopic Dermatitis
 2. Contact Dermatitis

3. Lice
4. Scabies
5. Parvovirus
6. Pinworms

2. Elderly
 1. Xerotic Eczema
 2. Contact Dermatitis
 3. Bullous Pemphigoid
 4. Herpes Zoster
 5. Mycosis Fungoides

Investigations:

1. Approach
 1. Base lab testing on history and physical
 2. Avoid broad shotgun approach to lab testing
2. Dermatologic Cause Evaluation
 1. Skin Scrapings for Scabies and Dermatophytosis
 2. Skin biopsy
 1. Mastocytosis
 2. Mycosis Fungoides
 3. Autoimmune Bullous Disease
3. Systemic Cause Evaluation
 1. Thyroid Function Test (evaluate for Hyperthyroidism)
 2. HIV Test for high risk behaviors
 3. Liver Function Tests (evaluate for cholestasis)
 1. Serum Bilirubin
 2. Alkaline Phosphatase
4. Hematologic tests
 1. Serum Ferritin
 2. Blood Urea Nitrogen (BUN)
 3. Complete Blood Count (CBC)
 4. Calcium
 5. Albumin
4. Radiology: Systemic Cause Evaluation
 1. Chest XRay (if Lymphoma suspected)
 2. Right upper quadrant ultrasound (for cholestasis)

Complications of persistent scratching

1. Bacterial superinfection
2. Lichen Simplex Chronicus
 1. Thickened skin in response to repeated scratching

Management:

Avoid precipitating factors

Decrease bath frequency, length, and temperature

Limit soap use

Add Bath Emollients (Bath Oils)

Avoid factors that cause drying -Excessive bathing, Rough clothing, alkali soap etc.

Wear comfortable clothing

Skin barrier protection with Skin Lubricants- Apply immediately after patting dry from bathing, Apply lotion frequently throughout the day.

Avoid rough clothing or fabrics. Avoid vasodilators if provoke itching.

Topical Agents for Pruritus

1. Avoid topical anesthetics and Antihistamines
 1. May sensitize exposed skin
 2. Risk of Contact Dermatitis
2. Avoid Topical Corticosteroids
 1. Risk of skin atrophy
 2. Mild Corticosteroids could be used briefly
3. Standard Topical antipruritic lotions
 1. Menthol/camphor (e.g. Sarna lotion)
 2. Oatmeal Baths (e.g. Aveeno)
 3. Pramoxine (PraxeGel, Prax, Pramoxone)
 4. Calamine (on weeping lesions only, avoid if skin dry)
4. Doxepin 5% cream (Zonalon)
 1. Dose: Apply qid up to 8 days
 2. Highly effective at reducing Pruritus
 3. High rate of Contact Dermatitis with prolonged use
5. Miscellaneous Options
 1. Burow's Solution (Wet Dressings)
 2. Unna Boot (also protects area from scratching)
 3. Tar emulsion

Systemic Antipruritic agents

1. Aspirin
 1. Anti-inflammatory action offer symptomatic relief
 2. Effective if kinin or prostaglandin mediated Pruritus
2. Doxepin (Sinequan)

1. Dose: 25 mg PO qhs
2. Highly effective antipruritic more potent than Atarax
3. Antihistamines
 1. Sedating Antihistamine: Hydroxyzine (Atarax)
 1. No antipruritic effect in eczema
 2. Sedation allows sleep at night
 3. Dose: 0.5 mg/kg up to 25 to 50 PO qhs
 2. Non-Sedating Antihistamine: Cetirizine (Zyrtec)
 1. Metabolite of Hydroxyzine
 2. Reduces Pruritus more than others in its class

Management: Specific Conditions

1. Cholestasis associated Pruritus

Management

1. Cholestyramine
(or Colestipol if Constipation limited)
 1. Has also been used in Pruritus of pregnancy
2. Ursodeoxycholic Acid
 1. Used in primary biliary Cirrhosis (intense itch)
3. Ultraviolet Radiation
 1. May be indicated in refractory cases
4. Ondansetron
5. Opiate receptor antagonist
 1. Naloxone IV
 2. Nalmefene PO

2. Renal Failure associated Pruritus

Management

1. Ultraviolet (UV) Light Therapy
 1. Ultraviolet B
 2. Ultraviolet A with Psoralen (PUVA)
2. Polidocanol (balneotherapy)
3. Activated Charcoal 6 grams per day
4. Topical Capsaicin 0.0255%
5. Cimetidine
6. Cholestyramine
7. Ineffective therapies
 1. Poor response to Antihistamines
 2. No relief with Naltrexone
 3. No relief with Ondansetron

3. HIV Infection related Pruritus

1. Responds to Antiretroviral therapy
2. Consider other causes of Pruritus in HIV

4. Psychiatric Illness related Pruritus

1. Antidepressants
2. Doxepin (Sinequan) 25 mg PO qhs
3. Anxiolytics (e.g. Benzodiazepines)
 1. Consider for short-term bedtime use
4. Pimozide
 1. May be indicated in Delusions of Parasitosis
5. Transcutaneous Electric Nerve Stimulation (TENS)

Prevention: Superinfection from scratching

1. Keep Fingernails short and clean
2. Rub with palms for irresistible urge to scratch

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REVIEW ARTICLE

Managing Itching in Elderly Population

Dr. Krina Patel

GMERS MEDICAL COLLEGE, SOLA

Introduction

Itch is the most common dermatological complaint in elderly people over the age of 65 years. The elderly population is increasing and with its impact of dermatological disorder in elderly is also causing additional burden on health system. Managing itch in an elderly population is a challenge as cause of itching may be anything from pre-existing dermatoses to age related skin changes. Increasing use of medicines for various other ailments also cause diagnostic confusions.

Pathophysiology of itching in elderly ⁽¹⁾

Various studies have shown different pathophysiological factors for itching in elderly

- A study by Hara et al in elderly patients with xerosis showed decreased amount of skin surface lipids including sebum derived triglycerides; thereby impairing the water holding capacity of stratum corneum. Stratum corneum gets thickened and keratohyaline granules decrease in size and number.⁽²⁾
- Study by Long et al. Showed dry skin in proportion to degree of itch, increased intracorneal cohesion.⁽³⁾
- Guillet et al found increased histamine release and skin hypersensitivity to histamine in senile pruritus.⁽⁴⁾
- Increase reactivity to environmental allergens is found in elderly with nummular eczema by Aoyama et al.⁽⁵⁾

Willan's Itch ⁽⁶⁾

Itching in elderly patient in whom primary skin disease, xerosis, drug reaction and underlying systemic disease is ruled out – is known as Willan's itch. This type of idiopathic pruritus in elderly was described by Robert Willan.

Pathogenesis for Willan's itch is poorly understood. Skin biopsy does not show consistent findings.

Neural mechanism for itch has been suggested by Kaposi postulating itch a result of peripheral nerve atrophy. Recently subclinical neuropathy due to age related changes in nerves which lead to increased touch and pain threshold. Just like phantom itch, 'pseudophantom itch' due to subclinical neuropathy as been proposed as a mechanism for senile pruritus.

Causes of pruritus in elderly

Pruritus in elderly can be divided in – generalised itching or localised itching with or without some kind of rash. Etiological causes for itching can be derived from it.

LOCALISED ITCH WITH RASH	LOCALISED ITCH WITHOUT RASH	GENERALISED ITCH WITH RASH	GENERALISED ITCH WITHOUT RASH
Seborrheic dermatitis	Nostalgia paresthetica	Psoriasis ([Occasional pruritus)	Xerosis
psoriasis	Maralgia pareshetica	Seborrheic dermatitis	Thyroid disoreder
Nummular eczema	Brachioradial pruritus	Asteotatic dermatitis	Iron deficiency
Dermatophytosis	Post stroke pruritus	Allergic contact DS	Renal failure
Stasis dermatitis	Phantom itch	Atopic DS	Cholestasis
Pompholyx	Brain tumor/ abcess	Mycosis fungoides	Lymphoma
Irritant contact DS	Multiple sclerosis	Mastocytosis	Leukemia
Allergic contact DS	Transverse myelitis	Urticaria	paraproteinemia
Asteatotic eczema	Diabetes mallitus	Polymorphous light eruption	Polycythemia rubra vera
		Bullous pemphigoid	Carcinoid tumor
		Dermatitis herpetiformis	Other tumors/ malignancy
		Drug rash	HIV
		Id eruption	Hepatitis C
		Scabies and related diseases	Dermatomyositis
			Drug hypersensitivity
			Anorexia nervosa
			neurodermatitis
			Psychocutaneous disorders

Evaluation of patient with itching should be methodical to arrive at some etiological conclusion. Presence of rash gives some clue but in the absence of rash history and physical examination; supported by laboratory investigations as and when needed are absolutely essential.

Points to be covered in history in nutshell are

1. onset of itch
2. severity of itch
3. timing of itch – day or night
4. relieving or exacerbating factors
5. association with bathing
6. bathing/ skin care history
7. previous treatments for skin condition
8. past medical history
9. medication history
10. social history
11. family history
12. history of pets in family
13. personal habits/ hobby of patient
14. symptoms of systemic diseases like thyroid, diabetes mallitus

History of other medicines is very important in aged people as vast number of drugs are likely to cause pruritus and drug interaction when multiple drugs are prescribed is also important.

Commonly prescribed Drugs likely to cause itching

- Aspirin and other NSAIDs
- Penicillin group
- Antibiotics
- Antimalarials
- antihypertensives like ACE inhibitor, beta blocker, calcium channel blockers etc
- thiazide diuretics
- AKT
- Statins
- Antivirals
- Interferon
- anabolic steroid
- opioid antagonists
- lithium
- anticonvulsants.

Physical examination should include apart from examination of skin, examination of GIT, lymphatics, thyroid and other organ systems. Presence of primary lesion do help but most of the time excoriations, secondary changes etc are prominent which should be co-related with cause of itching. For example, longer excoriations suggest pediculosis while shorter excoriations suggest scabies; lichenification suggest long standing itch. Examination of hair, nail and genitals is equally important. Underlying systemic illness may present as eczematous skin changes, non-specific pruritus etc.

Screening tests may include

- CBC
- Thyroid function tests
- Renal function tests
- Liver function tests
- Blood sugar level
- X-ray chest
- Age-related cancer screening

Treatment⁽¹⁾

Treatment of Willan's itch is often unsuccessful. For other types of pruritus when cause is evident, treatment is effective. In most elderly patients, findings of xerosis may be subtle but treatment for dry skin is recommended.

Treatment of xerosis

- Frequent applications of bland emollient especially immediate after bath.
- Avoid irritant soaps, hot bath, scented bath oils, excessive use of soaps
- Mild topical steroids may be used initially for inflamed skin

Symptomatic treatment of itch

Topical

- Manthol
- Steroids
- Tacrolimus (esp. In atopics)
- Doxepin (for short duration and in localised area)
- Capsacin

Systemic

- Doxepin
- Amitryptiline] found to be effective in post-stroke pruritus
- SSRIs] in chronic itch these agents may be tried.
- Gabapentin] has been shown to be effective in brachio-radial pruritus.
- Cyclosporine
- Opioid antagonists

Other non-pharmacological treatment options

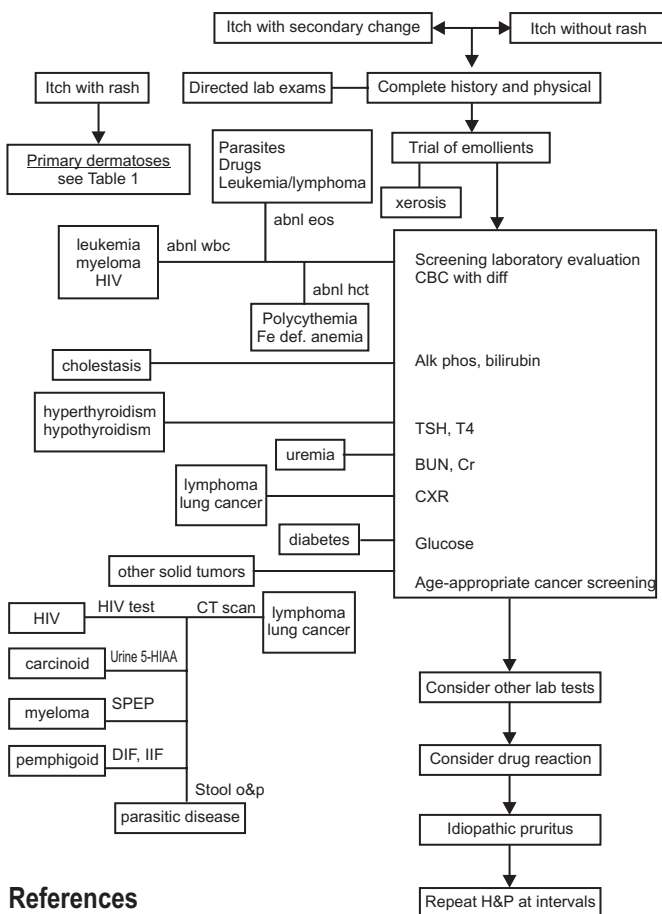
- UVL therapy
- Bright light therapy
- Cutaneous field stimulation
- Hypnosis

Conclusion

Itching due to multifactorial causes or idiopathic is common in elderly but why elderly are more prone to the development of itch is difficult to explain. The changes in stratum corneum leading to xerosis play a major role. Delayed barrier repair and hypersensitivity to histamine as well as environmental allergens lead to subclinical eczematous response. Recently described neurophysiology of itch – due to both decreased and increased innervations requires further study. Cause of pruritus when evident like scabies, atopic eczema may be easy to treat but most of the times symptomatic treatment has to be given for itching. Periodic re-evaluation may help to identify cause in such cases.

Evaluation of the elderly patient with generalized pruritus

(Figure 1)



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REVIEW ARTICLE

HIV/AIDS:

current scenario and future challenges

Dr. Santosh Rathod, Dr. Amit Mistry,
Dr. Aniruddha Vyas, Dr. D.G. Saple

Abstract:

Background: 30 years into the epidemic of HIV/AIDS, we have a renewed sense of scientific possibility and the knowledge to make a dramatic shift in the course of the epidemic.

Objective: The following review tries to identify our efforts to foster the search for a cure and build broader support for treatment as prevention.

Material and methods: The review studies the current epidemiological trends, distribution of various HIV subtypes, changing concepts in the associated co-infections and co morbidities, challenges in achieving cure from the infection and proposed theories and clinical research to overcome them.

Introduction:

- **Global distribution** of HIV-1 subtypes and recombinants in 2002-03 and 2004-2007:
 - ▶ Global and regional distribution of individual subtype and recombinants are broadly stable
 - ▶ Subtype-C still accounts for nearly half (48%) of all global infections. CRF2-AG doubled in 5 years.

• HIV-2 in the world:

- ▶ Prevalent in West Africa- can also be found in other African regions.
- ▶ Also in Spain, Portugal, France and India in – Micro-epidemics
- ▶ HIV-2 is naturally resistant to Non-nucleoside reverse transcriptase inhibitors (NNRTIs)
- ▶ HIV-2 accumulate more frequently Q151M mutations when compared to HIV-1 individuals treated with Zidovudine(AZT) or Stavudine(d4T)

Basic facts in pathogenesis:

- ▶ Sexual transmission of HIV is typically result of a single infectious event
- ▶ As HIV diverges from the founder to the chronically replicating virus it accumulates N linked glycosylation sites.

- **30 years of HIV/AIDS:**
1987- 1st FDA-approved anti-retroviral
Zidovudine – HIV with reduced sensitivity to
Zidovudine isolated during prolonged therapy
1987- AZT monotherapy
1994- two drug therapy
1996- three drug therapy
- **Life expectancy** of recently diagnosed
asymptomatic HIV-infected patients approaches
that of uninfected individuals¹.
 - ▶ Life-expectancy for HIV infected patients without
AIDS aged 25 years at 6 months post infection:
 - ▶ Men: an additional 52.7 years (vs. 53.1 year in
general population)
 - ▶ Women: an additional 57.8 years (vs. 58.1 years
in general population)
- **Major challenges** globally in the treatment of
HIV disease
 - ▶ <40% people in need of ARV therapy in low-and
middle income countries are receiving it
 - ▶ For every person put on ARV therapy in 2010, 2-3
other were newly infected with HIV
 Preventive tools: 1985 to 2011
 - ▶ Early 90s: Education
Behavior modification
Condoms
Clean syringes
Drug/alcohol treatment
 - ▶ 1994: AZT reduces mother to child
transmission of HIV
 - ▶ 2005-2007: Medically supervised adult male
circumcision
 - ▶ 2010: Effectiveness and safety of tenofovir
gel; an anti-retroviral microbicide for the
prevention of HIV infection in women. 1%
tenofovir gel reduced HIV incidence by 39%
overall, and by 54% with high gel
adherence(>80%)
 - ▶ 2011: Pre-exposure prophylaxis(PrEP) with
oral ART reduces risk of HIV acquisition
among heterosexual men and women
 - ▶ 2005-2011: The arrival of treatment as
prevention
-Decrease in community viral load is
accompanied by reductions in new HIV
infections in San Francisco.
 - ▶ 2011: ART reduces HIV transmission in HIV
sero-discordant heterosexual couples
(HPTN 052)
- **Scientific advances** over the years(1981- 2011)
 - ▶ 2009: RV144 trial in Thailand: 1st signal of
efficacy with an HIV vaccine

- ▶ Critical challenge in development of an HIV
vaccine is to convert neutralizing epitope into
potential immunogen.

A cure for HIV infection:

Definition of cure: “permanent remission in the
absence of requirement for therapy”

- ▶ Sterilizing cure: 'eradication of virus'
- ▶ Functional cure: 'permanent suppression of
viral replication without eradication'

HIV/SIV pathogenesis: lessons from distinct
outcome in human and non-human primates.

- ▶ Early determinants of HIV pathogenesis are
inflammation and chronic immune activation
that leads to viral reservoir and HIV
persistence, residual ongoing replication,
survival/proliferation of Tgm

Challenges beyond HIV in people living with HIV/AIDS(PLHIV)

- ▶ Cancer, lymphomas
- ▶ Aging diseases
- ▶ Cardiovascular diseases
- ▶ Immune defects, inflammatory and
autoimmune malignancies
- ▶ Chronic HIV infection on HAART

HIV and HSV-2

- ▶ Almost 30 years of epidemiologic data
supported a synergistic relationship between
HSV-2 and HIV
- ▶ HSV reactivation increases HIV susceptibility
and infectiousness as well as potentially
accelerates HIV disease progression
- ▶ Acyclovir 400mg twice daily given for 24 week
delayed disease progression among
HIV/HSV-2 co-infected individuals.
- ▶ HIV increases HSV outbreaks facilitating HSV
transmission.

HIV and Tuberculosis²

- ▶ PLHIV has an estimated 20-30 times greater
risk of developing active TB than people
without HIV infection
- ▶ HIV associated TB:
Estimated number of cases – 1.1 million
Estimated number of deaths – 3,80,000
- ▶ TB is responsible for one in four AIDS death
- ▶ 2011 WHO recommendations on
collaborative TB/HIV activities- 'The 3 Is'
 - i. Infection control
 - ii. Isoniazid preventive therapy (IPT)
 - iii. Intensified case finding
- ▶ ART in HIV/TB patients:
 - Mortality was reduced by 34% when

HAART was initiated 2 weeks vs. 8 weeks after onset of TB

- WHO recommendation:
- Start TB treatment first, followed by ART as soon as possible after starting TB treatment irrespective of CD4 cell count
- Treatment strategy for TB/HIV co-infected patients: 1st choice- standard TB regimen + 2NRTIs + Efavirenz
- Rifabutin can be used with lopinavir, atazanavir, fosamprenavir, darunavir, tipranavir along with ritonavir therapy
- Rifabutin is acceptable substitute for rifampicin in standard TB regimens but is not widely available and require loose drugs
- ▶ Early diagnosis crucial for MDR-TB among PLHIV:
Diagnostic capacity for MDR-TB inadequate in most countries (7% world-wide)
- ▶ Key messages:

HAART expansion key to reduce the burden of HIV associated TB

Improve communications between TB and HIV service to operationalize dual treatment
Actively promote rifabutin in resource limited setting after defining appropriate doses

- **HIV and malaria³:**

- ▶ Plausibility of interaction: malaria affecting HIV causes antigen stimulation leading to immune activation and increased viral replication with faster disease progression and further antigen stimulation.
- ▶ Influence of malaria on HIV disease: increase in viral load during malaria might be sustained for long enough to increase the risk of HIV transmission and accelerated disease progression
- ▶ HIV and malaria in pregnancy: HIV infected pregnant women are at increased risk of:
 1. Symptomatic malaria and placental malaria
 2. Severe malaria
 3. Maternal anemia and adverse birth outcome
- ▶ “Combination prevention” for malaria in HIV infected populations
 1. co-trimoxazole prophylaxis (CTx)
 2. Insecticide treated bed nets (ITNs)
 3. ART
- ▶ Clinical implications:

1. Malaria is rarely the cause of fever in individuals receiving CTx
2. Malaria accounted for only 4% of febrile episodes in an HIV-infected cohort compared to 33% in the HIV uninfected cohort.
3. Presumptive therapy for malaria in these groups should be avoided and careful evaluation for other causes of fever should be done
4. Malaria prevention in pregnancy is critical to minimize adverse pregnancy outcomes

- **HIV and HCV:**

- ▶ Epidemiology of HCV
 - 2-3% of world population
 - >40% undiagnosed
 - 30% chronic carriers will develop cirrhosis
 - HCV is the primary reason for liver transplantation
 - HCV is the major cause of liver cancer
 - No vaccine
 - **Only curable chronic viral infection**
- Predictors of response to HCV therapy
 - HCV genotype
 - Baseline serum with HCV-RNA
 - Liver fibrosis stage
 - RVR
 - EVR
- ▶ Challenges using DAA in HIV- HCV co-infection
 - More elevated HIV load more virological failures?
 - Faster selection of drug resistance
 - Drug-drug interaction
 - Overlapping toxicities- rash and anemia
 - Drug compliance with polymedication
 - Additional cost

- **Anti-retroviral therapy:** development of new agents

- ▶ **Lersivirine(LRV):** Next generation NNRTI with unique binding and potent activity against HIV-1
- ▶ Dolutegravir(DTG): rapid – robust and sustained antiviral response with once daily (QD) DTG, a next generation Integrase Inhibitor (INI) in combination therapy in anti-retroviral naïve adults.
No IN resistance mutations detected through therapy 48 weeks Well tolerated with fewer discontinuations than EFV and less impact on lipid parameters
- ▶ **Modern dual therapy:** 48 week results of

pilot study of Maraviroc(MVC) + ATV/r support the anti-viral activity of this one daily, two drug combination in treatment-naïve patients. Neither resistance nor change in phenotypic tropism was observed. No new unexpressed safety events.

- ▶ Efficacy of Maraviroc(MVC) administered once daily or twice daily with boosted protease inhibitors to treatment experienced patients.
- **HAART is not perfect:**
 - ▶ Long term toxicity
 - ▶ CD4/VL monitoring is required
 - ▶ Resistance
 - ▶ Cost
- **New objectives for the HIV therapy**
 - ▶ It should increase survival, stop progression and stop transmission
 - ▶ Decrease drug burden (number of ARV) to decrease cost and toxicity
 - ▶ One should be able to stop ART (at least transiently)
 - ▶ Should be able to achieve functional cure/eradication
- **Barriers to Cure:**
 - ▶ Latently infected T-cells
 - ▶ Residual viral replication/production
 - ▶ Anatomical reservoir

And they are not mutually exclusive
- **Interventions for HIV cure:**
 - ▶ Reduce Residual viral replication/production by optimizing HAART⁴
 1. Intensification
 2. Drug penetration
 - ▶ Reduce immune activation⁵
 1. Anti-inflammatory drugs
 2. Reduce gut damage and/or microbial translocation
 3. Anti CMV drugs
 - ▶ Target latent reservoirs
 1. IL-7 (eramune) or anti IL-7
 2. Histone deacetylase inhibitors (HDAC)
 3. Anti PD-1
 4. Gene therapy
 - ▶ Immune control of reservoirs
 - IL-7
 - HIV specific IgG2 is associated with control in long-term non-progressors (LTNP)^{7,8}
 - NK cells
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dermocare

TOPIKA

HERBAL HAIR OIL

Made Siddhi with:

Azadirachta Indica (Neem leaves) 2.5%
Eclipta alba (Bhringraj) 1%
Emblica officinalis (Amla) 1%
Indigofera tinctoria (Nilpuspha) 1%
Centella asiatica (Brahmi) 1%
Cyperus rotundus (Nagarmotha) 1%
Jasminum officinale (Jatichetika) 1%
Abrus precatorius (Gunja) 1%
Glycyrrhiza glabra (Yashtimadhu) 0.5%
Crateeva murvala (Varunbark) 0.5%
Pongamia pinnata (Karanj Beej) 0.5%
Solanum indicum (Bruhati Laxmana) 0.5%
Citrullus colocynthis (Indravaruni) 0.5%

INDICATIONS :

- ✓ Stimulates hair growth,
- ✓ Arrests falling of hair,
- ✓ Checks alopecia & dandruff,
- ✓ Helps regrowth of hair.

COMPOSITION :

- ✓ Sesamum indicum (Til) Oil 80%
- ✓ Cocos nucifera (Tranaraj) Oil 20%

APPLICATION :

Gentle Massage on Dry Hair.
Store in a cool dry place.

Pack : 100 ml.

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Nr. Mithakhali Under Bridge, Navrangpura
Ahmedabad 380009. (Gujarat) INDIA
Phone: +91 79 26461342, 65446888 Fax: +91 79 26430086
E-mail: dermocare2000@yahoo.com
Web: www.dermocareltd.com

PHOTO QUIZ – 1

Dr. Krina Patel, GMERS Medical College, SOLA

A Curious case of intractable itching

A 62-year old male patient presented for intractable itching all over body for 2 years. On examination; he had multiple, discrete, excoriated and crusted papules all over body. (figure1) Lesions started on legs and involved whole body in 2 years. Patient's general examination was normal and was not on any other medicine for any other systemic illness. None of the treatment taken for his skin condition gave him complete relief any time.

Biopsy from the crusted papule on his back is shown in figure 2.

What is your diagnosis?



Figure 1

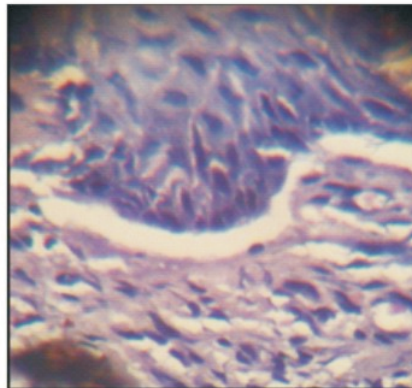


Figure 2

On suspicion, Patient was asked to stop all medicines including topical preparation for 2 weeks. Rebiopsy from lesion is shown in figure 3 and Direct immunofluorescence pattern is shown in figure 4

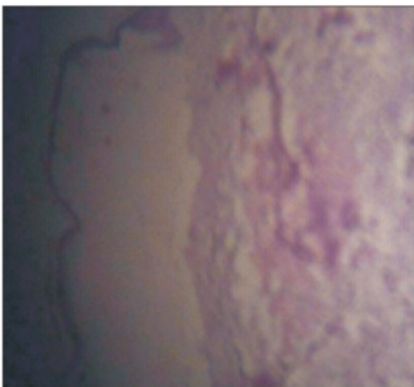


Figure 3

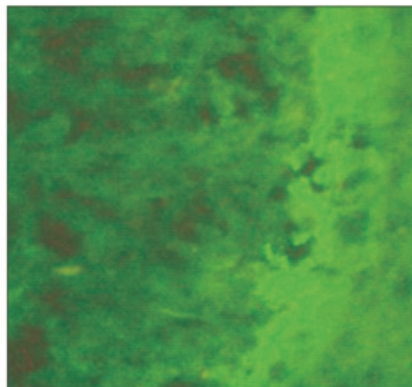


Figure 4

What is your diagnosis now?

Send your answers on - quarterderm@mail.com

PHOTO QUIZ – 2

A 22-year old male patient presented for sudden development of nodular lesions on his scalp noted for 1 month. Lesions gradually increased in size and number to involve entire scalp. Patient had complain of headache on and off. On examination, multiple, small to large, erythematous, shiny nodular lesions, asymptomatic in nature were present on scalp. (Figure 1) No lesions noticed anywhere else on body.

Skin biopsy was taken. Histology is shown in figure 2. Within 2 months of presentation for skin lesions, patient developed convulsions. CT scan showed multiple space occupying lesions. (Figure 3)

What is your diagnosis? [CLUE – histology was characteristic of this atypical presentation of a rare disease generally seen in children]



Figure 1

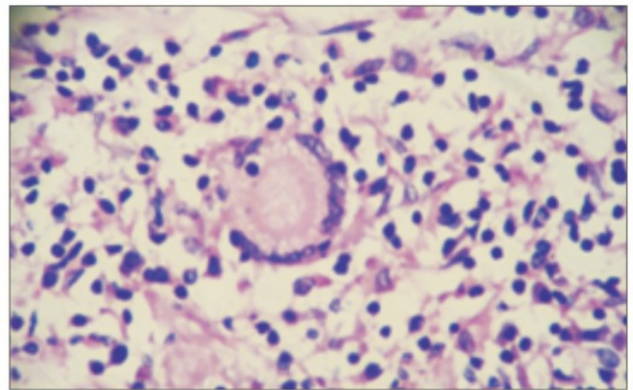


Figure 2

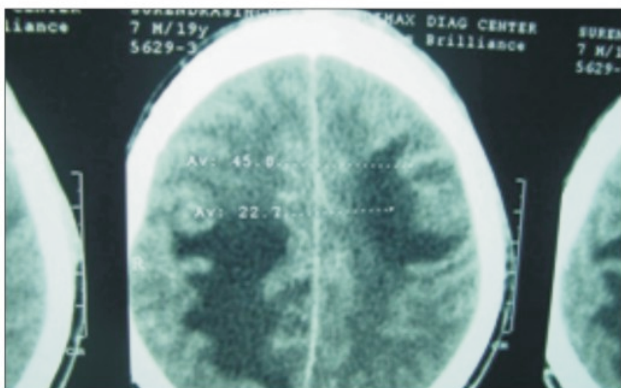


Figure 3

Send your answers on - quarterderm@mail.com

topilen_{cream}

lecithin + dimethicone cream



*Terms and conditions apply

dermocare

for dry skin treatment

Topilen cream contains primarily lecithin, a phospholipid water-binding agent found naturally in the skin. Each phospholipid molecule of water, restoring the natural moisture balance.

Topilen cream is non-greasy and absorbs quickly into skin. It contains **no parabens, lanolin or mineral oil** which can irritate sensitive skin.

Indication

It is indicated for the relief of xerosis, dry eczema, ichthyosis, pruritus, for alternate use with topical steroid, senile dry skin, neurodermatitis and fissures.

Safety

It is Hypoallergenic, free of fragrance shows no acne formation

Composition

Each gm Contains

Lecithin U.S.P

Propylene Glycol I.P.

Dimethicone I.P.

Diazolidinyl Urea (Preservative)

In a cream base

1mg.

15 mg.

5mg.

1.5 mg.

Q.S.

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Regd. Office: 210, Aakash Avenue, B/h. Ashwamegh
Nr. Mithakhali Under Bridge, Navrangpura
Ahmedabad 380009. (Gujarat) INDIA
Phone: +91 79 26461342, 65446888 Fax: +91 79 26430086
E-mail: dermocare2000@yahoo.com
Web: www.dermocareltd.com

CASE REPORT

Monilethrix: A Study of 2 Cases With Pedigree

Dr. Palak Mehta,^{3rd} Year Resident,
Dr. Ranjan Rawal, Professor & HOD
V.S.Hospital, Ahmedabad.

INTRODUCTION:

Monilethrix(monile=necklace & thrix=hair) is an autosomal dominant trait having mutation in gene coding for human hair keratin hHb1 & hHb6 on chromosome 12p13. There is defective cortical cell keratinization involving type 2 cortex keratin causing hair constrictions and breakage⁽¹⁾.

CASE REPORT:

CASE 1: A 5-year old male child born of non-consanguineous marriage presented with partial loss of hair on the whole scalp, eyebrows and eyelashes since childhood. Presence of similar complaints was there in his 35 years old father and 2 years old female sibling. (Figure 1)

CASE 2: A 7-year old female child born of non-consanguineous marriage presented with diffuse loss of hair and brittleness on whole scalp since childhood. Presence of similar complaint was there in her 3 year old female sibling. Her male sibling of 5 years of age had no such complaint. (Figure 2)

There were no associated history of involvement of any other body area, teeth and nail involvement, retarded growth, associated autoimmune conditions or any other congenital disorder in both the cases.

On examination of both the cases, there was generalized hair loss seen on the whole scalp with the affected hair beaded and brittle. 1-2 cm long broken hair with alternate zones of spindle like thickening and thinning was seen placed 0.7-1 mm apart.

In case 1, there was presence of follicular keratotic papules on the occiput and nape of neck. Also white scales were seen on the eyelashes of this patient. (Figure 3)

All routine investigations were normal in both the cases.

Trichoscopic examination showed characteristic beaded appearance of the hair with nodes and internodes is seen placed at 0.7-1 mm distance. Broken hair stubs are seen due to break at internodes. (Figure 3)

Trichogram showed hair with regularly spaced spindle shaped nodes separated by intermittent abnormal constrictions. (Figure 4)

In both the cases avoidance of chemical and mechanical trauma, sunlight, dyes, bleaches, permanent straightening and curling devices was advised. Advice for use of mild shampoo was given. Patients were counseled and given reassurance for the course and prognosis of the disease. Use of hair wig was advised for cosmetically improved appearance.

DISCUSSION:

Monilethrix is a rare genetic disorder having autosomal dominant transmission. It can affect several generations of the same family. Autosomal recessive inheritance is also reported. Autosomal dominant monilethrix is caused by mutations in hair keratin genes *KRT81*, *KRT83*, or *KRT86*, whereas the autosomal recessive form results from mutations in the desmoglein 4 gene (*DSG4*)⁽³⁾. There is no racial or sexual predilection seen in this disorder.

Lanugo hair usually appears normal at birth. Several months later, it is replaced by moniliform hair, which is dry, brittle, fragile, and lusterless. The scalp hair breaks spontaneously at 0.5-2.5 cm length. Broken short hair looks as if it has been burnt. In more severe cases, hair over the whole scalp can be affected, as well as pubic hair, axillary hair, eyebrows, eyelashes, or hair on the arms and legs⁽⁵⁾. The patient may rarely have associated trichorrhexis nodosa, nail and teeth defects, retarded growth and juvenile cataracts⁽¹⁾. True monilethrix must be differentiated from pseudomonilethrix, an artifact produced by tweezers or compressing hairs between two glass slides⁽²⁾.

Light microscopy examination shows the nodes having the diameter of normal hair and medullated,

whereas the internodes having no medulla and are the sites of fracture. Pigment is present in both segments.

Trichoscopic examination shows thin hair with regularly spaced elliptical, fusiform or spindle-shaped nodes separated by intermittent abnormal constrictions which are the sites of fracture.

In our cases, the patient were normal at birth and the defect stated appearing after 2- years of age. The hair started becoming brittle with beading. This was followed by hair fall due to breaks at internodes. There was presence of follicular keratotic papules on the occiput and nape of neck in case 1. There was no associated growth retardation as well as, no involvement of teeth, nails and eyes in either case.

This condition improves over years mainly after puberty and also during pregnancy. Oral etretinate has been used with limited success. Griseofulvin can temporarily restore normal hair growth⁽²⁾. Topical minoxidil could be tested in postpuberal patients⁽⁴⁾. Iron and vitamin supplementation should be given. Hyperkeratosis is treated with topical retinoic and/or glycolic acids.

No consensus about genetic and antenatal diagnosis in monilethrix has been reached yet. Perhaps, for some of the more severe phenotypes, prenatal diagnosis may be requested and this can now be performed from chorionic villus samples at an early stage of the pregnancy.



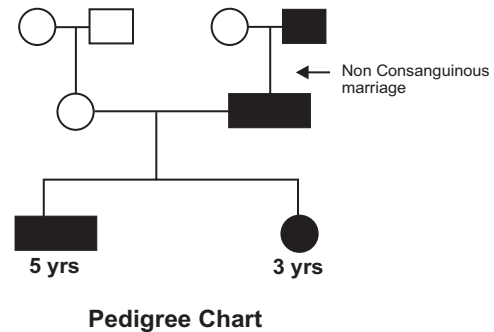
(Figure 1)



(Figure 2)



(Figure 3)



(Figure 4)

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CASE REPORT

Varied presentations of Syphilis

Dr. Patel Shruti R. (Junior resident, D.V.D.)
Dr. Patel Neela (Assistant Professor, M.D. D.V.D.)
Dr. Parikh Anugrah (Associate Professor, M.D. D.V.D.)
Department of dermatology, venereology and leprosy
L.G. Hospital, MANINAGAR Medical College, Ahmedabad.

Abstract: Syphilis is a sexually transmitted disease caused by the spirochete *Treponema pallidum*. In the 2000s rates of infection have increased in many countries because of unsafe sexual practices. Diagnosis of syphilis is challenging because its variable clinical presentations. Syphilis co-infection with HIV causes altered and florid skin lesions. We are reporting 4 cases of syphilis, all have different presentations. Clinicians should familiarize themselves with the many and varied presentations of syphilis.

Key words: syphilis, chancre redux, follman's balanitis

INTRODUCTION: Syphilis has a myriad of presentations however signs and symptoms can be vary depending on which of the stage it presents. It is known as "Great imitator" because it mimics various surgical and medical conditions.

CASE HISTORY:

Case 1: A 37year old male presented with asymptomatic lesions on the body for 1 month. Patient had history of unprotected sexual exposure with MSM 3 months back. He had no history of lesion on genitalia in past. On examination he had

- Multiple papular lesions with central necrosis present on all over the body
- Corymbose lesions present on neck and upper back
- Split papules present at angle of mouth and nasolabial folds
- Condylomalata lesions on perianal region
- Multiple genital ulcerations
- Generalized lymphadenopathy

Investigations showed S.VDRL in 1:64 positive, TPHA positive, and HIV positive.
Thus this was a case of secondary syphilis with florid lesions.

Case 2: A 30year old male presented with multiple, asymptomatic, elevated lesions over face, arms, trunk and legs for 1 month. Patient had history of penovaginal-unprotected exposure with Commercial Sex Worker (CSW) before 4 months. Patient also had ulcer on genitalia for 1 month. On examination multiple papulo-nodular lesions over face, arms, trunk and legs. Single, indurated, non-tender ulcer present on coronal sulcus. Patient also had history of ulcer at same site before 3 months that subsided by its own. Bilateral inguinal and epitrochlear lymph nodes were enlarged, multiple and non-tender.

Investigations showed S.VDRL 1:64 positive, TPHA positive and HIV negative.

Thus this was a case of secondary syphilis with chancre redux

Case 3: A 21year old male presented with genital lesion and painful swelling on groin for 1 month. Patient has taken treatment with Roxithromycin 150mg twice a day for 21 days without improvement. Patient had history of multiple unprotected, penovaginal exposure with known lady. Patient denies history of lesion on genitalia in past.

On examination three erythematous elevated patches on glans; tiny papules on shaft of penis, scrotum and few on chest were present. Busckel-olendroff sign was positive. Big painful, firm lymph node 4x3cm size was palpable in right inguinal region. Epitrochlear lymph nodes were also palpable on both sides and non tender.

S. VDRL was positive in 1:2 dilutions. But with strong suspicion of syphilis on clinical manifestations we did TPHA, which came positive. We treated with benzathine penicillin 2.4 million units intramuscularly ½ in each buttock after negative test dose. Patient responded to our treatment within 3 days.

Case 4: A 35year old male presented with fever with rigors, painful lesions and swelling of genitals for 15 days. He had multiple unprotected sexual contacts with 4 CSW in last 1 month. On examination multiple, superficial ulcers on glans with inflammation around urinary meatus and paraphimosis. Bilateral inguinal lymph nodes were enlarged and non-tender. case diagnosed as balanitis on the basis of clinical presentation. On routine STD investigations S.VDRL was positive in 1:64 dilutions. Therefore for the confirmation TPHA was done, which also came positive. After investigations we revised our diagnosis as syphilis with follman's balanitis. Prepuce biopsy report

shows multilocular pustules full of polymorphs. Patient is treated with standard syphilis treatment and he showed 50% improvement in 48 hours. Thus this was a case of syphilis with Follman's balanitis.

DISCUSSION:

Syphilis has a myriad of presentations and can mimic many other diseases. Hence it is known as "the great imitator". The signs and symptoms of syphilis vary depending on which of the stage it presents in.

Our first case has classical presentation of secondary syphilis with types of lesions. Skin lesions like papular lesions, corymbose lesions on neck and upper back; mucous lesions like split papules, condyloma lesions around perianal lesions, genital ulcerations. Patient was HIV positive. So, in HIV cases detailed history and careful examination is required to note all type of lesions.^[1]

Our second case has genital ulcer, which was single, non-tender, indurated ulcer on coronal sulcus. Patient also had papulo-nodular lesions on other parts of the body. So, here patient was showing characteristic of primary chancre with secondary syphilis skin lesions. If primary lesion develops at the same site in secondary stage, it is known as chancre redux. Our patient gave history of

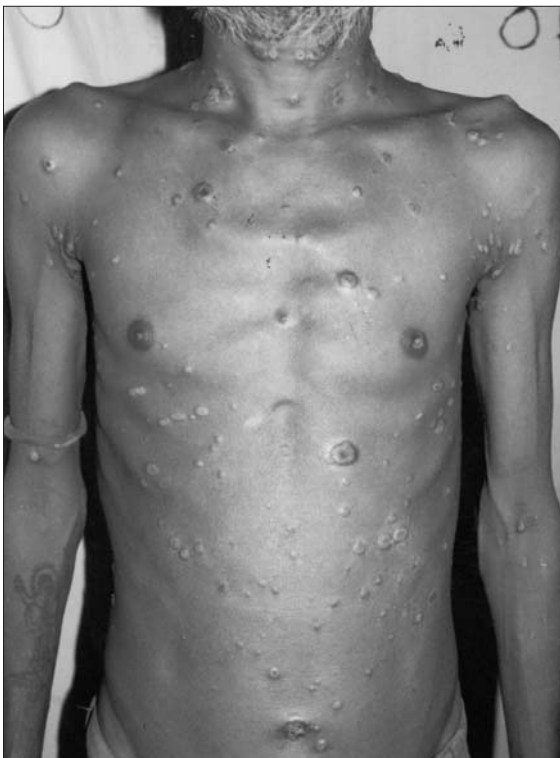
lesion at same site 1 month ago. So, on the basis of clinical manifestations and investigations we diagnose it as a case of chancre redux. Relapse of primary chancre (chancre redux) is rare and occurs due to inadequate or no treatment.^[2]

For the diagnosis of syphilis S.VDRL should be positive in 1:16 dilutions. And it should be confirmed with more specific treponemal tests. In our case S. VDRL was 1:2 dilutions but TPHA came positive. This can be explained with prozone phenomenon. Prozone phenomenon is a false negative response of antigen antibody reaction, which occurs due to high antibody titre. Prozone phenomenon occurs more commonly with secondary syphilis, HIV co-infection, pregnancy etc. ^[3] Every clinician who is suspecting syphilis should be aware of prozone phenomenon.

Follman's balanitis is erosive chancre occurring on the prepuce. It can be confused with balanoposthitis. Follman's balanitis can be a sole presentation of primary syphilis. ^[4,5]

Conclusion:

Because the manifestations of syphilis are non-specific and may masquerade as many other diseases, the physician must keep a high index of suspicion regarding the possible diagnosis of syphilis.





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Ultrasound in Dermatology

Dr. Nishith Bodiwala (3rd Year Resident),

Dr. Ranjan Rawal (Professor & HOD)

Smt. NHL Municipal Medical College,

Ahmedabad

PRINCIPLES:-

Ultrasound imaging reflects the sound waves through tissue. It depends on the intrinsic variation in tissue structure, vascularity and density, which reflects differences in keratin, collagen and water content. Ultrasound measurement entails the transformation of sound waves into visual images. B-mode scanning is the method of choice in dermatology. There are mainly two types of ultrasound:

- Low frequency which is mainly used for deeper structures, such as internal organs.
- High frequency is mainly used for higher resolution of tissues closer to the transducer.

Dermatologic ultrasound is generally within the range 13.5-100 MHz and most high resolution ultrasound is performed at 20 MHz.

There are two subtypes: 1. Conventional ultrasound 2. Doppler ultrasound

Conventional ultrasound techniques image tissues with stationary interfaces. Conversely, Doppler ultrasound monitors tissues in motion and it has several subtypes, including pulsed wave Doppler, continuous wave Doppler, and duplex ultrasonography. Pulsed wave Doppler determines the depth of the moving object while continuous wave Doppler continuously monitors the presence of moving object without gathering information concerning the depth. Duplex ultrasonography creates a picture of flow. It is color coded to assess directionality of flow or amplitude coded to demonstrate the volume of flow.¹

Ultrasound elastography is a technique used to create an image of the strain on a tissue imposed by force. Elastography calls for comparison of echoes

pre-compression and post-compression of a given tissue area. Currently it is being considered as an adjunctive mode of non-invasive imaging in the evaluation of prostate, breast, thyroid, and liver masses, as well as general lymphadenopathy. Also it has considerable clinical potential in the assessment of lower extremity vascular disease, pressure ulcers, lymphedema, in the understanding of age related changes in the skin and benign and malignant neoplasms.²⁻⁵

DIAGNOSTIC USES:-

SKIN CANCER

Malignant melanoma, SCC and BCC seen as hyperechoic surroundings and hypoechoic tumors.^{6,7} 7.5-10 MHz may also be important in evaluation of lymphatic spread and lymph node involvement of skin cancer and it is more sensitive and specific in detecting lymphatic spread of melanoma than physical examination alone, detecting 25% to 30% more patients with regional lymph node metastases.^{8,9}

INFLAMMATORY DISEASES/INFECTIOUS DISEASES/BENIGN NEOPLASMS

Ultrasonic imaging with frequencies upto 50 MHz have also been used for the characterization of inflammatory diseases, including scleroderma, localized morphea, lichen sclerosus et atrophicus, spongiotic dermatitides and hidradenitis suppurativa.¹⁰

Ultrasound can be used to discriminate lichen sclerosus et atrophicus from morphea¹¹, also to calibrate response to treatment with the patterning of dermal collagen in localized scleroderma using high resolution ultrasound with histopathological correlation.¹² it can be used to determine the extent of subclinical cystic lesion in hidradenitis suppurativa.¹³

In infectious diseases, especially radiolucent and difficult to pick up with other means of imaging, ultrasound examination using a 7.5 MHz frequency in emergency department may be useful. Ultrasound has been used in detection of abscesses¹⁴, necrotizing gangrene of genitalia, necrotizing fasciitis.

Using a 7.5 MHz ultrasound; lymph nodes melanoma metastases lymphosarcomas, hemangiomas, lipomas, cysts and trichoepithelioma can be studied. MM metastases are nearly anechoic whereas benign subcutaneous tumors are echogenic. Lipomas are more echogenic than subcutaneous fat due to additional features (fibrolipoma, angiolipoma) and they are elongated isoechoic or echogenic masses within subcutaneous tissue. Trichoepitheliomas are homogeneous echogenic tumors.

THERAPEUTIC USES:-

-It can be used for therapeutic purpose using 25 KHz acoustic waves to rapidly and successfully achieve debridement of chronic wounds.¹⁵

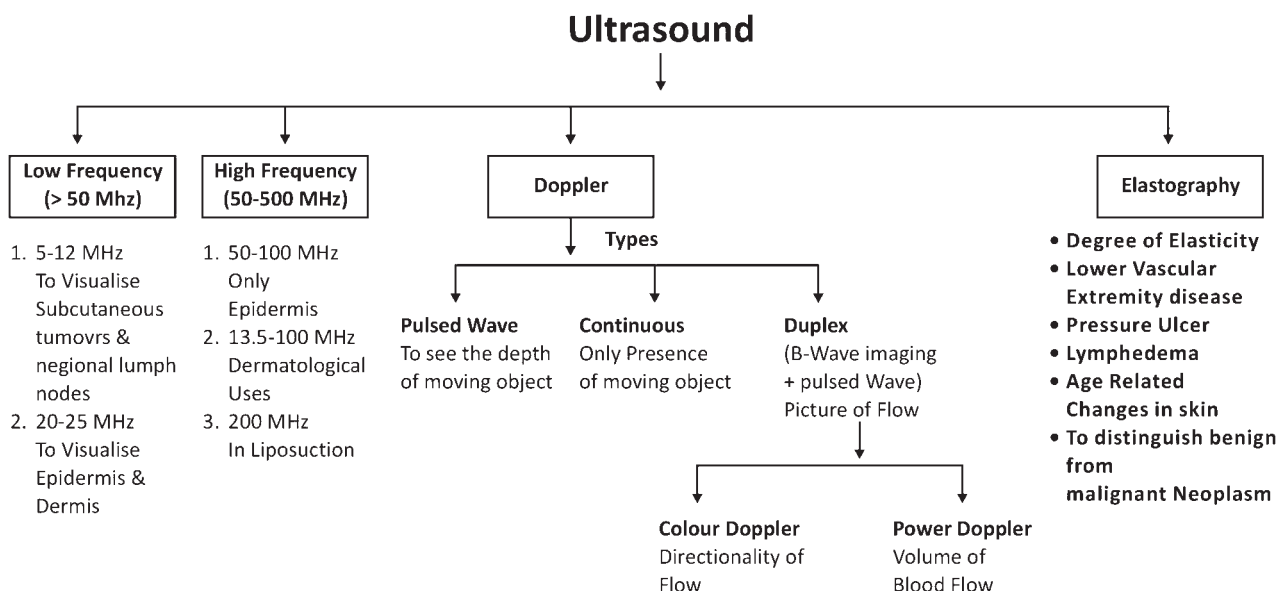
- Ultrasound is helpful in assessing condition of epidermal cyst wall aiding the surgeon in determining the best method of excision.

-Ultrasound assisted liposuction (UAL) uses a probe 20 to 27 kHz frequencies to selectively destroy adipose tissue. It allows surgeon an easier hand in approaching fibrous areas especially in

secondary liposuction and a more rapid evacuation of fat. UAL offers benefit of targeting adipocytes while sparing nerves and smaller blood vessels and superficial UAL was said to cause superior collagen contraction and skin tightening.¹⁶

- A recent ultrasonic device as a means of non-ablative rejuvenation of the face and neck has emerged. 4 and 7 MHz probes deliver energy to depths of 4.5 or 3 mm with pulse durations of 25 to 40 milliseconds creating zones of coagulation necrosis in the dermis and subcutis and promoting visible skin tightening. This prototype creates cone shaped zone of necrosis in the reticular dermis without affecting overlying epidermis obviating need for concurrent tissue cooling. A low echogenic band just below epidermis in sun damaged skin called a subepidermal low echogenicity band.

-Ultrasound devices have emerged in the field of body contouring as a means of ablating adipocytes. This technology allows for delivery of a small amount of ultrasonic energy in the epidermis and dermis and an additive amount in



subcutis, disrupting adipocytes and promoting fat resorption.

- Therapeutically ultrasound has been used as a method for improving penetration of cosmetic topical in the process of phonophoresis.¹⁷

CONCLUSION:

There are many applications of ultrasound in the field of dermatology; some are currently under investigation and it also requires significant operator skill and training.

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History of Dermatology

Snippet 1

Dr. Krina Bharat Patel

Associate Professor & H.O.D., GMERS Medical College, SOLA, Ahmedabad.

BARON JEAN-LOUIS ALIBERT (1768-1836) – Founder of dermatology in France (Figure 1)

Dermatology emerged as a distinct medical specialty in the mid-1800s. Pioneering physicians from England, France, the German-speaking countries, and the United States made contributions to the speciality dermatology through their work.

Post-French revolution, the national assembly of France signed a decree creating 'Ecoles de Sante' in 1794 which is considered as the '*moment of modern medicine*' in France. Alibert was one of the brightest students of this new 'Ecoles de Sante' and he emerged as one among generation of outstanding physicians along with Bichat, Laennec, Broussais etc.

Alibert is the first example in France of a physician –

- Looking at skin diseases
- Describing skin diseases
- Representing skin diseases in paintings and engravings of huge, expensive attages.

Born on May 2, 1768 in a small town of France, he studied philosophy, literature, Greek, Latin and theology in his school days. As a medical student in Paris, he wrote many medical as well as philosophical papers.

Alibert worked as a medical in-charge of hospital Saint Louis of Paris. He was one of the pioneer medical practitioner of skin diseases. His hospital had 600 beds continuously occupied by patients with skin diseases. Alibert was a famous teacher of his time. He wrote many books. Alibert was the one who was chiefly responsible for the establishment of St. Louis hospital as dermatological centre. He with Gilbert recognised the important role played by parasites in skin disease, they did revolutionary work in treatment of scabies and fungal infection.

Planck was a librarian dermatologist of his time, Wilan saw patients mainly in out-patient set up. But

Alibert was the one associated with hospital, drew atlases of day to day evolution of skin diseases by close observation. Alibert is credited with the first description of mycosis fungoides which he observed in his patient named Lucas (Figure 2), pityriasis amiantacea (Figure 3), keloids, cutaneous leishmaniasis etc. 'Dermatoses' and 'Syphilides' are his famous terminologies.

He was a personal physician to King Louis XVIII and after King Louis's death to King Charles X who rewarded him as 'Baron'.

What made him famous also made him fail. Spectacular representation of tree of dermatoses made him known world over but tree was ahead of its time; as it was impossible to classify all skin diseases on logical basis; which is impossible even today.

BUT ALIBERT IS STILL AHEAD OF OUR TIME. SALUTE TO A GREAT STARTER!

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Jean Alibert (Figure 1)



Patient named Lucas in Alibert's drawing depicting Mycosis fungoides (Figure 2)



Pityriasis amiantacea in Alibert's drawing of his patient (Figure 3)

Resident Roundup

Dr. Santosh Rathod

Assistant Professor, AMC-MET MEDICAL COLLEGE, Ahmedabad.

Bones, Eyes, and Nails

This is the first installment in a series designed to bring to light “askable factoids” for the dermatology examinations. This installment focuses on important (or frequently asked) findings in bone, eyes, and nails. The list is by no means exhaustive, but ... enjoy!

CONDITION	BONE	EYES	NAILS
5-FU & AZT			Blue Lunula
Acne Fulminans	Osteolytic Lesions		
Alopecia Areata			Nail Pits Red and Spotted Lunula
Apert's Syndrome	Synostosis		One Large Fingernail
Argyria		Blue Sclera	Slate Blue Lunula
Arsenic			Mee's Lines
Ataxia-Telangiectasia (Louis-Bar's)		Bulbar Telangiectasia	
Behçet's Syndrome		Retinal Vasculitis, Uveitis, & Hypopyon	
Bushke-Ollendorf Syndrome	Osteopoikolosis		
CHIME Syndrome		Colobomas of Retina	
Cicatricial Pemphigoid		Symblepharon	
Cirrhosis			Terry's Nails
Cockayne's Syndrome	Dwarfism	Salt & Pepper Retinitis Pigmentosa with Optic Atrophy	

Conradi-Hünemann Syndrome	Unilateral Limb Shortening, Chondrodysplasia Punctata	Asymmetric Focal Cataracts	
Darier-White Disease			Red and White Bands, V-nicking
Ehlers-Danlos IX	Occipital Horns		
Ehlers-Danlos VI		Keratoconus	
Fabry's Disease		Whorl-like Corneal Opacities, Spoke-like Cataracts	
Fanconi's Syndrome	Absent Radius or Thumb Strabismus	Retinal Hemorrhages	
Gardner's Syndrome	Craniofacial Osteomatosis	Congenital Hypertrophy of Retinal Pigmented Epithelium	
Goltz's Syndrome	Osteopathia Striata, lobster claw deformity	Colobomas	
Homocystinuria	Marfanoid Habitus, Genu Valgum	Downward Lens Displacement	
Hypoalbuminemia			Muehrcke's Nails
Incontinentia Pigmenti		Strabismus, Coloboma, Cataracts, Optic Atrophy	
JXG		Hyphema, Hypopyon	
KID		Keratitis	
Lamellar Ichthyosis		Ectropion	
LEOPARD		Hypertelorism	
Linear Morphea	Melorheostosis		

Lipoid Proteinosis (Urbach-Wiethe)		Sickle-Shaped Beanbag Eyelid "String of Pearls"	
Maffucci's Syndrome	Enchondromas, Chondrosarcoma		
Marfan's Syndrome	Marfanoid Habitus	Upward Lens Displacement	
McCune-Albright Syndrome	Polyostotic Fibrous Dysplasia		
Nail-Patella Syndrome	Posterior Iliac Horn, Absent Patella	Lester Iris	Triangular Lunula, Micronychia, Anonychia
NF-2		Posterior Subcapsular Lenticular Cataracts	
Osteogenesis Imperfecta	Fragile Bones	Blue Sclera	
Pachyonychia Congenita	Natal Teeth		Thickened Nails, Pincer Nails, Staph Paronychia
Papillon-Lefèvre Syndrome	Tentorial & Chondroid Plexus Calcification		
PXE (Gronblad-Strandberg)		Angioid Streak	
Refsum Syndrome		Salt & Pepper Retinitis Pigmentosa	
Renal Disease			Lindsay's Nails
Retinoids, Indinavir, and Estrogen			Pyogenic Granuloma around nails
Richner-Hanhart		Pseudoherpetic Keratitis	
Sjögren-Larsson Syndrome		Glistening Dots Retinitis Pigmentosa	

Sturge-Weber Syndrome	Tram-Track Calcifications on Skull X-Ray		
Trichinosis, Endocarditis & Trauma			Splinter Hemorrhages
Trichorhinophalangeal Syndrome	Cone-shaped phalangeal epiphyses		
Tuberous Sclerosis	Astrocytic Hamartomas	Koenen's Tumor	
vonRecklinghausens's (NF-1)	Sphenoid Wing Dysplasia	Lisch Nodules	
Waardenburg's Syndrome		Dystopia canthorum, Heterchromia Irides	
Wilson's Disease		Kayser-Fleischer ring	Blue lunula
X-Linked Ichthyosis		Posterior comma-shaped corneal opacities (Descemet's membrane)	

Journal Squeeze!!!

Dr. Santosh Rathod

1. Toxins Detected in Laser Hair Removal Plume

IMNG Medical Media, 2013 May 03, N Osterweil

Laser plumes emitted during the procedure contain “a cocktail of volatile organic compounds,” at least 13 of which are known to be hazardous to human health, Dr. Gary S. Chuang, of the department of dermatology at Tufts Medical Center, Boston, said at the annual meeting of the American Society for Laser Medicine and Surgery. The findings further highlight the potential for harm that have already been demonstrated in association with laser procedures in the absence of safeguards such as adequate ventilation, smoke evacuators, and adequate personal protection.

Dr. Chuang and his colleagues at Massachusetts General Hospital, Harvard School of Public Health, and Boston University subjected donor hair samples to a single pulse from a diode or Alexandrite laser, captured the plumes produced, and examined them with gas chromatography. They detected the presence of approximately 300 distinct chemical compounds, 40 of which occurred in higher concentrations and 13 of which have been shown to be harmful in human and animal studies.

The compounds included:

- Benzene, toluene, and ethylbenzene (commonly found in car exhaust, cigarette smoke, glue, paint, wax and detergents, and linked to leukemia and bone marrow abnormalities.
- 2-Methylpyridine, which can cause headache and nausea.
- Diethyl phthalate, used in cosmetics and fragrances, has been shown to cause birth defects in pregnant rats.
- Trimethyl disulfide, which is primarily responsible for the foul odor from singed hair.
- Various soap and perfume components of unknown toxicity.

The researchers also collected dust samples over time to look for the concentration of particles smaller than 1 micron with and without a high-efficiency particulate air (HEPA) equipped smoke evacuator.

Normal street-level concentrations of ultrafine particles are about 4,000/cm³ per cubic centimeter, Dr. Chuang noted. When the investigators took the dust counter into the laser center waiting room, the level jumped to about 16,000/cc. During a laser procedure, the levels rose to nearly 450,000/cc. The levels slowly declined over the next 20 minutes, but still remained about fourfold higher

than normal concentrations, he said.

“The National Institute of Occupational Safety and Health recommends that with any surgical procedure that produces a plume, you want a capture velocity of about 100-150 ft/minute, and hopefully, (the evacuator) will have a HEPA filter or ultralow penetrance filter that can remove about 99.97% of airborne particulates up to 0.3 microns or greater,” he said.

Additionally, the vacuum must be no farther than 2 inches from the source, because the suction velocity decreases at greater distances. All personnel in the treatment room should wear surgical masks with a NIOSH rating of N95 or greater, he recommended.

“With chemicals, most masks are useless, so hopefully you will get an evacuator that has a chemical cartridge impregnated with charcoal, and that’s able to take out the majority of the [chemicals],” Dr Chuang said.

The study was internally supported. Dr. Chuang reported having no relevant financial disclosures.

TAKE-HOME MESSAGE

Repeated exposures could be hazardous

I think these findings raise a significant concern about safety, especially for those who repeatedly perform laser hair removal procedures. My guess is that we and our staff are at risk when we do these procedures, and so probably are the patients in that room, and the patients in the neighboring room and the hallway. For those repeatedly performing the procedure, those risks are magnified.

Short of wearing a re-breather-type respirator such as those worn by workers who handle hazardous materials, masks and evacuators may not offer sufficient protection against prolonged, repeated exposures to the chemical constituents of laser plumes.

2. Griseofulvin vs Fluconazole for Tinea Capitis in Children

J Dtsch Dermatol Ges 2013 Apr 10;[EPub Ahead of Print], A Shemer, IB Plotnik, B Davidovici, et al

TAKE-HOME MESSAGE

An Israeli study compared griseofulvin, currently the standard therapy for tinea capitis, and newer fluconazole in children, both in low and high dosages, finding no significant between-treatment difference in efficacy, although higher doses were associated with an earlier cure. Of note, the most effective dose of griseofulvin is considerably higher than that recommended by the FDA (11–18 mg/kg/day, depending on the weight of the patient). Although the distribution of the fungal species differs from that in the US (almost 50% of the children in the study had *Microsporum canis*), the study results do offer an alternative option for patients, which may be useful depending on availability, cost, and tolerability.

Review of Dermacon 2013

**Dr. Neela Bhuptani(Professor & Head),
Dr.Bharti Patel(,Professor)**

Dept. of Dermatology,STD & Leprosy, PDU Govt.
Medical college, Rajkot.

The 41st National Conference of IADVL - Dermacon 2013 was held between 24th-27th January 2013 at Ahmedabad, the financial capital of Gujarat. The key members of the Organizing committee were Dr. Suresh Joshipura (Organizing Chairperson), Dr. Ranjan Rawal (Organizing Secretary), Dr. Y.S. Marfatiya (Scientific Chairperson), Dr. Bela Shah (Scientific Chairperson), Dr. Krina Patel (Scientific Secretary) and Dr. Keyur Shah (Treasurer). The conference was attended by more than 6000 delegates.

The venue for the conference, Gujarat University Convention & Exhibition Centre, was class of its own. It was located in the heart of the city and was equipped with 5 scientific meeting rooms. The registration counter was well accessible to all delegates. There was no chaos or queues to get registered or to receive the certificates.

Pre-conference workshop was inaugurated by Dr. Bharat J. Shah, Dean, B.J. Medical College, Ahmedabad. It was planned very well by the co-ordinators Dr. Janak Thakkar, Dr. Aarti Shah and Dr. Jagdish Sakhia. There were procedures of Hands on training – lasers, fillers, botulinum toxin and newer peels. The unique feature of this workshop was that each batch was having an International Faculty, National Faculty and Expert Trainer.

CME was inaugurated by Dr. Pankaj Patel, Dean, N.H.L. Medical College, Ahmedabad. The theme of CME, 'Current concepts in Dermatology', very useful for day to day practice, was covered very well by all the speakers.

The theme of conference 'Evidence based Dermatology' was excellent.

The conference was inaugurated on the CME evening by Hon. Mayor of Ahmedabad Municipal Corporation Shree Asit Ravindraprasad Vora. Prof. K C Kandhari foundation award was conferred on Dr. Bhushan Kumar, Professor and Head, Dept. of DVL, PGIMER, Chandigarh. The orations presented in the conference were the B.M. Ambady oration on the concept of 'Clinico-cellular stability and its importance in autologous mini punch

grafting and NB UVB phototherapy in replenishment and reactivation of melanocyte in stable vitiligo' by Dr. Kaushik Lahiri, IADVL Neutrogena oration on 'Sclerotherapy in dermatological disorders' by Dr. S. Sacchidanand and IADVL systopic oration on 'Lichen Scrofulosorum – is it truly rare? – reality unleashed' by Dr. Archana Singal.

There were 12 award papers and 85 free papers. There was introduction of 'Young Dermatologists Forum' in which the post graduate students had actively participated. The innovative feature of this conference was exclusive e-posters (750 e-posters) for the very first time in the history of Dermacon.

IADVL-GSK National Quiz Programme for Post Graduates was conducted on the second day of conference. At the state level, the team from Govt. Medical College, Surat stood first and the team from Govt. Medical College, Bhavnagar stood second. The team from South Central Railway Hospital, Mettuguda, Secundrabad was the winner at the National level.

First time in history of Dermacon, there were inbuilt, pillar less, and centrally air conditioned 50,000 Sq.ft. exhibition halls for stalwarts of Pharmaceutical industries.

Alongwith an exhaustive menu for the mind, the food was extraordinarily good for all the 5 days, satisfying the taste buds of all the delegates. Gujarati food was the special attraction for all delegates. Dining area was very spacious and well planned.

Organizers of the conference took every care for the entertainment of family members. Lectures were also arranged for the accompanying persons on post menopausal problems and its management, tips on daily hair care& skin care on 25th and 26th January, which were quite informative. Extracurricular activities like Yoga, Pranayam and science of breathing were also arranged for all delegates as well as accompanying persons.

After the hectic schedule of scientific programme, the cultural programmes in the evening were very relaxing. Devang Patel and Ami Patwa Garba night on the first day was an attraction for the delegates. Live performance by renowned singer K.K. was enjoyed to the fullest by all delegates. On the last day, a theme based musical evening by Prince Group alongwith state participation was very entertaining. All national and international delegates actively participated in the cultural programmes.

The conference was very well organized and was a grand success because of the excellent team effort of the organizing committee.

CROSSWORD I

Dr. Krina Patel

Associate Professor & HOD, GMERS Medical College, SOLA, Ahmedabad

Mail your answers to – quarterderm@mail.com

Or by post – Dr. Krina Patel

Chief Editor – Quarterderm

GMERS Medical College and Hospital, SOLA,

SG Highway, Nr Gujarat Highcourt, Ahmedabad – 380 013

Two correct entries will be entitled for prize. All correct entries will be acknowledged in next issue.

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KEYS

ACROSS

1. Fibre in the skin
4. Monster linked to manifestations of SLE
6. Genetic syndrome related to Turner syndrome but with normal karyotyping.
8. Chevron nail - synonym
12. Histology of this disorder resembles that of DH (abbr.)
13. Sign elicited in Parkes-Weber syndrome
14. Commonest Pigment for dermatologists
16. Blood level of this may be used as adjunct to diagnosis and treatment response in sarcoidosis
17. This amino acid may be useful in recurrent Herpes simplex infection.
18. Typically Involved in relapsing polychondritis
21. Acute erythema and immediate burning on exposure to tar
23. Skin lesions likened to – in kwashiorkor
24. Free clinical search engine giving the best of evidence based medicine
25. Milan citran skin

DOWN

- 1 Multiple lentiginos syndrome
- 2 Friction dermatitis due to spectacles
- 3 Diseases which are rare and not many pharmaceutical companies want to develop medicines for the same are known as
5. This famous tennis player died of AIDS due to transfusion
7. For diagnosis and staging of MF this evaluation is must
9. This US president had melanoma and his daughter died of melanoma
10. Local anesthetic agent likely to cause methhemoglobinemia
11. Low grade slowly developing MF
13. Umbilical cord in congenital syphilis
15. Commonly Involved in sarcoidosis, leishmaniasis.
17. Malignant syphilis
19. Metabolic disorder may be associated with eruptive syringoma
20. Blood Test helpful for allergic disorders
22. Sensory function often first to be impaired in lepromatous leprosy

NEWS & POSTS

Dear Dermatologists,

After a long innings - 62 years in practice,
in Ahmedabad I would like to retire and
would like to find a colleague,
who will eventually take over my practice.

Location: Doctor House, Ellisbridge,
Ahmedabad a land mark building for Doctors.
After sale, for a smooth transition,
I will attend the clinic for some time.

Interested Doctors.
Pl. contact Rajen - 98253 21996, or
drcfshah@gmail.com



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laboratories (guj) pvt. ltd.

210, 'Aakash' Avenue, Nr. Mithakhali Underbridge, Navarangpura, Ahmedabad-380 009.
Phone : (079)-26461342 / 65446888 Fax : (079) 2643 0086
Email : dermocare2000@yahoo.com

www.dermocarelab.com