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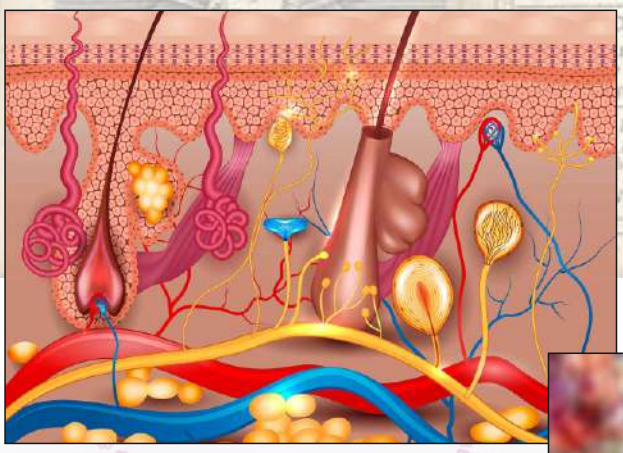

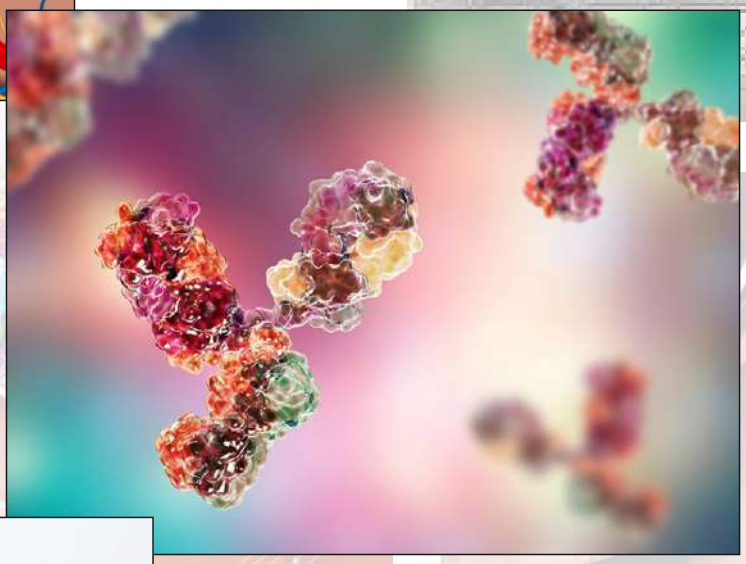

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Handwashing – Why It Is Important

Editorial

Handwashing helps prevent spread of infectious diseases

A number of infectious diseases can be spread from one person to another by contaminated hands.

These diseases include gastrointestinal infections, such as salmonellosis, and respiratory infections, such as influenza, colds and coronavirus(COVID-19).

Washing your hands properly with soap and water can help prevent the spread of the germs (like bacteria and viruses) that cause these diseases.

Some forms of gastrointestinal and respiratory infections can cause serious complications, especially for young children, the elderly, or those with a weakened immune system.

Dr Kakali Sen
Hony. Editor, Your Health

When to wash your hands

You should wash your hands thoroughly:

- after using the toilet
- after changing nappies
- before, during and after preparing food
- between handling raw and cooked or ready-to-eat food
- before eating
- after blowing your nose, coughing or sneezing
- after using a tissue or handkerchief
- before and after attending to sick children or other family members
- after smoking
- after handling rubbish or working in the garden
- after handling animals
- when you get home, arrive at other people's homes, at venues or at work.

Avoid touching your eyes, nose, and mouth with unwashed hands.

If you feel a cough or sneeze is coming on, make sure to cough or sneeze into a tissue and then throw it away and wash your hands.

If you do not have a tissue, cough or sneeze into your elbow. It's a part of your body less likely to touch other surfaces and will help stop the spread of nasty germs.

How to wash your hands properly

To wash hands properly:

- **Wet your hands with clean, running water, turn off the tap.**
- **Apply soap and lather well** for 20 seconds (or longer if the dirt is ingrained).
- **Rub hands together rapidly** across all surfaces of your hands and wrists.
- Don't forget the backs of your hands, your wrists, between your fingers and under your fingernails.
- If possible, remove rings and watches before you wash your hands, or ensure you move the rings to wash under them, as microorganisms can exist.
- **Rinse well under running water** and make sure all traces of soap are removed.

- **Dry your hands using a clean towel or air dry them.**
- It is best to use paper towels (or single-use cloth towel).
- Dry under any rings, as they can be a source of future contamination if they remain moist.
- Hot air driers can be used.

At home, give each family member their own towel and wash the towels often.

Use running water

Use running water instead of a basin of standing water that could become contaminated through use.

Warm water may be better than cold for handwashing as soap lathers (soaps up) better with warm water. However, cold water and soap are still suitable.

Hot water can damage the skin's natural oils. Over time, this can cause dermatitis.

Soap is important

Washing hands with soap and water will remove substantially more disease-causing organisms than washing hands with water alone.

For people who find that soap causes skin irritation, it is useful to note that soaps can have a different pH – they may be neutral, slightly alkaline or slightly acidic, and perfumes in soap may also cause irritation. Changing soap may help some people.

Liquid soap is best

Generally, it is better to use liquid soap than bar soap, particularly at work. However, bar soap is better than no soap.

No advantage to using antibacterial soap

When following the handwashing steps outlined above, all soaps are equally effective at removing disease causing germs. Antibacterial soap is unnecessary and does not offer an advantage over regular soap.

Soap and water is better than hand sanitiser

Alcohol-based hand sanitisers are effective against some viruses (such as coronavirus), however they are not effective against gastroenteritis.

Washing hands with soap and water is the best way to prevent gastroenteritis infection.

It is best to wash hands with soap and water. If unavailable, use alcohol-based hand sanitiser containing at least 60% alcohol.

Take care of your hands

Handwashing is only one part of hand hygiene. Looking after your skin generally is important, as your skin is your most effective barrier against infection.

After your hands have been dried thoroughly, you can help to look after your hands if you:

- Apply a water-based absorbent hand cream 3- 4 times a day, or more frequently if your hands are constantly in water.
- Use gloves when washing dishes to protect your hands.
- Use gloves when gardening to prevent a build-up of ingrained soil or scratches.
- Consult a doctor if a skin irritation develops or continues.

Teach hand hygiene to children

The creation of healthy habits during childhood is important to ensure lifelong healthy decisions and actions.

Get kids involved with ''Soapy Hero training to help keep their classmates, family and friends healthy and stop the spread of dangerous infectious diseases with hand hygiene.

Watch this video about hand hygiene.

From the Desk of Secretary

Dr Samarendra Kumar Basu
Hony. Secretary, Your Health

Skin covers the body's entire external surface. It is made up of three layers, the epidermis, dermis, and the hypodermis, all three of which vary significantly in their anatomy and function. The skin's structure is made up of an intricate network which serves as the body's initial barrier against pathogens, UV light, and chemicals, and mechanical injury. It also regulates temperature and the amount of water released into the environment.

Epidermis : The thickness of each layer of the skin varies depending on body region and categorized based on the thickness of the epidermal and dermal layers. Hairless skin found in the palms of the hands and soles of the feet is thickest because the epidermis contains an extra layer, the stratum lucidum. The upper back is considered thickest based on the thickness of the dermis, but it is considered “thin skin” histologically because the epidermal thickness lacks the stratum lucidum layer and is thinner than hairless skin.

Dermis : The dermis is connected to the epidermis at the level of the basement membrane and consists of two layers, of connective tissue, the papillary and reticular layers which merge together without clear demarcation. The papillary layer is the upper layer, thinner, composed of loose connective tissue and contacts epidermis. The reticular layer is the deeper layer, thicker, less cellular, and consists of dense connective tissue/ bundles of collagen fibers. The dermis houses the sweat glands, hair, hair follicles, muscles, sensory neurons, and blood vessels.

Hypodermis : The hypodermis is deep to the dermis and is also called subcutaneous fascia. It is the deepest layer of skin and contains adipose lobules along with some skin appendages like the hair follicles, sensory neurons, and blood vessels.

A daily skin care routine has four basic steps you can do once in the morning and once before you sleep. **Cleansing:** Choose a cleanser that doesn't leave your skin tight after washing. Clean your face no more than twice a day, or just once, if you have dry skin and don't wear makeup. Avoid washing for that squeaky-clean feeling because that means your skin's natural oils are gone.

Serums: A serum with vitamin C or growth factors or peptides would be better in the morning, under sunscreen. At night, retinol or prescription retinoids work best.

Moisturizer: Even oily skin needs moisturizer, but use one that is lightweight, gel-based, and non-comedogenic, or doesn't block your pores. Dry skin may benefit from more cream-based moisturizers. Most brands will label their products as gel or cream on their packaging.

Sunscreen: Apply sunscreen with at least 30 SPF 15 minutes before heading outdoors, as it takes a while for sunscreen to activate. Darker skin tones actually need more sun protection because hyperpigmentation is harder to correct.

There are many diseases related to skin like Seborrheic dermatitis, Moles, Rosacea, Lupus Psoriasis, Eczema, Vitiligo, Acne , Hives, Fungal nail infection, cold sore, Candidiasis , Skin cancer, Hemangiomas, Measles, Impetigo etc. In this book the authors have elaborated on various skin diseases. I am thankful to all.

Dermatology: The Shifting Paradigm

Guest Editorial

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If we have to tender a presumption about dermatology as a whole, we may better take a look at the following lines from the first editorial printed in Indian Journal of Dermatology in its very first issue in 1955, which may be based on a diametrically opposite perspective and written in a time completely different from ours, but ironically, the words are very much relevant even today: "In this country, however, the study of Dermatology has not advanced as satisfactorily as it should have done. General practitioners have to tackle an increasingly large number of cases because skin complaints are generally more prevalent among the poorer and labor classes. The study of skin disease occupies but a small corner in the undergraduate curriculum, and very little importance is attached to this particular branch. Questions on skin disease figure rarely, if ever, on the MBBS question papers. As a result, students' interest in the skin and its complaints is not properly stimulated. They feel the consequent gap in their knowledge when they go out to practise. Not unnaturally, patients have to suffer at their hands both physically and economically."[1]

In the last almost seven decades after that editorial, the composite discipline of Dermato-Venero-Leprology has undergone a phenomenal change, quality and quantity wise.

This change is more evident in the last 10 years.

This distinct and dramatic metamorphosis is based on some decisive shift in our understanding of the pathomechanism of several diseases at the molecular level. This was supplemented by a multitude of fresh evolving therapeutic modalities with the application of cutting-edge technologies. With advent of AI based programs the change is certainly faster than we could ever imagine.

Different tentacles of the specialty such as esthetic surgery, phototherapy, dermatosurgery, cosmetology, and biologics have stretched the horizon of the subject itself.[2]

Astonishingly voluminous progression and growth of dermatology in recent times have evidently outshined several other medical disciplines with remarkable pace, grace, and poise.[2,3]

Globally, the dermatology contract research organization (CRO) market was valued at US$ 5,154.8 million in 2021 and is projected to grow at a compound annual growth rate of 8.7% during the forecast period 2022–2032.[4]

The dermatology market is the new emerging king of the pharmaceutical industry with great demand in the market. The Rs. 8,700 crore dermatology market has been the fastest-growing therapy segment in the domestic market over the last 3 years exceeding the other major categories of the pharmaceutical sector.[4]

Dermatology is now one of the most preferred medical specialties in India.

I feel privileged for getting this honor as the guest editor of this special issue on dermatology for Your Health, the Heritage publication of IMA

This issue has been crafted and enriched with contributions from some top thought leaders in this field.

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Misuse of Topical Corticosteroids Causing Damage to Skin- A National Disaster of Epidemic Proportions

Abstract:

Topical corticosteroids (TC) have been used by dermatologists in a large number of dermatological conditions for the past seventy years. . However, a recent spurt in the misuse of the drug has emerged as a major medical problem of epidemic proportions. The drug is prescribed by non-dermatologists in a number of diseases where TC is contraindicated. It is also used by laymen as OTC drugs. Fixed drug combinations (FDC) produced by pharmaceutical companies also add fuel to the fire. The result is either aggravation of existing diseases (for example dermatophytosis) or the development of newer disorders such as Topical steroid damaged/dependant face (TSDF). To combat this misuse dermatologists individually and as members of IADVL are campaigning vigorously in various print, electronic and social media

KEYWORDS

Topical corticosteroids, Misuse, Skin damage, Addiction, TSDF

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Introduction:

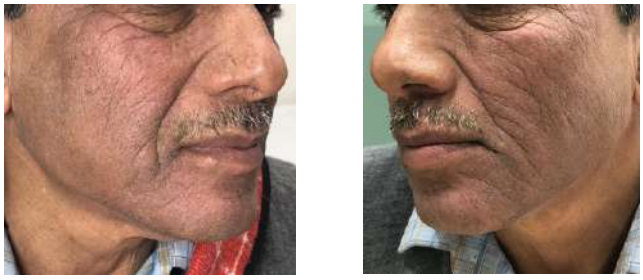
The largest organ of the human body - the skin - covers it entirely thus protecting all the internal organs. It forms the first line of defence against various external factors. However, due to various misconceptions and malpractices by human beings, the skin itself can be harmed. Injudicious application of cosmetics, drugs and other allergy-producing substances may result in various forms of damage to the skin. One such drug is the TC which has a highly beneficial action in a number of critical skin disorders. However, it is quite frequently overused and misused causing substantial damage to the skin. In recent years, abuse of topical corticosteroids has increased to epidemic proportions.[3]

Topical Corticosteroids:

Corticosteroids are the synthetic analogues of the steroid group of hormones. Their discovery in 1949 by Edward Kendall was a turning point in the history of modern medicine because these were life-saving drugs that revolutionized the treatment of a large number of serious, acute and chronic disorders.[1] Later Schulzberger and Witten introduced the first TC in 1952 (Compound F)[2] Since TC also helped in ameliorating a large number of dermatoses, dermatologists welcomed the drug with open arms. . As with any new drug, TCs were not only used extensively in proper indications but were also misused in inappropriate conditions. While TCs benefitted a large number of patients when used judiciously, its misuse and abuse led to the development of various side effects and also, dependence on the drug.[4]

Rational USE of TC

TC is a drug which like all other drugs should only be applied to the skin when prescribed by a practitioner of modern medicine (popularly known as allopath). They should never be purchased OTC (over the counter).[5] They should only be prescribed by doctors in dermatological disorders which are steroid-responsive and where the application of TC is not contradicted. They should never be prescribed by practitioner s of alternative medicine. It should always be borne in mind that each and every topical medication (creams, ointments, lotions etc) applied on the skin pass through the layers of the skin and are subsequently absorbed internally. Thus, if applied irrationally TCs may have an adverse effect on the internal organs. TCs have been classified into 7 classes



Topical Steroid misuse on Face

according to their potencies which are determined by their properties (particularly their vasoconstrictive properties) . The least potent are Class I and II. They are thus suitable for application on the soft skin of the face and body folds as also in children and the elderly (in whom the skin is thin and hence vulnerable to the ill effects of potent TCs) . Class VI and VII are classified as super-potent TCs. Though useful in a number of allergic disorders they should be applied very cautiously because they can pass through the skin very rapidly and if used for a period of more than 2 weeks continuously may cause some major damage to the internal organs. Hence rational use of TC needs a lot of knowledge, thought and rationality.[6]

Physical Damages due to TC Misuse

Misuse of TC may cause a plethora of local and systemic damage. The most prominent effects on the skin are monomorphic acneiform eruptions, bacterial infections, fungal infections, atrophy, perioral dermatitis , increase in skin transparency and brightness, striae or stretch marks , scars , telangiectasia , hypertrichosis, rosacea (red face), ulcers and hypopigmentation.[7]Systemic effects include suppression of adrenal glands resulting in Cushing's syndrome, hypertension, glaucoma, cataracts (particularly when potent TC are applied on face) and growth retardation in children.[8]

TC ADDICTION

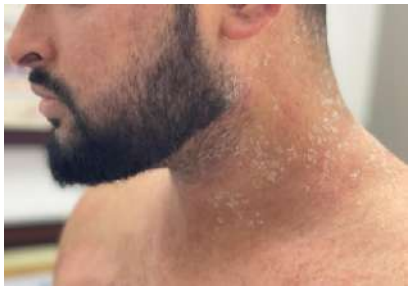
Chronic misuse of TC can cause psychological and physical (cutaneous) dependence on the drug. The patient becomes addicted to the TC resulting in Topical Corticosteroid Addiction (TCA); a condition which is similar to chronic alcohol addiction[9]. TCA was first reported by Burry et al in1973. [10] When the patient attempts to stop the TC there is a rebound or flare of symptoms. This may be physically and psychologically distressing to the patient. There is intense redness of the face (erythema and flare) followed by desquamation. This discomfort may be unbearable to the patient who can start re-using the drug in order to maintain normal social functioning resisting any further attempt to withdraw it or the patient may cooperate with the dermatologist and avoid using it. If the patient does not re-use the drug then there will be a few more episodes of redness followed by peeling before the patient is completely cured. The usual sites of TCA are the face (commonest), flexures, groins, perianal and genital areas.[11] By far the commonest site of such misuse is the face. [12]

Misuse of TC in India

TC misuse in India has developed into a rampant epidemic. A number of social, legal and financial factors have helped in adding fuel to the fire. The health authorities who are more concerned with diseases causing mortality, are least concerned or may not be aware about misuse of TC since it causes a major amount of morbidity but no mortality. Hence while only one TC (Betamethasone valerate) figures in the list of Essential medicines 2022 , the price of 99.8% TCs are not controlled by the Drug price control order. This apathy is further exemplified by the fact that out of about 20 TC molecules, only 4 TC molecules are listed as Schedule H drugs. However, due to lax policing by the authorities, any Indian citizen can purchase even these molecules from the chemists as OTC. Hence chemists sell all TC molecules at random without any fear of legal backlash. Two more factors play an unfortunate role in this uncontrolled sale of TCs. First, there is a a mistaken knowledge among a large number of Indians that since salesmen at chemists are handling drugs daily they possess equivalent or even more knowledge about TC than doctors. Secondly, they can avoid the doctor's fees by purchasing the TC directly from the chemist. This last factor also causes some patients to “repeat” prescriptions that is present the same prescription over and over again for months on end without ever visiting the doctor. Such a malpractice can easily lead to disastrous consequences. For example, many patients continue to use super-potent TCs for months on end and develop most of the local and systemic side effects of TCs.[6]

Pharmaceuticals also join in this rat race of financial

Topical Steroid misuse on Face



Topical Steroid misuse on Neck

bonanza by manufacturing and marketing fixed drug combination drugs (FDC) - for example TC + antifungal + antibiotic, which are totally unethical products and banned in most other countries. In September 2018, in a welcome move, the Government of India banned 28 such FDC containing - and potent TC. Another FDC which is rampantly used to increase "fairness" contains mometasone, a mid-potent TC which should never be used on the face[14].

The medical representatives of such companies also contribute by promoting TC to practitioners of alternative medicine and quacks who possess no knowledge of the uses and side-effects of TCs.[3]

But he maximum misuse is done by the end- users that is by the patients and laymen. Many patients do not consult dermatologists at all and apply TCs on the recommendation of friends, neighbours, relatives and sundry other "well-wishers".. They apply TCs in disorders which are not steroid-responsive or which may be aggravated on application of TCs //for example fungal infections).[13] However, they ultimately visit the dermatologist only when their disease is aggravated or when complications set in.

One of the major instances of TC misuse is the application of TCs on the face as fairness creams. TCs may cause an increase in skin brightness and hypopigmentation resulting in a phenomenon that most laymen (perhaps inspired by beauticians) understand as "glow". However, they do not realize that the skin that they flaunt as glowing skin is actually diseased skin. ,he fairness craze due to enhanced beauty consciousness induces laymen to apply TCs of various potencies for prolonged periods on their faces. One particular brand of the TC molecule Betamethasone valerate is perhaps the most popular fairness creams sold randomly as OTC.[14] The result is an epidemic of skin rashes appearing as side-effects of TC and severe attacks of TC addiction resulting in a condition which has been named as " Topical steroid damaged/dependant face. (TSDF)"[15]

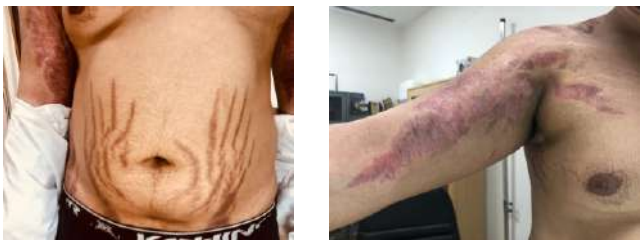
TSDF is defined as the semi-permanent or permanent damage to the skin of the face precipitated by the irrational, indiscriminate, unsupervised, or prolonged use of TCs resulting in a plethora of cutaneous signs and symptoms and psychological dependence on the drug.[15] The misuse of TC misuse on the face was first reported in India in 2006[16] Later that year, a proposal named "Stop OTC supply of potent topical steroids" An alarming rise in the number of cases of TC misuse on face resulting in steroid dependence was observed and the entity was labelled as "Topical steroid-dependent/damaged face" by Lahiri K in March 2008. Later that year a first of its kind IADVL sponsored multi-centric study on TSDF was conducted in 12 centres all over India and its report was presented at the national conference of IADVL (DERMACON) at Bengaluru in January 2009. The report was subsequently published in 2011.[12]

Suggested Steps to Counter TC Misuse

The galloping spread of TC misuse in India may be halted or at least decelerated with a few suggested steps. Manufacturing of FDC containing TC should be stopped forthwith by Pharmaceutical companies. TC advertisements in lay media and sale of TCs OTC should be banned. Prescription of TCs by unauthorised persons like quacks and alternative practitioners should be declared illegal. The regulatory authorities should ensure strict measures to enforce the laws enacted by them to this effect. Dermatologists and non-dermatologists should be sensitized about the bad effects of misuse of TC. Finally, an Intensive print and social media campaign against TC misuse may help in alleviating the misery of millions of Indians.

CONCLUSION

TC misuse has emerged as a major cause of concern for dermatologists particularly in India. While the treatment of the side-effects is easy, TC addiction is difficult to be cured in view of the easy OTC availability of TC. A concerted campaign by Indian dermatologists to stop TC abuse his gradually gained momentum over past couple of decades. Dermatologists have been posting photographs and discussing this issue in a Facebook group named "No steroid cream on face without a doctor's prescription". The print and electronic media are also being sensitized on this issue. To give a concerted and effective push to the hitherto scattered efforts by dermatologists, the Indian Association of Dermatologists, Venereologists and Leprologists (IADVL), the largest body of Indian



Stretch Marks due to Topical Steroid misuse

dermatologists formed a taskforce named IADVL Taskforce against topical steroid abuse (ITATSA) to sensitize the government, pharmaceuticals, patients and laymen. A PIL has also been lodged in the Delhi High Court by ITATSA on behalf of IADVL. One may surely be optimistic that these efforts will bear fruit someday in the near future and the rampant misuse and abuse of TC will ultimately come to a halt.

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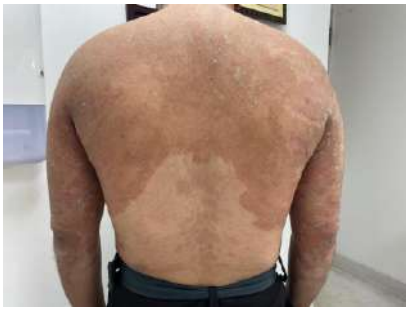

Topical Steroid misuse on Back

Resulting in fungal infection

Urticaria

Abstract

Urticaria is characterized by appearance of transient wheals with or without angioedema. Urticaria can be classified to be spontaneous or inducible, as well as acute or chronic. Acute spontaneous urticaria can have an allergic basis with infections like URTI, drugs being the known causes along with idiopathic causes. Although autoimmune etiology has been mentioned in approximately one-third of patients with CSU (chronic spontaneous urticaria), almost half of the cases remain idiopathic. Nonsteroidal anti-inflammatory drugs, foods, alcohol, stress, and infections can act as a trigger to aggravate CSU but these are not the underlying causes. Inducible urticaria occur in response to a particular trigger as mentioned by the history and can be elicited by the provocative tests. The primary effector cell of urticaria is the mast cell, and histamine from mast cells is the major mediator of pruritus and wheals. Angioedema is also mediated by mast cell mediators like histamine, but bradykinin is the mediator in angioedema without wheals as in hereditary angioedema and angiotensin-converting enzyme (ACE) inhibitor-induced angioedema. Second generation anti-histamines form the first line therapy for urticaria. Omalizumab (anti-IgE antibody) and immunosuppressives (cyclosporine) can be used in refractory cases along with anti-histamines.

Keywords: Urticaria, Chronic spontaneous urticaria, inducible urticaria, Autoimmune urticaria, Angioedema.

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Urticaria

Introduction: Urticaria is characterized by occurrence of wheals with or without angioedema. A wheal usually lasts for less than 24 hours and is histamine mediated. It has to differentiated from the common urticaria mimics such as urticarial vasculitis. Chronic urticaria can lead to a poor quality of life by causing significant adverse impact on sleep, performance and financial status of the patients.

Definition: Urticaria is a condition characterized by the development of wheals (hives), angioedema, or both.[1]

A wheal is identified by following features (Fig 1)

a sharply defined, superficial swelling of variable size and shape, generally surrounded by reflex erythema,

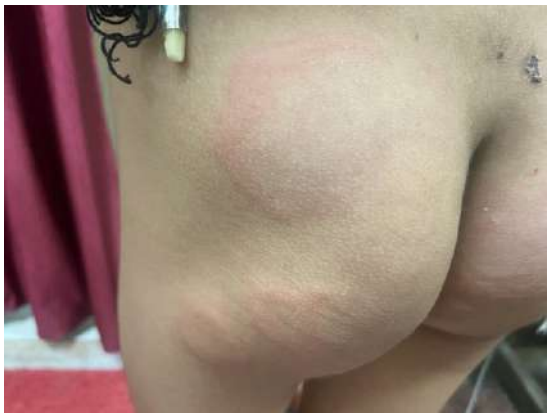

Fig 1: An erythematous wheal in a 14 year old child

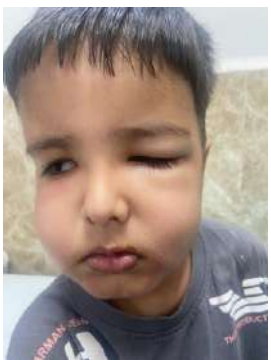

Fig 2: Angioedema in a child showing swelling of the face with eyelid involvement

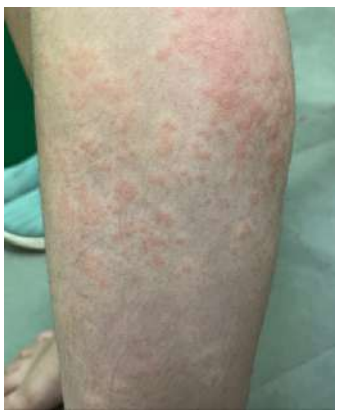

Fig 3: A case of acute urticaria with erythematous wheals over leg.

an itching or sometimes burning sensation,

a history of being transient in nature, with the skin returning to its normal appearance, usually within 30 min to 24 h.

Angioedema is characterized by a transient vascular reaction of deep dermal/subcutaneous tissues or mucosal/submucosal tissues as a result of the increased permeability of blood vessels resulting in localised tissue swelling.[2] Angioedema can occur with or without wheals in different clinical settings. (Fig 2) Both wheals and angioedema can be part of a more serious, life threatening hypersensitivity reaction which involves various organ systems along with the cutaneous changes called anaphylaxis.[3]

Epidemiology: Urticaria is a very common skin condition presenting to the dermatology clinic. Based on the age range and method of sampling, the estimated lifetime prevalence of urticaria in the general population ranges from 8 to 22% and in chronic urticaria it ranges from 2 to 3%. Prevalence of urticaria is more common in women with a female: male ratio of ~2:1 for chronic spontaneous urticaria.[4,6]

Classification of urticaria: Urticaria is classified based on its duration, as acute or chronic. However, all urticarias are acute initially. Acute urticaria is defined as the occurrence of wheals, angioedema, or both for 6 weeks or less. Most of these patients probably fall into the spontaneous category, because physical urticarias and urticarial vasculitis tend to persist beyond 6 weeks.

Causes of acute urticaria is depicted in a Fig 4 where infections i.e. URTIs account for 40 % of the causes of acute urticaria. Notably food allergy accounts for only 1% and in half of the cases the cause is idiopathic.[5]

Chronic urticaria is defined as the occurrence of wheals, angioedema, or both for more than 6 weeks. The term "chronic urticaria" should only be applied to continuous urticaria occurring at least twice a week off treatment. Urticarial episodes which are less frequent than this over a long period is called episodic (or recurrent). Chronic urticaria can also be classified as spontaneous or inducible based on definite trigger factor (Fig 5)[1]. Inducible urticaria is characterized by definite and subtype-specific triggers in the development of wheals, angioedema, or both. These triggers are definite because wheals, angioedema, or both always occur when the trigger is present and absent when the trigger is absent.[6]

Chronic spontaneous urticaria has been associated with autoimmune thyroid disease and other autoimmune conditions, such as vitiligo, insulin-dependent diabetes, rheumatoid arthritis, and pernicious anaemia. Autoimmune urticaria represents those patients with functional autoantibodies against FcεRI or the Fc portion of IgE.[7] A possible association between Helicobacter pylori infection and urticaria has been suggested and studies shows a higher frequency of urticaria remission when the infection was eradicated. Parasitic infections such as intestinal strongyloidiasis, dental infections or gastrointestinal candidiasis have been mentioned as very uncommon cause of urticaria.

Inducible Urticarias (syn. Physical Urticarias): It refers to a distinct subgroup of the urticaria that is induced by an exogenous stimulus rather than occurring spontaneously. The inducible urticarias are classified by the predominant stimulus that triggers wheals, angioedema, or anaphylaxis.[8] The lesions of most physical urticarias occur within minutes of provocation and generally resolve within 2 hours, apart from a few urticarias (e.g., delayed pressure urticaria, delayed dermographism) which develop after a delay of several hours and persist for 24 hours or longer. The wheals are generally localized to the stimulated area of skin. Types of inducible urticaria is depicted in Table 1

Classification of inducible urticariasSymptomatic dermographismDelayed pressure urticariaSolar

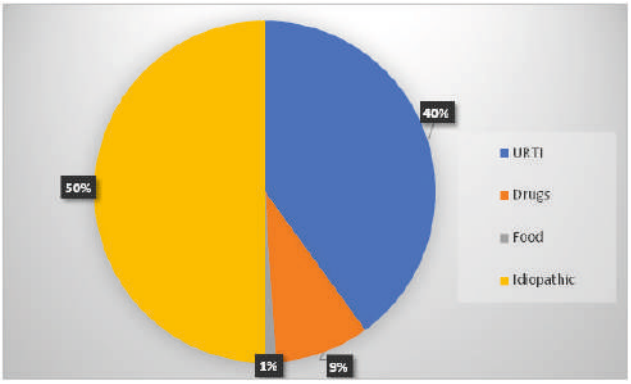

Fig 4: Causes of acute urticaria[5]

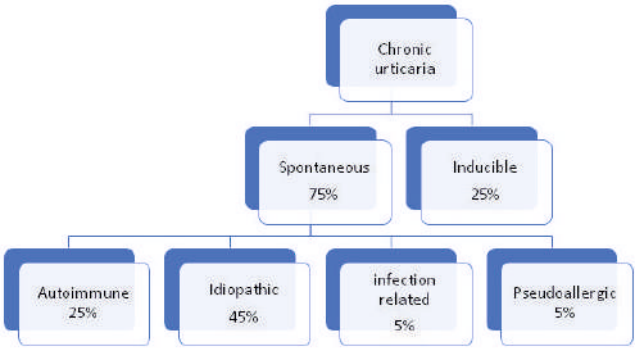

Fig 5: Causes of chronic urticaria (with the percentage distribution frequency)[10]

urticaria Cholinergic urticaria Contact urticaria Aquagenic urticaria Cold urticaria Heat urticaria Table 1: Types of inducible urticaria [1,6]

Pathophysiological aspects

Primary effector cell of urticaria are mast cells and they play a central role in the pathogenesis of the disease. These cells are widely distributed throughout the body and express high-affinity IgE receptors (FcεRI) and are therefore capable of participating in IgE-dependent allergic reaction.[9] Cross-linking of two or more adjacent FcεRI on the mast cell membrane initiates a chain of calcium- and energy-dependent steps leading to fusion of storage granules with the cell membrane and externalization of their contents. This is known as degranulation.

Degranulation of the mast cells leading to the release of pre-formed mediators can occur by cross-linking of two or more adjacent FcεRI on the mast cell membrane. Binding of the receptor-bound specific IgE by the allergens, anti-IgE and anti-FcεRI antibodies are the known immunological degranulating stimuli. Classic immediate hypersensitivity reactions involve binding of receptor-bound specific IgE by allergen. This is the basis of *Type 1 autoimmune urticaria.* Immunologic degranulating stimuli act through the IgE receptor antibodies such as anti-IgE and anti-FcεRI antibodies in *Type II autoimmune urticaria.* Substance P stem cell factor, C5a, Codeine are few non-immunological stimuli for degranulation of mast cells. (Fig 6)

Post degranulation, both preformed mediators such as histamine, proteases, heparin and newly synthesized proinflammatory mediators i.e. Prostaglandin D2, leukotrienes are released from mast cells. Histamine and other proinflammatory mediators such as TNF, IL-8 released after degranulation bind to the receptors on postcapillary venules in the skin, leading to vasodilation and increased permeability to large plasma proteins, including albumin and immunoglobulins. These mediators also upregulate the expression of adhesion molecules on endothelial cells which promotes the migration of circulating inflammatory cells including eosinophils, basophils, neutrophils in the urticarial lesions.[11] Functional IgG autoantibodies that release histamine (and other mediators) from mast cells and basophils have been detected in the serum of 30–50% of patients with chronic spontaneous urticaria, based on in-vitro assay.

Histologically, wheals are characterized by oedema of the upper and mid dermis, with dilatation and increased permeability of the postcapillary venules as well as lymphatic vessels of the upper dermis. In angioedema, similar changes occur primarily in the lower dermis and the subcutis. There is a mixed inflammatory perivascular infiltrate of variable intensity, consisting of T cells, eosinophils, basophils, and other cells in the area of wheal.[12]

Diagnosis of urticaria: The first step in the diagnostic workup of all urticaria patients is a detailed history taking about the frequency and severity of the wheals. History should also include triggers of the disease, drug intake and response to treatment.

The second step is the physical examination of the patient. As wheals and angioedema are transient and may not be present at the time of physical examination, it is important to review patients' documentation of signs and symptoms (including pictures of wheals and/or angioedema).

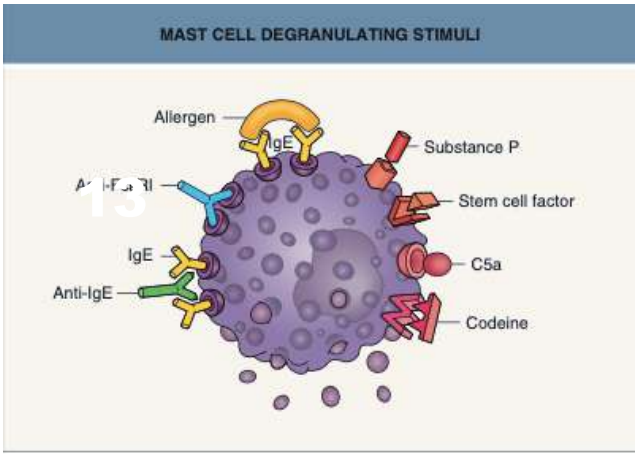

Fig 6: Mast cell degranulating stimuli.
Both immunologic and non- immunologic stimuli can lead to release of mediators.[10]

Classification of inducible Urticarias

1. Symptomatic dermographism
2. Delayed pressure urticaria
3. Solar urticaria
4. Cholinergic urticaria
5. Contact urticaria
6. Aquagenic urticaria
7. Cold urticaria
8. Heat urticaria

The third step, in chronic urticaria, is a basic diagnostic workup, with limited tests as recommended by the guidelines. In acute spontaneous urticaria, no investigation is needed. The only exception is the suspicion of acute urticaria due to a type I food allergy in sensitized patients or drug hypersensitivity, especially for non-steroidal anti-inflammatory drugs (NSAIDs). In these cases, allergy testing and relevant patient education can be useful to allow patients to avoid re-exposure to relevant causative factors.[1]

In CSU, differential blood count, ESR and/or CRP, IgG anti-TPO and total IgE are the basic tests to be done. Further detailed tests for functional autoantibodies (eg, basophil test); allergen avoidance test, eg, avoidance diet should be done after a detailed history and in specific cases after the basic tests have been done. For inducible urticaria, there is a set of provocative tests which has been mentioned such as ice cube test in cold urticaria, pressure test for delayed pressure urticaria, UV and visible light of different wavelengths for solar urticaria.

Patient assessment: Urticaria activity score (UAS) is the commonest tool to objectively assess the severity of symptoms and for follow up on treatment. Angioedema activity score (AAS), the CU quality of life questionnaire (CU-Q2oL), the angioedema quality of life questionnaire (AE-QoL), the urticaria control test (UCT), and the angioedema control test (AECT) are other few tools for the same.

In the Urticaria activity score (UAS), scoring is done as per the wheals intensity (0-3) and pruritus (0-3) to calculate the total score. (Table 2)

ScoreWhealsPruritus0NoneNone1Mild (<20 wheals/24 h)Mild (present but not annoying or troublesome)2Moderate (20–50 wheals/24 h)Moderate (troublesome but does not interfere with normal daily activity or sleep)3Intense (>50 wheals/24 h or large confluent areas of wheals)Intense (severe pruritus, which is sufficiently troublesome to interfere with normal daily activity or sleep) Table 2: Urticaria activity score

Differential diagnosis: Anaphylaxis, autoinflammatory syndromes, urticarial vasculitis, or bradykinin-mediated angioedema including hereditary angioedema (HAE) are the differential diagnosis to be kept in mind when treating urticaria. It is important to distinguish urticaria from *urticarial dermatoses,* such as urticarial drug eruptions, eosinophilic cellulitis and the urticarial phase of pemphigoid which are other urticarial dermatoses which needs to be differentiated from urticaria. These are separate clinical conditions and should be dealt with accordingly.

Management of urticaria

Aim of treatment: The goal of treatment is to offer effective and safe treatment to achieve continuous UAS7 = 0, complete control and a normalization of quality of life. Aim should be to search for and possibly eliminate the underlying causes and avoidance of eliciting factors. For example, in CSU, avoidance of stress or the intake of NSAIDs can help to reduce the disease exacerbations.[13] Elimination of H. pylori infections have been reported to lead to disease remissions in CSU.[14] A pseudo-allergen free diet, containing only low levels of natural and artificial food pseudo-allergens, has been tested in patients with variable improvement in the symptoms.[15] Other form on non-pharmacological therapy is the induction of tolerance which works in inducible urticaria such as solar urticaria where a rush hardening therapy with UV-A has been reported to be effective within 3 days.[16] However, tolerance induction lasts for a few days; thus, a consistent daily exposure to the stimulus just at threshold level is required for constant relief.

Symptomatic pharmacological treatment: Current treatment options target mast cell mediators such as histamine or activators such as autoantibodies.(Fig 4)

H_1-antihistamine treatment

The older 1st generation H_1-antihistamines have anticholinergic and sedative effects, and many drug interactions. They can also interfere with rapid eye movement (REM) sleep and have an impact on learning and performance. In a GA²LEN position paper, it has been strongly recommended not to use 1st generation H_1-antihistamines any longer in allergy both for adults and especially in children.[17] Modern 2nd generation H_1-antihistamines are minimally or non-sedating and free of anticholinergic effects with an exception of astemizole and terfenadine (withdrawn due to cardiotoxic effects). [18] Most 2nd generation H_1-antihistamines have been tested specifically in urticaria, with supporting evidence of use of bilastine, cetirizine, desloratadine, ebastine, fexofenadine, levocetirizine, loratadine, and rupatadine. The use of a standard-dosed modern 2nd generation H_1-antihistamines is recommended as the first-line symptomatic treatment for urticaria.[1] However, no recommendation has been made on the preference of 2nd generation H_1-antihistamines as well-designed clinical trials comparing the efficacy and safety of all modern 2nd generation H_1-antihistamines in urticaria are largely lacking. Up-dosing of a 2nd generation H_1-antihistamine up to fourfold in patients with chronic urticaria unresponsive to a standard-dosed 2nd generation H_1-antihistamines as second-line treatment before other treatments are considered

has been recommended as per the guideline. The guideline also recommends the use of non-sedating 2nd generation H_1-antihistamines daily, to prevent the occurrence of wheals and angioedema, rather than on demand. Combination of different anti-histamines at the same time is not recommended.

Omalizumab treatment: Omalizumab is the only FDA approved treatment in urticaria for patients who do not show adequate benefit from treatment with a 2nd generation H_1-antihistamine, and therefore is the next step in the algorithm. Omalizumab (anti-IgE) has been found to be very effective and safe in the treatment of CSU. The recommended initial dose in CSU is 300 mg every 4 weeks. Dosing is independent of total serum IgE level. Updosing of omalizumab treatment can be done (off-level) to increase the dose up to 600mg per month or by reducing the frequency to every fortnight in patient having insufficient response to the recommended dose of omalizumab.[19-,22]

Ciclosporin treatment: Patients with urticaria who do not show sufficient benefit from treatment with omalizumab, should be treated with ciclosporin 3.5–5 mg/kg per day. Ciclosporin is an off-label treatment for urticaria and is recommended only for patients with severe disease refractory to any dose of antihistamine and omalizumab in combination. It works as an immune-suppressive and has a moderate, direct effect on mast cell release.[23,24]

Other treatment options: Leukotriene receptor antagonists have been used in the treatment of urticaria. The level of evidence for the efficacy of leukotriene receptor antagonists in urticaria is low but best for montelukast. In acute urticaria and acute exacerbations of CSU, a short course of oral corticosteroids, that is, treatment of a maximum of up to 10 days can be helpful to reduce disease duration/activity.[25,26] Immuno-suppressive like methotrexate and mycophenolate mofetil have also been tried in CSU with variable results. For the treatment of CSU and symptomatic dermographism, UV-B (narrow band-UVB), UV-A, and PUVA treatment for 1–3 months can be added to antihistamine treatment.[27,28]

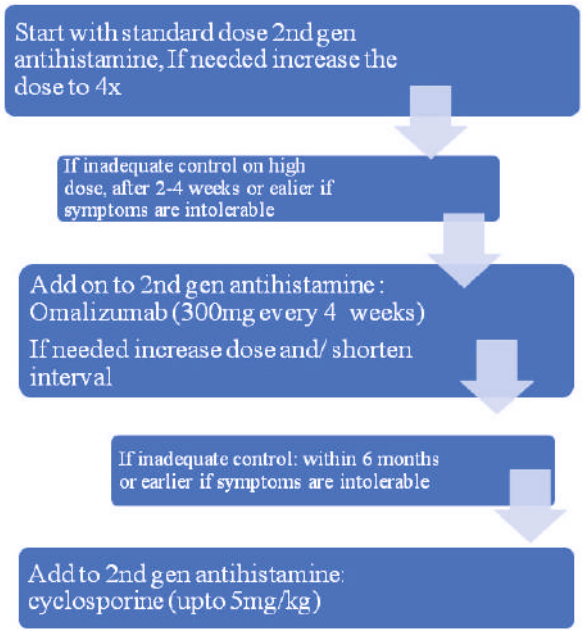

Fig 4: Algorithm for treatment of urticaria[1]

Treatment in special population

Children: The guidelines suggest using the same treatment algorithm with caution (eg, weight-adjusted dosage) in children with chronic urticaria. Second gen H_1-antihistamines with proven efficacy and safety in the pediatric population include bilastine, cetirizine, desloratadine, fexofenadine, levocetirizine, loratadine, and rupatadine.[29,30]

Pregnant and lactating women: Modern 2nd generation H_1-antihistamines is to be preferred such as loratadine with the possible extrapolation to desloratadine and cetirizine with a possible extrapolation to levocetirizine. 1st generation H_1-antihistamines should be avoided. Omalizumab can also be used in severe cases as per guidelines.[31,32]

Conclusion

Urticaria is a common dermatological condition causing significant morbidity in patients. Proper history taking, examination and evaluation is mandatory for management of patients. Pharmacological treatment of the patients should be done as per the guidelines.

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Score	Wheals	Pruritus
0	None	None
1	Mild (<20 wheals/ 24 h)	Mild (present but not annoying or troublesome)
2	Moderate (20–50 wheals/ 24 h)	Moderate (troublesome but does not interfere with normal daily activity or sleep)
3	Intense (>50 wheals/24 h or large confluent areas of wheals)	Intense (severe pruritus, which is sufficiently troublesome to interfere with normal daily activity or sleep)

Common Bacterial, Viral and Fungal Infections of Skin

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Skin infections encompass a wide range of pathological conditions ranging from superficial self limiting lesions to severe necrotizing and life threatening conditions.Bacterial ,viral and fungal infections form the bulk of commonly encountered cutaneous infections.

Bacterial infections

Cutaneous bacterial infections are one of the commonest clinical conditions encountered in dermatological practice .The bulk of cutaneous bacterial infections is attributed to the two gram positive bacteria: Staphylococcus aureus and Streptococcus pyogenes.The cutaneous infections caused by these two pyogenic bacterial strains are often referred to as pyodermas.The various types of cutaneous infections caused by these bacteria can be categorized as given in Table 1.[1,2]

Primary infections (by direct infection of unbroken skin)	
Follicular	Non follicular
Folliculitis	Impetigo
Furuncle	Ecthyma
Carbuncle	Erysipelas
	Cellulitis
	Abscess
Secondary infection of an underlying dermatosis like eczema ,scabies,miliaria,pediculosis ,etc	
Cutaneous disease due to bacterial toxin ▯ Staphylococcal scalded skin syndrome ▯ Toxic shock syndrome	

By far,primary pyodermas are the most common cutaneous bacterial infections observed in clinical practice.

Folliculitis

It is an acute inflammatory condition involving the pilosebaceous unit.Bacterial folliculitis is caused primarily by S.aureus, other bacterial pathogens implicated being Streptococcus, Pseudomonas and coliform bacteria.[3] Depending on the depth of involvement,folliculitis can be

- **Superficial** (Bockart's impetigo) in which the inflammation is confined to the follicular infundibulum.Classical clinical findings include inflamed follicular papules and pustules that usually develop in crops , common sites being scalp,face,upper trunk,buttocks and axillae.While pruritus is a commonly present,pain and tenderness are unusual.[2,4]
- **Deep folliculitis** (Sycosis) where inflammation involves the entire follicle and usually affects the beard area of the face and neck.It often presents with erythematous plaques studded with pustules (resembling a fig).The plaques are formed by coalescence of individual oedematous follicular papules and pustules known to recur at irregular intervals over years.[4]

Furunculosis

Furunculosis is a deep necrotic infection of the hair follicle and the perifollicular tissue almost always caused by S.aureus.It starts as a small perifollicular, erythematous, nodular swelling and eventually develops into a fluctuant mass that drains to leave a permanent scar.[5]Pain and tenderness are constant features.Fever and constitutional symptoms may also

be associated with a furuncle.Face neck,upper limbs and buttocks are commonly affected.

Carbuncle

In a carbuncle, the infection involves multiple, contiguous hair follicles. Essentially it is a collection of multiple interconnected furuncles extending into the surrounding tissue including the subcutaneous fat.It typically presents as an erythematous, tender, fluctuant mass with multiple draining sinus tracts or ulceration on the surface. Systemic symptoms and regional lymphadenopathy are common.[6] The lesions leave evident scars on healing.

Impetigo

Impetigo is a highly contagious,superficial cutaneous bacterial infection commonly seen in preschool children.In addition to direct contact,spread through fomites is quite common.[7]Two clinical forms are recognized:bullous impetigo and non bullous impetigo.Table 2 enumerates the clinical differences between these two types of impetigo.

	Non bullous impetigo (Impetigo contagiosa of Tilbury Fox)	Bullous impetigo
Occurrence	More common (nearly 3/4th of all cases	Less common
Distribution	Predominantly involves the face(perioral,perinatal areas) and limbs	Intertriginous areas (axillae,neck,diaper area)
Clinical appearance	Superficial erosions covered with characteristic honey coloured crusts	Vesiculobullous lesions persist for a few days & eventually rupture to form erosions with a shiny erythematous base
Number of lesions	Few	Multiple
Regional lymphadenopathy	Common	Uncommon
Systemic symptoms	Rare	Common

Ecthyma

This cutaneous bacterial infection extends into the dermis and may be considered a deeper form of impetigo. It presents as an erythematous plaque with overlying vesicles or pustules which rapidly rupture leading to formation of the characteristic adherent crusts which if removed gives rise to a punched out ulcer.The lesions are often multiple and commonly involve the lower limbs.

Erysipelas

Erysipelas is a β hemolytic streptococcal infection involving the dermis and superficial dermal lymphatics.Erysipelas affects all age groups but is more commonly seen in the extremes of age.[8] Any inflammatory dermatoses or traumatic insult to the skin can lead to erysipelas by serving as a portal for the entry of microbes. A sharply demarcated, erythematous, tender plaque with raised edges is typically seen in erysipelas. In more severe cases, vesicles , bullae or necrosis may be seen.

Cellulitis

Cellulitis is caused chiefly by β hemolytic streptococci though S.aureus is also implicated occasionally.The infection involves the dermis and the subcutaneous tissue thus extending deeper than in erysipelas.Diffuse erythema,edema and tenderness with poorly demarcated borders is characteristic of cellulitis. Constitutional symptoms are frequently present.Lower limbs are the most commonly affected sites and complications like necrosis and abscess formation are more common than in erysipelas.

Abscess

An abscess is a walled off collection of pus within the dermis and deeper cutaneous tissues.They are mostly caused by S.aureus of which a significant number is that of methicillin resistant Staphylococcus aureus (MRSA)[9]It presents as a painful, erythematous, tender, fluctuant mass and may be surrounded by erythema and induration.

Treatment of primary pyodermas

In folliculitis and impetigo, topical antibiotics form the treatment of choice in most cases with mild disease or limited skin lesions.The topical agents commonly used include mupirocin and fusidic acid.It is recommended to treat ecthyma, erysipelas and cellulitis with systemic antibiotics.[10]The systemic antibiotics commonly used in primary pyodermas include cotrimoxazole, tetracyclines, first generation cephalosporins, ciprofloxacin, clindamycin and linezolid.Furuncle, carbuncle and skin abscesses are managed by a similar approach.Incision and drainage forms the mainstay of treatment in these conditions which can be combined with topical or systemic antibiotics.

OTHER COMMON BACTERIAL INFECTIONS

Erythrasma

It is a superficial bacterial infection of the skin caused by Corynebacterium minutissimum.It has a predilection for the intertriginous regions like axillae,

groins and interdigital spaces of the toes.In intertriginous areas,it usually presents as a well defined reddish brown patch or plaque with a fine scale and wrinkled surface.In the interdigital spaces,the skin is often white and macerated.Lesions are usually asymptomatic but may be associated with pruritus.

Pitted keratolysis

It is a chronic, non inflammatory superficial bacterial infection of the skin caused by Kytococcus sedentarius,Dermatophilus congolensis and Corynebacterium species.The condition usually affects the soles especially in individuals who walk barefoot.Numerous small, shallow, circular, crateriform pits in the form of irregular erosions are seen over the plantar aspect of the foot.The lesions are usually asymptomatic.

Both erythrasma and pitted keratolysis respond to oral and topical erythromycin. Other treatment options include topical azole antifungals (miconazole, clotrimazole),mupirocin and fusidic acid.

VIRAL INFECTIONS

Viral infections of the skin are widespread, highly contagious and often recurring in nature. They can occur due to direct viral inoculation, spread from an internal focus or due to systemic infections. The commonly encountered cutaneous viral infections in clinical practice are discussed here.

Herpes simplex virus(HSV) infections

Herpes simplex virus infections commonly cause recurrent infections of the skin and mucous membranes.HSV-1 mostly leads to infections in the perioral and facial areas whereas HSV-2 is commonly responsible for the genital disease.Where most children tend to have antibodies to HSV-1 by 5 years of age, HSV-2 infections mainly occur after puberty.[11]Herpes virus infections may present as:

- Primary herpetic gingivostomatitis

This is a common clinical presentation of HSV -1 infection in children between 1-5 years of age though adults may also be affected.Mucosal lesions are preceded by a prodrome in most cases.Multiple, transient vesicles appear over the palate,buccal and labial mucosa that rupture to form painful superficial ulcers.Gingival inflammation is characteristic and lymphadenopathy is a usual association.

- Recurrent orofacial herpes

Recurrent episodes are usually milder ,of shorter duration and rarely associated with systemic symptoms.A prodrome of itching,burning and pain lasts for a few hours which is followed by the appearance of vesicles which form erosions and crusts.

- Genital herpes

Both HSV-1&2 are implicated in genital herpes. Primary genital herpes when symptomatic, begins with a prodrome which is followed by appearance of vesicles over the shaft and glans of penis in men and labia minora, introitus and urethral meatus in women. The vesicles rupture leading to painful superficial ulcers and erosions .

Non primary genital herpes where patients have pre existing antibodies against HSV-1; and recurrent genital herpes tend to be less severe with no systemic involvement.

Varicella

Varicella results from a primary infection with varicella zoster virus (VZV).Varicella is a highly contagious infection with a secondary household attack rate of >90% in susceptible individuals.[12] The classical presentation is that of a prodrome followed by development of generalized, pruritic, maculopapular and vesicular rash. The rash appears in successive crops over several days resulting in simultaneous existence of lesions at varying stages of development which is a characteristic feature of the skin rash in varicella. An enanthem involving the oropharyngeal or conjunctival mucosa may also be seen. Following varicella ,the VZV establishes latent infection in the trigeminal and dorsal root ganglia.

Herpes zoster

Herpes zoster is caused by endogenous reactivation of the latent VZV. Due to decline in the VZV specific cell mediated immunity, the incidence rates progressively increase with age.In addition, disease related or iatrogenic immunosuppression also pose a significant risk for the development of herpes zoster. The clinical manifestations include a vesicular rash (grouped vesicles) in dermatomal distribution (single or multiple contiguous dermatomes) associated with acute neuritis. Transmission to varicella naïve individuals through vesicular fluid or via airborne transmission is possible.[13]The vesicles become hemorrhagic and form crusts which fall off in 7-10 days.

Treatment of HHV infections includes systemic antiviral agents like acyclovir, famciclovir, valacyclovir and penciclovir. Additional supportive treatment measures are required for alleviation of pain and discomfort in oral lesions of herpetic gingivostomatitis while non steroidal anti inflammatory drugs, gabapentinoids and tricyclic anti depressants play a role in the management of neuritic pain in herpes zoster.

Pityriasis rosea :

It is a acute,self limited papulosquamous cutaneous disease hypothesized to be a manifestation of HHV-6 & HHV-7 reactivation. Commonly seen in older children and young adults, the eruption begins with a 'herald' patch which is a single, round or oval scaly lesion often found on the neck, chest or back.This is followed few days later by numerous, symmetrical ,scaly oval patches on the trunk and proximal extremities, the long axes of which tend to be oriented along the lines of cleavage of the skin. This gives rise to the characteristic 'fir tree' or 'Christmas tree' appearance. A collarette of scales is seen along the edges of individual lesions. Treatment includes antihistamines, topical steroids and emollients. Oral acyclovir and macrolides may lead to faster resolution of lesions[14]

Warts

These are the most prevalent mucocutaneous lesions resulting from human papilloma virus (HPV) infection of the skin.Table 3 gives an overview of the various types of warts commonly caused by HPV.

Type of wart	HPV type	Clinical features	Common sites
Verruca vulgaris	HPV 2,27,57	Single/multiple papulonodules with a rough surface, asymptomatic	Back of hands,fingers
Plantar warts	HPV 1,2	Superficial(mosaic warts) or deep (myrmecia) Usually painful	Pressure points of plantar skin
Verruca plana	HPV 3,10	Small,flat,rounded or polygonal skin coloured to hyperpigmented with smooth surface	Face,back of hands
Filiform warts	HPV 2	Pedunculated/speculated lesions growing perpendicular or oblique to the skin surface	Face and neck
Condyloma acuminata (Anogenital warts)	HPV 6,11	Papulonodules or soft filiform growths,hypopigmented/erythematous /hyperpigmented	Mucosal aspect of genitalia,anal/perianal skin

Treatment options include destructive modalities in the form of topical salicylic acid, cantharidin, caustics(trichloracetic acid, phenol, silver nitrate), cryotherapy, radiofrequency and laser ablation; antimitotics like podophyllin,podophyllotoxin , bleomycin and 5-fluorouracil; immunotherapy and surgery.

Molluscum contagiosum

It is a benign viral infection of the skin caused by molluscum contagiosum virus(genus molluscipox). Molluscum contagiosum is a common disorder of childhood although healthy adolescents and adults may also be affected. Characteristic skin lesions are firm, dome shaped, rounded, pink waxy papules with central umbilication. Spontaneous resolution is common in healthy individuals.

FUNGAL INFECTIONS

Fungal infections of the skin can be classified into superficial ,subcutaneous and deep mycoses among which superficial fungal infections are the ones commonly encountered.Superficial fungal infections include:

Dermatophytosis

These are the superficial mycoses caused by dermatophytes which are filamentous fungi that feed on keratinized tissue thus infecting the skin ,hair and nails.The three genera responsible for human disease are Microsporum,Trichophyton and Epidermophyton.Depending on the site of involvement,dermatophytoses are classified into various types:

- Tinea corporis : Dermatophyte infection of the glabrous skin excluding the palms,soles and groins
- Tinea cruris (groins,perianal and perineal areas)
- Tinea pedis (feet or toes)
- Tinea manuum (hands)
- Tinea faciei (facial skin)
- Tinea barbae (beard and moustache area in males
- Tinea capitis (scalp and hair)
- Tinea unguium (nails)

Characteristic lesions include annular plaques with an erythematous scaly border and central clearing.Vesicles and pustules may be seen at the border in cases with severe inflammation.The morphology of lesions however varies according to the site of involvement.

Treatment of dermatophytosis

Similar topical and systemic antifungals are used for the treatment of different forms of dermatophytosis. However the treatment regimes vary depending on the site involved. Topical antifungals used include azole (clotrimazole, miconazole, sertaconazole, etc.) ,allylamines (terbinafine), ciclopirox and amorolfine.Systemic antifungals commonly used include terbinafine, itraconazole, fluconazole and

griseofulvin.

Candidiasis

This includes the superficial fungal infections caused by the yeasts of the genus Candida.The common candidal infections of the skin and mucous membranes include oral candidiasis,vulvovaginal candidiasis,candidal intertrigo and candidal paronychia.

Pityriasis versicolor

It is a common superficial fungal infection of the skin caused by Malasezzia that typically presents as hypopigmented, hyperpigmented or erythematous coalescing macules with characteristic fine brawny surface scales.Upper trunk and proximal upper extremities are most commonly affected whereas facial involvement is common in children[16]Patients are usually asymptomatic.Treatment includes topical agents like selenium sulfide,ketoconazole and terbinafine and systemic azole antifungals.

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Legend to the tables

Table 1. Classification of cutaneous bacterial infections

Table 2: Clinical differences between bullous and non bullous impetigo

Table 3: Description of various types of warts caused by human papilloma viruses

Chemical-Induced Vitiligo: What We Need to Know

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The uneven distribution of skin color has a deep and lasting impact on our society's psychological and social well-being. Indian dermatologists often receive patients with depigmented skin lesions who are extremely worried. For over 75 years, chemical-induced depigmentation or vitiligo of the skin has been documented. Initially, it was considered an occupational menace, but it now includes those exposed to common household and commercial products. As in developing countries like India, the number of patients with chemical-induced vitiligo has been increasing in recent years and this skin disease resembles vitiligo closely causing a psycho-social reaction, hence, the clinico-etiological diagnosis of this skin disorder is very much essential.

What do we mean by chemical vitiligo?

Chemical vitiligo denotes an acquired hypo- or depigmentation usually induced by repeated exposure to specific chemical compounds. These chemicals are thought to be toxic for melanocytes, particularly in patients having specific genetic susceptibility to vitiligo. This condition is separate from post-inflammatory depigmentation and koebnerisation in vitiligo and is also independent of the sensitizing potential of the implicated chemical.

Contact leukoderma/occupational leukoderma/ chemical leukoderma/ chemical-induced vitiligo: what's in a name?

The above terminologies have often been used interchangeably to describe the condition. Chemical leukoderma or the more recent terminology chemical-induced vitiligo is a rational and justified term to describe the disease.

Meanwhile chemical-induced vitiligo and non-chemically-induced vitiligo are on the same spectrum clinically, histologically, and pathogenically, chemical-induced depigmentation better is called “chemical-induced vitiligo”, rather than other names that have been used, such as occupational vitiligo/leukoderma, and contact vitiligo/leukoderma/depigmentation.

As the depigmentation is not precisely confined only to the site of contact and pathogenetically the condition is not similar to contact dermatitis, the term ''Contact Leukoderma' 'is confusing. Furthermore, the majority of the cases are induced by non-occupational household objects, the term “Occupational leukoderma'' is misleading.

Historical aspects:

In 1939, Oliver et al. first reported chemical leukoderma in workers using 'acid-cured' rubber gloves in a leather manufacturing company, where mono benzyl ether of hydroquinone (MBEH), an antioxidant used in the rubber industry was the offending agent. Since then, several reports of occupational leukoderma caused by *phenolic compounds* were published in different countries. Chemical leukoderma was first reported in India in 1985 by Pandhi and Kumar originating from adhesive *'bindi'& footwear.*

What are the common etiological factors?

Common occupational etiological factors include rubber gloves, lubricating and motor oils, detergents, printing inks, and chemical laboratory agents. Chemicals in the phenol and catechol families are the most widely documented causative agents.

On the other hand, several household objects may also have causal roles in chemical-induced vitiligo. e.g. hair

dye, toothpaste, black socks and shoes (containing PPD), deodorant, spray perfume (containing PTBP), detergent, watch straps, cleansers (PTBP), Insecticides (PTBP), adhesive 'bindi' (PTBP), rubber sandals (MBEH), condoms, synthetic leather wallets, rubber gloves, eyeliner, lipliner, lipstick, 'alta' (Azo dyes), fur toys, amulet string color (Azo dyes/congo red/ PPD), etc.

How it occurs?

Several hypotheses do exist. Some of them are as follows:

- Offending chemicals may directly exert their melano-cytotoxic effect independent of their sensitizing potential by cell-mediated immunity
- Chemical-induced oxidative stress in melanocytes initiates an autoimmune response, leading to their destruction. Subsequently, this immune response can affect other melanocytes in distant regions that are not directly in contact with the chemical.
- Most chemicals that induce vitiligo are phenols and act as tyrosine analogues to disrupt melanocyte function, resulting in autoimmunity.
- The condition may sometimes be associated with a genetic background of vitiligo.
- Chemical-induced vitiligo probably represents an environmental disease rather than a genetic one.
- The activation of several innate immune cells, the release of cytokines and chemokines, that recruit autoreactive cytotoxic T-cells, ultimately lead to T-cell-mediated autoimmune destruction of melanocytes.
- At the molecular level the offending agent may act by inhibiting tyrosinase transcriptions & glycosylation, inhibiting enzymes such as tyrosinase or peroxidase, reducing agent or radical oxygen scavenger, tyrosinase degradation, and inhibiting melanosome transfer.

What are the stages of the disease?

Several stages of chemical–induced vitiligo or chemical leukoderma syndrome have been described. They are enumerated below.

Stage I: Chemical leukoderma only at the site of contact

Stage II: Local spread of chemical leukoderma through the lymphatics beyond the site of contact

Stage IIIA: Distant spread of chemical leukoderma through a hematogenous route beyond the site of contact

Stage IIIB: Distant spread of chemical leukoderma through a hematogenous route beyond the site of contact along with systemic organ involvement

Stage IIIC: A systemic introduction (injection, inhalation, or ingestion) other than skin contact causing chemical leukoderma with or without systemic organ involvement

Stage IV: Distant spread of vitiligo-like patches even after 1 year of strictly withholding exposure to offending chemicals.

Who is affected by this disease?

Patients of all age groups (from pediatric to geriatric) from both sexes are affected. However, there is a female predominance of the disease.

What are the common sites affected?

The skin depigmentation may be localized or diffuse. The face is the most common site affected. A high incidence of the disease was also reported over the hands and feet. The scalp is usually the least affected site.

What are the causes of the spread of the disease?

Several factors are responsible for the spread of the disease including auto-transfer, hetero-transfer, contact at multiple sites, and doubtful lymphatic spread.

How can you diagnose the disease?

The diagnosis is essentially clinical. A detailed history and thorough clinical examination may clinch the diagnosis. The presence of i) acquired vitiligo-like depigmented lesion(s), ii) history of repeated exposure to specific chemical compounds, iii) patterned vitiligo-like macules conforming to the site of exposure, and iv) confetti macules favor the diagnosis of chemical-induced vitiligo. Any three of the above four criteria should be present to diagnose a case of chemical leukoderma clinically.Pruritus may sometimes be present in chemical leukoderma.

The role of patch tests in the diagnosis of chemical vitiligo is controversial. This is because there is a lack of standardization and agreement about patch testing in the diagnosis of chemical vitiligo, medico-legal issues, ethical issues, and psychological grounds. Moreover, as patch tests may also give rise to new lesions, consent for patch testing may not be available in the majority of cases.

Histopathology of the lesional skin reveals an absence of melanocytes. It cannot differentiate the condition from idiopathic vitiligo. Electron microscopy of chemical-induced vitiligo may express swollen mitochondria.

What are the similar-looking conditions of chemical-induced vitiligo?

Any cause of congenital or acquired hypo-/depigmentation may simulate chemical-induced vitiligo e.g. idiopathic vitiligo, post-inflammatory depigmentation, and koebnerisation in vitiligo among others.

How to differentiate chemical leukoderma and idiopathic vitiligo?

Pea-sized or confetti macules will be usually absent in vitiligo but present in the chemical leukoderma. Also, features like trichrome, leukotrichia, and Koebner will be present in idiopathic vitiligo and usually absent in chemical leukoderma.

Is it associated with any systemic symptoms?

In most of the patients, there is no systemic ailment. Thyroid disease, hepatosplenomegaly, and abnormal liver function tests have rarely been reported in association with chemical-induced vitiligo.

Chemical vitiligo in India vies a vies in the Western world – are they similar?

Chemical vitiligo has been reported mostly from industrial exposure and reported generally in adults in Western countries. On the other hand in India, it has been reported mostly from household objects used by pediatric to geriatric age groups.

Why there are discrepancies between Western and Indian data regarding chemical–induced vitiligo?

Lack of quality control and ethical consideration during consumer product manufacturing in India, lack of reporting from industrial sectors, and comparative lack of industrialization are the main reasons for the discrepancies.

What is the overall prognosis of this condition?

Its prognosis is better than vitiligo. Early cases show good response after the cause is withdrawn. After repigmentation, recurrence is rare. Pure chemical-induced vitiligo patients respond better to treatment than chemical vitiligo arising in patients with pre-existing vitiligo.

Is it treatable?

Yes; however, the most important thing is the strict & permanent removal of causes. Currently, there is no universally accepted treatment protocol for chemical leukoderma. However, a topical steroid is usually the first preferred treatment in localized chemical vitiligo. Tacrolimus may also be used. Also, in extensive chemical vitiligo, psoralen, ultraviolet A, narrowband ultraviolet B phototherapy, 308 nm excimer laser therapy, low dose systemic steroid pulse, cyclosporine, other immunosuppressive agents, and combination therapy may be used, depending on disease activity and site.

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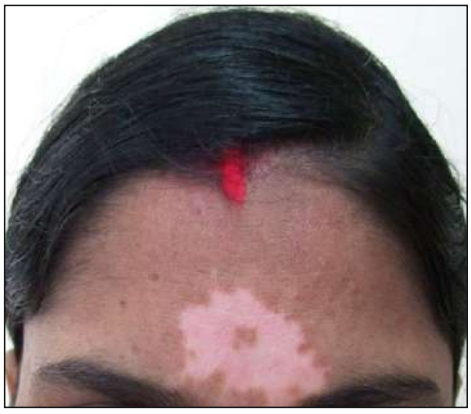

Figure 1: Bindi- induced chemical vitiligo

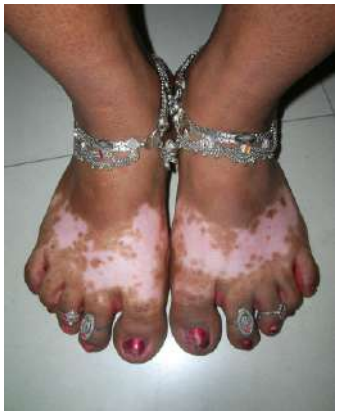

Figure 2: Foot-wear-induced chemical vitiligo

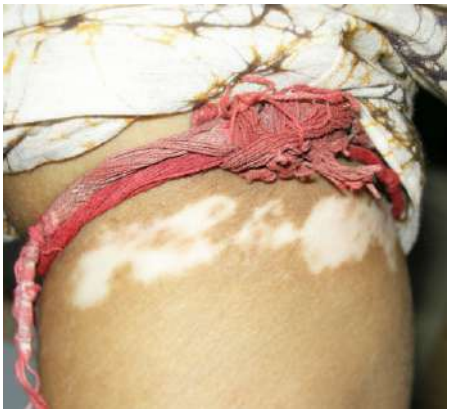

Figure 3: Colored string-induced chemical vitiligo

Challenges of Generic Drug Usage in Dermatology

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Abstract

A generic drug is defined as a medication created to be the same as an already marketed brand name drug in dosage form, safety, strength, route of administration, quality, performance characteristics and intended use. The issue of generic drugs recently came into the fore following a National Medical Commission directive (since kept in abeyance) to make prescribing drugs in generic names a mandatory practice in India. The lack of preparedness on the part of the Government may be understood by the fact that there is simply no definition of a 'generic drug' in the Drugs and Cosmetic Act and Rules. Thus, currently any pharma marketing generics in India can do that with hardly any checks and balances. However, generic substitution of drugs is debated in dermatology even in highly controlled markets, such as the United States, where standards of bioequivalence and therapeutic equivalence are stringently applied. This is due to the associated bioequivalence issues in topical drugs. Several variables like drug and vehicle attributes, type of skin and mode of application may make controlled studies in bioequivalence between different formulations extremely difficult, if not impossible. Another consideration with topical drugs as opposed to oral drugs is irritancy potential and complexity of skin allergies. So, in the conceivable future, even if the Indian drug market attains enough maturity to ensure meaningful regulation of generic drugs, dermatologists will always find it tough to balance concerns of quality on one hand and that of rational drug therapy and cost effectiveness on the other.

Keywords: Generic drugs, bioequivalence, irritancy, vehicle

Take home messages

- A generic drug is defined as a medication created to be the same as an already marketed brand name drug in dosage form, safety, strength, route of administration, quality, performance characteristics and intended use.
- Ideally, a generic drug must establish bioequivalence and therapeutic equivalence that are not significantly different from the reference (innovator) drug.
- Some argue that it is almost impossible to determine bioequivalence of topical formulations through well-controlled studies due to variables like drug and vehicle attributes, type of skin and mode of application.
- There are several studies that show the association between use of generic-only prescriptions and rational therapy and driving down cost of drugs.

Introduction

The Food and Drugs Administration (FDA) of the United States (US) defines a generic drug as a "medication created to be the same as an already marketed brand-name drug in dosage form, safety, strength, route of administration, quality, performance characteristics, and intended use."[1]Going by this definition, a generic drug would aim to match the branded product in terms of active ingredient and dosage form by bioequivalence to the Reference (Innovator/Branded) drug. This seems pretty straightforward for oral medications.[2] However, there are several limitations of bioequivalence studies

which we shall come to later.

The issue of generic drugs came to the fore in this country following a National Medical Commission directive (since kept in abeyance) to make prescribing drugs in generic names a mandatory practice in India. The lack of preparedness on the part of the Government may be understood by the fact that there is simply no definition of a 'generic drug' in the Drugs and Cosmetic Act and Rules. Thus, currently any pharma entity marketing generics in India can do that with hardly any checks and balances.

The case for generics

Why is the worldwide clamour for generics these days? With the ever-increasing cost of medical care, the issue of drug pricing has attracted headlines. There is enough evidence that greater use of generic drugs leads to increased competition among the pharmaceutical producers, thus driving down the price of medicines. The negative association between the increase in drug price and the median number of manufacturers of generic topical dermatologic drugs indicates a role for market competition in controlling the costs of generic drug prices within dermatology as well.[3] Conversely, increased usage of brand-name drugs leading to medical cost escalation has also been amply demonstrated.[4]

There is also enough evidence that a low usage of generic drugs in prescriptions is associated with irrational therapy.[5] Rational therapy is described as use of the right drug at the right dose by the right route at the right time for the right patient.[6] Irrational therapy among dermatologists in India is also a phenomenon that is well-documented.[7] In different studies on dermatological prescriptions carried out in India in different settings, the proportion of generic-only prescriptions has varied from zero to 42.91%.[5,8-12]

Why generic-only prescription policy may be inappropriate for dermatology

In spite of such compelling logic in favour of generic prescribing, there has been genuine concern that such a policy will be impractical, particularly in a field like dermatology. Such concerns are beyond the ones regarding absence of quality assurance in our country, which are, of course, legitimate. We are concerned more about the strictly scientific aspects of the limitations of generic formulations in dermatology.

In order to understand these limitations, we have to grasp the way generic formulations are tested in a well-regulated market, such as the United States. In the US, the bioequivalence protocol requires that the test product (generic) does not differ significantly from the reference (innovator) drug. The level of significance is defined as 20%. That translates to a 45% variability, meaning that a generic can vary 45% (almost half) from the innovator brand or from another generic.[13]

Also, this 'sameness' only pertains to the active ingredient. Inactive ingredients added to a drug to help with binding, flavouring, colouring, preserving or with drug transport need not be the same.[14] Although these compounds are usually ones that have been used in other approved drugs with identical routes of administration in the same amount and daily exposure, and for which data exist indicating that use of these different compounds will not alter a product's safety and effectiveness, their unique combination in a particular drug is apt to affect that product's overall pharamacokinetics.[15]

There are also other factors that are not included in establishing bioequivalence. For example, genetic polymorphism in drug metabolizing enzymes, which could potentially lead to significant variations in drug metabolism, and enteric coating son generic drugs, which may impact the rate and extent of drug absorption, are not evaluated inroutine studies of bioequivalence.[16]

There are other limitations with the reliance on bioequivalence studies. These studies are generally two-treatment crossover studies involving a small number of patients, usually ranging from 24to 36 patients but with an FDA-recommended minimum of 12 patients in the US.[17] The study participants are a homogeneous group of healthy18- to 55-year-old adult male volunteers of normal height and weight who do not take any concurrent medications, do not smoke and receive a controlleddiet.[18] While the use of normal, healthy subjects reduces the probability that any bio in equivalence could be related to changes in the disease process over time rather than differences in drug formulation, it is important to realize that the drug pharmacokinetic properties in healthy individuals may not accurately predict the pharmacokinetic properties in patient subgroups because of patient variables including disease comorbidities, concurrent medications, differences in first-pass metabolism, varied diets, gastrointestinal variations (i.e., gastric pH, blood flow, and bacterial flora) and even placebo and nocebo responses.[19] This risk is greater in the elderly population, in which age-related physiologic changes that affect drug absorption, metabolism, and excretion may exist, but could be missed in younger, healthier populations.[20] It is important to realize that these concerns are further

multiplied in a predominantly chronic care speciality such as dermatology.

Bioequivalence studies are usually performed using single doses of drugs because single-dose studies are generally more sensitive at measuring the release of the active metabolite from a drug product into the circulation than multidosestudies.[21] The maintenance of a steady state in the circulation and consequent therapeutic benefit, however, typically requires higher drug concentrations than can be achieved through single dosing.[22]

The issues with bioequivalence take on more complex dimensions when we come to topicals. First, drug attributes such as solubility in the vehicle, size, charge and membrane permeability may affect bioavailability. Second, vehicle attributes like drug solubility and dissolution rate and spreading ability may have an effect. Third, the type of skin to which the drug is applied and method of application can also make a difference. All of these variables make it difficult to compare the bioequivalence of two different drugs in a controlledmanner.[23] Another consideration with topical drugs as opposed to oral drugs is skin irritation. While the generic version of a topical drug might be just as effective as its brand-name counterpart, it may be much more irritating to skin for some patients.[24]

Thus, the nature of dermatological drugs warrants caution when substituting brand-name drugs with the generic equivalents. For topical corticosteroid preparations, research has shown the superiority of brand-name for cream, ointment and lotion vehicles over similarly formulated generic equivalents.[25] Few case reports indicated the adverse events inpatients who were switched to generic equivalents for their topical medication therapy.[26] Thesereports necessitate vigilance on the part of dermatologists while performing generic drug substitutions for dermatological products.

Topical treatments, which are essential approaches to skin diseases, are associated with poor adherence for all the reasons stated above. Thus, dermatology requires personalized treatment protocols perhaps more than any other branch of clinical medicine. Topical vehicles have the primary purpose of ensuring drug effectiveness (by modulating drug stability and delivery, as well as skin properties) but have a marked impact on treatment outcomes as they influence patient satisfaction and, consequently, adherence to topical treatments. There is also a wide variety of vehicles available for topical formulations, which can complicate the decisions of clinicians regarding the most appropriate treatments for specific skin disorders. Pharmaceutical technologists have developed patient-centric drug-product design as a possible strategy to improve topical treatment adherence.[27] This is why, it is almost impossible to substitute one topical formulation for any other with the same active ingredient in a particular case – the so-called inactive ingredients have a lot to contribute to patient perception and, even, effectiveness.

Conclusion

Topical therapy remains a cornerstone of dermatological care. Topical formulations are all unique as there can be a large degree of variability in terms of bioequivalence, quality of excipients, therapeutic effectiveness, penetrability, optimal dosing frequency, irritancy potential of vehicles, etc. Thus, substituting one topical formulation for another may not be possible rationally on a case-to-case basis. In this scenario, a move towards generic-only prescription may be too premature in our country, which still lacks an official definition of 'generic' drugs.

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Skin care of the newborn

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Introduction : Skin is the largest organ of the body which acts as a protective barrier against thermal, mechanical and physical substances. The process of birth marks transition of a child from safe, wet, sterile uterus to a dry, exposed infectious environment. Moreover, skin of a newborn is structurally and functionally different from that of adult skin (Table 1).

Acclimatization,functional maturation and development of the skin takes place gradually in infancy and childhood. The immature newborn skin needs proper handling and immaculate skincare to withstand this environmental change. Gentle and correct skin care practiceswill support the continuing maturation and prevent secondary infections. There are varying traditional newborn skincare practices across different cultures and religions widely prevalent in our country. Moreover, the market is also presently flooding with a wide range of baby skin care products. Ignorance and incorrect usage often impede the normal process of skin development and can also trigger dermatological conditions like xerosis, atopic eczemas, diaper dermatitis, seborrheic dermatitis. Hence, standardisation of newborn skin care and parent education assumes prime importance in this arena. This article aims to review optimum newborn skin care, choosing the appropriate skin care product with special consideration to skin care in atopic children.

General principles of skin care in healthy, term neonate:

The skin care in a newborn is primarily aimed at decreasing the trans epidermal water loss, using hypoallergenic non-irritating skin care products, minimising infections, protection against physical injuries and enhancing physiological maturation of skin.

Immediately after birth, neonate skin and hair should be wiped with a dry, clean and warm cloth. If blood or meconium is present on baby's skin, it should be gently wiped away with a wet cloth. Direct skin to skin contact between mother and child should be immediately allowed for at least one hour to prevent hypothermia [1]. If the mother is not able to do so, the baby should be properly covered in a warm, soft and dry cloth to prevent heat loss. The room temperature should be around 25-28 degrees.

Role of vernix caseosa: Vernix caseosa acts as natural biofilm composed of hydrated corneocytes in a lamellar lipid matrix [2]. It serves as a protective mantle providing innate immunity, thermal regulation, lower surface pH and defence. As per WHO, it should not be removed from the baby's skin as it plays a

Skin properties	Pre term	Term	Adult	Clinical relevance
Epidermis diameter(micron)	20-25 IL-1α ↑↑ Antimicrobial peptides and natural moisturising factor (NMF) ↓↓ Immature lipid barrier	40-50 IL-1α ↑ Antimicrobial peptides ↓ NMF ↓	>50	Increase in trans epidermal water loss (TEWL), susceptibility to infections, penetration of allergen and topical lotions/oils
Dermo epidermal junction	Flat	Rete ridges begin to form	Deep rete ridges	Prone to abrasion and shear injuries
Skin surface pH	6.2-7.5	6.2-7.5	5.0-5.5	Activation of proteases, bacterial colonisation
Thermoregulation	Unable to sweat	Higher density of sweat glands but increase threshold for eccrine sweating	Normal density and normal sweating	Increase risk of evaporative heat loss
Hair	Lanugo hair	Vellus hairs	Vellus and terminal hairs	

Table 1: Difference between newborn and adult skin and its clinical relevance - Skin properties Pre term Term Adult Clinical relevance

crucial role in adapting to extrauterine environment. It should be allowed to dry and peel of naturally. In one study, vernix retention provided greater skin hydration and lower skin pH suggesting that it does help in forming acid mantle of skin [3]. Only in conditions like maternal HIV and Hepatitis B, early removal should be done to prevent disease transmission via mother's blood.

Bathing : Bathing can lead to hypothermia, increased oxygen requirements and an increased risk of intraventricular haemorrhage. Hence, a baby should be first bathed only when its cardiorespiratory parameters has been stable for six hours. WHO recommends delaying the first birth to 24 hours and not earlier than six hours as this would promote maternal bonding via breastfeeding and skin to skin contact. In one study however it was found that timing of bath did not have any significant impact on temperature[4].Immersion tub bathing is preferred over sponge bath as it facilitates tactile bonding and improves thermoregulation[5]. The frequency of bath should be two to three times per week. It should be provided in a warm room and the water temperature should be around 37 degrees. It should be checked properly by the caregiver with their hand. The bath time should be limited to five minutes to limit skin hyperhydration and fragility. They should be dried thoroughly and wrapped from head to toe.Following bath, emollient application to slightly damp skin is advised to maintain skin hydration. Petroleum jelly is an inexpensive allergen free emollient ideal for daily use in neonates. In our culture, the use of natural oils like sunflower, mustard, coconut and olive oils is quite widespread for massage and as moisturisers. Mustard oil and olive oil are not recommended as they impede skin barrier recovery [6]. Mustard oil has an allergen called allyl isothiocyanate which can lead to contact dermatitis. Olive oil may also exacerbate seborrheic dermatitis by promoting growth of Malassezia species. Sunflower oil has a high linolenic acid concentration and speeds up skin repair and decreases TEWL. When applied to preterm infants, it showed decrease in mortality rates and nosocomial infections [7]. Virgin coconut oil and mineral oilsincreases skin surface lipids and hydration. Parents should be educated to use oils cautiously in summer months to prevent folliculitis and miliaria.

Massage: Massage is defined as rhythmic application of touch. It is a deep-rooted traditional practice in our country which promotes blood circulation, neonatal growth and relaxes the body. It may be given by parents, grandparents or the nurse with clean hands using oil in a warm room [8]. The strokes should be firm yet gentle enough for the infant. It can be given for 15 to 30 minutes preferably a couple of hours after the feed when the child is alert. Ideally, it should be given before bath in summers and after bath in winters. One study showed that mothers who gave massage to preterm infants also had significant mood improvement thus offering dual benefit to baby as well as mother [9]. Another study reported significant rapid acid mantle development when babies were massaged regularly with sunflower oil compared to mustard oil [10]. Overall, oil massage offers warmth, nutrition, decreased TEWL, improved hydration, bone mineralisation, thermoregulation, sleep rhythm and neuromotor development [11].

Care of umbilical cord : The umbilical cord dries and falls off within five to ten days after birth. The cord should be cleaned with lukewarm water and dried. Mild soap can be used to clean soiled stump. As per WHO, no other topical agentshould be applied to cord stump [12]. The caretaker's hands must be clean.

Care of diaper area : The diaper area is a warm, moist environment continuously exposed to faeces, urine, friction and microbes contributing to infections, dermatitis and pain. It is extremely important to use superabsorbent diapers and frequently change them to prevent any maceration and damage of skin barrier[13]. Cloth napkins if being used should be washed in warm water and dried in sunlight.Moist soft cloth or cotton ball soaked in water should be used to clean the area from front to back and then it should be pat dried. Fragrance, detergent and alcohol-free mild wipes can also be used alternatively. Diaper free periods should be ensured and barrier creams containing zinc oxide or petrolatum jelly can be applied to prevent diaper dermatitis.

Care of scalp and nails : First head wash should be given after the umbilical cord has dried off and fallen[14]. If the child has cradle cap, application of mineral oil can be done to soften the scales. Crusts can be removed after two to three hours. Tear free mild shampoos can be used once weekly for scalp health. Nails should be trimmed and kept clean.

Skin care in Preterm Child : The skin of a premature infant is prone to trauma, trans epidermal water loss, percutaneous toxicity and infections. Immediately after birth, babies should be placed in a humidification incubator with ambient humidity of >80%. Humidity increases breakdown of filaggrin to release natural moisturizing factors that in turn improve skin hydration [15]. Humidity is reduced in a stepwise manner after one week to decrease chance of infection. Strict aseptic handling of the neonate is advised.

Immersion tub bathing should be given to healthy late

preterm infants between gestational age 34 to 36 weeks [16]. The water should cover the entire body to prevent heat loss. In neonates having gestational age between 30-36 weeks of gestation in NICU, swaddled bathing was found to be effective in maintaining thermal regulation and vital parameters [17].

Preterm neonates have an increased body surface area compared to weight and hence are at high if percutaneous toxicities. Care should be taken not to apply any strong astringents and alcohol-based antiseptics. Chlorhexidine is a relatively safer alternative for handling neonates. These newborns are also susceptible to iatrogenic cutaneous injuries by ventilation equipment, electrodes, dressings, disinfectants and cannulas. Epidermal peeling can occur after removal of adhesive dressings. Gentle soft silicon and hydrogel-basedadhesives should be used to secure the cannulas and skin should be softened with paraffin before their removal [18]. The newborn's position should be frequently changed to prevent pressure sores. Polyurethane can be used as wound dressings. Nasal prongs and electrode positions should be changed to prevent nasal injuries and skin burns respectively.Mild emollient application is advised to prevent high levels of trans epidermal water loss in these neonates.

Choosing the right skin care product : The market is currently flooded with baby care products. They claim to be safe, however they do have ingredients that be damaging to a baby's skin. Leave on products have an increased risk of percutaneous absorption, irritation and contact sensitization leading to systemic toxicity and contact dermatitis. Educating the parents and helping them choose the right skin care product is hence very crucial.

Cleansers and shampoos : Cleansers have surfactants that emulsify the lipids on skin surface and increase skin permeability. Soaps on the other hand leads to alkalization of skin surface disturbing the acid mantle. An ideal cleanser for the child should be soap less liquid, non-irritant, fragrance and dye free with a neutral or acidic pH and minimal preservatives. The same body cleanser can be used for scalp and hair. Tear free formulations of shampoo are available with very mild surfactant complexes to attain desirable pH. Shampoos containing ketoconazole, selenium sulphide and zinc pyrithione are helpful in cleaning the crusts in seborrheic capitis. Minimal contact time should be ensured to prevent contact dermatitis.

Emollients : Gentle application of a suitable emollient can be helpful in maintaining skin hydration. Emollients can be hydrocarbons (Vaseline, paraffin), fatty substances (cetyl alcohol and steryl alcohol), waxes (bees wax, lanolin) and oils (mineral oil, vegetable oil, synthetic oils) [14]. Oils have been used in our tradition since many years for massages and moisturisation. Coconut oil and sunflower oils are preferred oils but should be used cautiously in hot weather. Creamy moisturisers are preferred over thick ointments to prevent sweat gland occlusion. Petrolatum based, water-miscible, fragrant, dye and preservative free emollients can be used in preterm babies. Use of lanolin and detergents like sodium laurylsulfate is best avoided [8].

Talcum powders : Their use is controversial. Talcum powder use should be best restricted in new born period to decrease the risk of inhalation. Over application can lead to miliaria. In excessive hot and humid climates, the mother can be directed to apply powder gently with her hands in body folds. Authors strongly discourage the use of talcum powder.

Special considerations – Atopic dermatitis : Atopic dermatitis is a chronic, relapsing inflammatory dermatosis affecting 1 in 5 children globally [19]. The pathogenesis of atopic dermatitis encompasses a genetic background, environmental triggers and epidermal barrier dysfunction. These patients have a familial background of atopy. Moreover, there are mutations in gene encoding structural and functional proteins of epidermis and also proteins that modulate immunity. Filaggrin is an important protein in the epidermis that helps in formation of cornified envelope and also breaks down to release natural moisturising factors. These individuals have filaggrin gene mutations leading to skin dryness, increased water loss, high skin pH and am imbalance in levels of ceramides and free fatty acids [20]. The is overt activity of epidermal proteases and release of proinflammatory cytokines leading to inflammation and eczema.

Atopic dermatitis usually presents at 2 months of age. The role of prophylactic barrier therapy in asymptomatic neonates via emollient application is still debatable. 5-year findings from a Barrier Enhancement for Eczema Prevention trial showed that daily emollient application does not prevent atopic dermatitis [21]. Long term emollient application in asymptomatic neonates also carries a risk of sensitization. However, another study suggested that daily application of emollient for 32 weeks reduced the risk of eczema in infants [22]. They also found that sensitisation was associated with eczematous skin and not moisturiser usage. Authors strongly believe emollient intervention is a potential way to prevent atopic dermatitis in high-risk baby.

Topical emollients are the mainstay of therapy in

symptomatic infants with atopic dermatitis. They should be liberally applied within minutes of bath to slightly damp skin. Reapplication is advised every 4 to 6 hours to prevent dryness. Moisturisers help to control active flares, decrease severity and increase the time of relapses [23]. Moisturisers not only prolong remission but also act as steroid sparing agent and reduce requirement of systemic immunosuppressives too in long run. In country like our simple coconut oil or sunflower oil will do over expensive moisturizer. Even urea, glycerol containing conventional moisturisers can repair damaged skin barrier. Choice of expensive ceramide containing moisturiser depends on multiple factors like severity of the disease and cost of therapy.

Conclusion: Newborn skin care should be thoughtful and intricate keeping in mind the ongoing physiological maturation of skin after birth. All skin care products must be used with caution with due consideration to the gestational age and coexisting skin conditions. Evidence based guidelines for neonatal skin care are still few and need further validation. Parental counselling by dermatologists and paediatricians assumes utmost importance for proper newborn skin care.

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Treatment of Nevus Spilus over the Left Cheek using Q-Switched Nd-YAG Laser: A Case Report

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Abstract :

Here we present a case of nevus spilus treated with QS ND YAG laser for 3 sessions with significant improvement. Laser sessions were office procedure, under topical anaesthesia, uneventful and without any side effects.

Introduction:

Nevus spilus is a form of pigmented birthmark that appears as a brown patch containing darker specks or blotches. Although it is typically benign, some patients may desire its removal for cosmetic purposes. It has been demonstrated that Q-switched Nd-YAG laser is an effective option for therapy for nevus spilus.(1)(2)

Case Presentation:

A 28-year-old female presented to our clinic with a nevus spilus on her left cheek that had been present since birth. The patient was self-conscious about the appearance of the birthmark and wished to have it removed. After a thorough evaluation, the patient was deemed a suitable candidate for treatment with Q-switched Nd-YAG laser.

Treatment:

The Q-switched Nd-YAG laser was used for a total of 3 sessions at a spot size of 1 and power of 40-60 mj/cm2 under one hour of topical anaesthesia EMLA(eutectic mixture of topical anaesthetic). The second sessions were done with a gap of 1 month, followed by a gap of 2 months. Each session lasted approximately 15 minutes, during which the laser was applied over the entire nevus spilus.

Outcome:

After completion of 3 sessions, the nevus spilus was significantly lightened and its borders were less distinct. The patient was pleased with the outcome and reported improved self-confidence. No adverse effects or complications were observed during or after the treatment.

Discussion:

Nevus spilus is a common pigmented birthmark that typically appears in early infancy or is present at birth. It has a light brown or tan background with numerous dark brown to black macules or freckle on top. Despite the fact that nevus spilus is typically benign and causes no health issues, its appearance can cause patients cosmetic concern. (1)The Q-switched Nd-YAG laser emits high-intensity light in brief pulses. The laser is utilised in a variety of dermatological procedures, including the removal of pigmented lesions, tattoos, and vascular lesions. The ability of the Q-switched Nd-YAG laser to selectively target pigments within the epidermis is one of its primary advantages. The pigments absorb the laser energy, causing their fragmentation and eventual eradication. This selective targeting reduces the risk of tissue injury, making the Q-switched Nd-YAG laser an effective and safe treatment option.It has been demonstrated that Q-switched Nd-YAG laser is a safe and effective treatment option for nevus spilus. Utilising multiple sessions separated by intervals allows for optimal results while minimising adverse effects. Not all patients will achieve complete clearance of the birthmark with laser treatment, and the possibility of recurrence must be considered. (3)

Conclusion:

The In dermatological practise, the Q-switched Nd-YAG laser is a versatile and effective instrument. It is a safe and effective treatment option for a variety of skin conditions due to its ability to selectively target pigments and its low risk of adverse side effects. Q-switched Nd-YAG laser is a safe and effective option for removing nevus spilus for cosmetic. Multiple sessions separated by suitable intervals can effectively diminish the birthmark with minimal risk of side effects.

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Legend

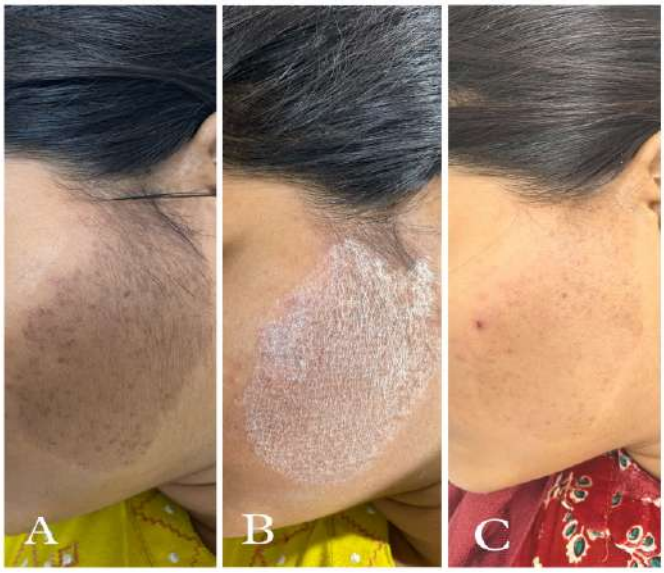

Figure A: Left cheek showing nevus spilus before treatment

Figure B : Appearance of the lesion immediately after treatment

Figure C: Significant clinical improvement after 3 sessions of QS ND YAG

Generalized Juvenile Xanthogranuloma with Ocular Complications: A Case Report

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Abstract :

Juvenile xanthogranuloma is a rare histiocytic disorder usually presenting in infancy with cutaneous papulonodular lesions. Systemic involvement is infrequent, with ocular manifestations being most common. We report the case of an infant presenting with generalized cutaneous lesions histologically confirmed as juvenile xanthogranuloma, associated with unilateral blindness.

Introduction :

Juvenile xanthogranuloma (JXG) is an uncommon benign proliferative disease belonging to the group of non-Langerhans cell histiocytoses. It was first described by Adamson in 1905 as congenital xanthoma multiplex.[1] It is seen most commonly in infancy, frequently presenting as a solitary asymptomatic well-defined yellowish-brown papule or nodule, typically in the head and neck area.[2] Although cutaneous lesions are self-limiting, systemic involvement, most commonly ocular lesions may lead to irreversible complications.[3]

In this report, we describe the case of an infant with disseminated cutaneous lesions of JXG associated with ocular complications.

Case Report :

A 7-month-old male presented with multiple reddish brown skin lesions on the head, trunk and upper limbs. The lesions had started at birth over the face and scalp, and gradually increased in number to involve the other areas. There was no associated itching or pain. In addition to the cutaneous lesions, the child had photophobia since birth, which had progressed to blindness in the right eye. He was diagnosed with congenital glaucoma, and had undergone surgery of the right eye at 5 months of age. No other systemic complaint was present. Birth history was unremarkable and the mother had an uncomplicated antenatal period followed by normal vaginal delivery at term. There was no history of similar cutaneous lesions running in the family.

On examination, there were more than 50 discrete well-defined reddish-brown papulonodular lesions distributed over the scalp, face, upper extremities, upper chest, back and gluteal region, ranging in size from 3 to 12 mm [Figure 1]. The overlying surface did not show any bleeding or ulceration. The lesions were soft to firm in consistency, non-fluctuant, non-indurated and non-tender. Mucosa, hair and nails were uninvolved. Ocular examination revealed enlargement of the right eyeball (buphthalmos), corneal opacity and blindness in the right eye [Figure 1]. The left eye appeared normal. Other systems did not show any abnormality. Growth parameters and developmental milestones were appropriate for his age.

Routine laboratory work-up showed normal results and serological tests for syphilis, rubella, cytomegalovirus, toxoplasma and herpes virus infections were negative in both the infant and mother. Lesional biopsy showed an atrophic epidermis and dense dermal infiltration by spindle-shaped or polygonal mononuclear histiocytes with indistinct borders [Figure 2]. Some of the cells showed foamy changes or vacuolation, although multinucleate giant cells of Touton were not obvious. On immunohistochemistry, the cells stained positively for CD68 and were negative for CD1a and S100, confirming them to be non-Langerhans cells. Thus, we arrived at a diagnosis of JXG. Owing to the extensive

cutaneous involvement, abdominal ultrasound, chest radiograph and CNS imaging were advised; however, no other organ involvement was found. The parents were counselled regarding the benign and self-limiting course of the cutaneous lesions. They were advised to consult the ophthalmologist for the management of eye complications and assessment of the functional eye.

Discussion :

JXG is the commonest among the non-Langerhans cell histiocytoses.[4] Although its etiology is unknown, it has been postulated to occur as a result of abnormal macrophage response to a non-specific injury or viral infection. Cutaneous lesions are known to present at birth in approximately 10% of cases, and within the first year of life in up to 85% infants.[5] A male predilection (M:F = 1.5:1) has been observed.[4]

The most common presentation is a solitary cutaneous asymptomatic reddish brown to yellowish papulonoduar lesion on the head and neck region.[5] Patients having multiple lesions usually present at birth or within six months of age.[2] Although there is no consensus regarding the number of lesions required to identify the condition as disseminated JXG, Selcen Kundak et al. suggested the term “eruptive JXG” for patients with six or more lesions.[6]

Presence of multiple lesions and appearance of lesions below two years of age are important risk factors for extracutaneous involvement, which is known to occur in up to 4% cases.2 The eye is the commonest extracutaneous location of JXG and ocular manifestations may be seen concomitantly with skin lesions.[5] Eye lesions are often unilateral, and range from iris lesions (most common) to eyelid nodule, conjunctival mass, uveitis, and posterior segment involvement.[2] When left untreated, these may progress to complications like spontaneous hyphema, secondary glaucoma, and even loss of vision.[4] Unilateral vision loss with a history of glaucoma in our patient suggests a strong possibility of complications arising from ocular JXG although such a diagnosis had not been made earlier. Other organs that have been reported to be involved are central nervous system, liver, spleen, lungs, pericardium, bones, bone marrow, and kidney.[2,4] Thus, a thorough systemic evaluation is warranted in these patients. JXG is also documented to be associated with neurofibromatosis type I and juvenile chronic myeloid leukemia.[4]

Cutaneous lesions should be differentiated from lesions of blueberry muffin syndrome, Langerhans cell histiocytosis, Spitz nevi, urticaria pigmentosa and xanthoma. Histopathologically, dermal infiltration with lipid-laden histiocytes having vacuolated or foamy cytoplasm is characteristic. In 85% cases, there may be multinucleated Touton giant cells, characterized by wreath-like distribution of nuclei around an eosinophilic cytoplasmic core; however, these were not prominent in our patient.[7] Notably, despite the presence of xanthomatous changes in the cutaneous lesions, serum lipids and metabolic profile remain unaffected. Immunohistochemistry staining positive for factor XIIIa, CD68, CD163, fascin and CD14, and negative for CD1a and S100, is confirmatory and helps to differentiate it from Langerhans cell histiocytosis.[3,4]

Cutaneous lesions usually undergo spontaneous resolution within a few years, with some residual hyperpigmentation. Surgical removal is warranted only in giant lesions greater than 2 cm in size. Extracutaneous lesions, on the other hand, lead to significant morbidity and mortality from complications, as is evident in our case. Therapeutic options in systemic disease include immunosuppressives, chemotherapy and radiotherapy.[4]

To conclude, JXG is a rare and benign disease, typically seen in the pediatric age group. Although cutaneous lesions are self-resolving, extracutaneous, particularly ocular lesions are frequently seen in those presenting early with multiple skin lesions. This case highlights the importance of timely diagnosis of this rare disease and detection of extracutaneous involvement to prevent the development of permanent complications.

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Figure Legends

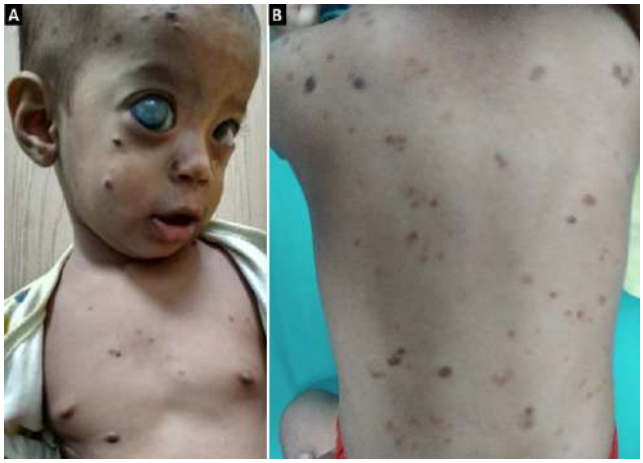

Figure 1:
A. Reddish-brown papulonodular lesions over the head and neck region and upper chest, along with enlarged eyeball and corneal opacity of right eye; B.Papulonodular lesions distributed over back

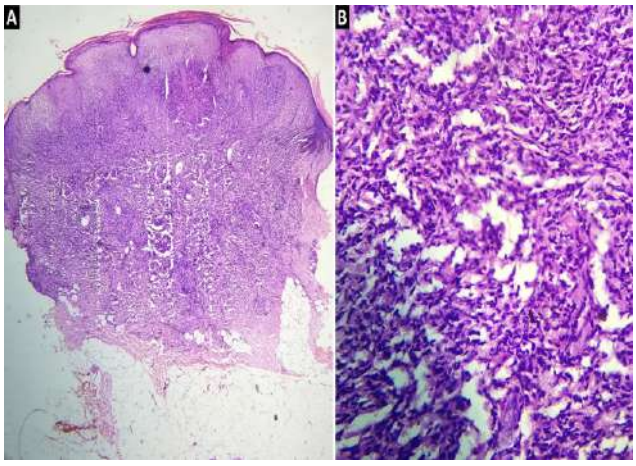

Figure 2:
A. Dense dermal infiltrate consisting of spindle-shaped mononuclear histiocytes (H&E stain, 40X); B. Infiltrating cells have indistinct borders, with few cells showing vacuolation giving rise to a foamy appearance (H&E stain, 400X)

A mysterious Circular Verrucous lesion on the Cheek

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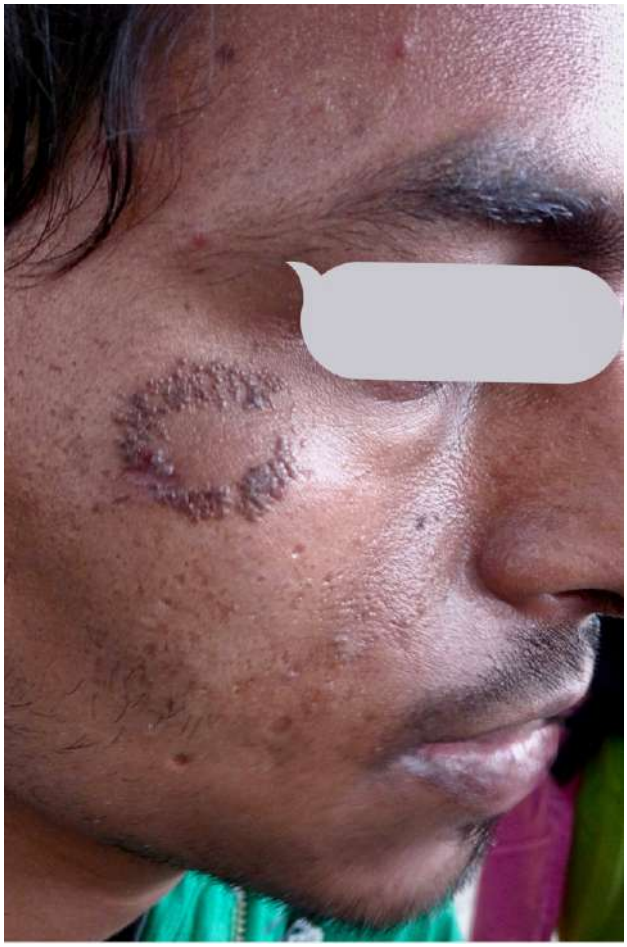

A 25 year old male presented with a well-defined brownish round lesion approx. 4 x4 cm in size, over right cheek since puberty. Lesion is gradually increasing to its current size and showing a distinct central clearance.Lesion was mostly asymptomatic with minimal itching. Other cutaneous and mucosal examination was normal. Clinically it was like a verrucous epidermal nevus (VEN) but central clearance couldn't be explained as patient denied any procedure or traumatic removal of central area. Histopathology (punch biopsy mark on 5o clock position) proved it as VEN. Regrowth of normal cells with increasing age, within the mosaic verrucous cells could be a possible hypothesis for this kind of circular VEN.

Air pollution and its effects on skin - an urgent concern to take action

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Air pollution is a major environmental concern that has been linked to a wide range of health problems, including skin problems. The skin is the largest organ of the body and is constantly exposed to the environment, making it highly susceptible to the effects of air pollution. Air pollution is a complex mixture of various pollutants, including particulate matter (PM), ozone (O3), nitrogen oxides (NOx), sulfur dioxide (SO2), and volatile organic compounds (VOCs). Each of these pollutants has been independently linked to skin problems in numerous studies. PM has been associated with increased skin dryness, roughness, and wrinkles.(1) O3 has been linked to skin irritation, rashes, and hyperpigmentation. NOx and SO2 have been associated with skin inflammation and eczema. VOCs have been linked to skin aging and increased risk of skin cancer.

In addition to these specific pollutants, air pollution has also been linked to an overall increase in skin problems. A study conducted in China found that individuals living in areas with high levels of air pollution had an increased risk of developing skin diseases such as acne, eczema, and psoriasis. Another study conducted in Mexico City found that individuals living in areas with high levels of air pollution had an increased risk of developing atopic dermatitis.

The mechanisms by which air pollution contributes to skin problems are not fully understood, but several theories have been proposed. One theory is that pollutants can cause oxidative stress in the skin, leading to inflammation and damage to DNA. Another theory is that pollutants can disrupt the skin's barrier function, making it more susceptible to irritation and infection. A third theory is that pollutants can increase the production of melanin, leading to hyperpigmentation.

There are a few measures that can be taken to reduce the impact of air pollution on the skin. One way is to limit outdoor activities during times of high pollution. Another way is to use sunscreens and other protective creams to create a barrier between the skin and the pollutants. Washing the face and hands frequently, using gentle cleansers, and using a humidifier to keep the air inside the home moist can also help.

In conclusion, air pollution is a serious environmental concern that has been linked to a wide range of skin problems. The skin is highly susceptible to the effects of air pollution and studies have shown that individuals living in areas with high levels of air pollution have an increased risk of developing various skin diseases. There are several theories that have been proposed to explain the mechanisms by which air pollution contributes to skin problems, but more research is needed to fully understand these mechanisms. While it is not possible to completely eliminate exposure to air pollution, there are several measures that can be taken to reduce its impact on the skin, and it's important for individuals to be aware of the dangers of air pollution and take the necessary precautions.

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Pyogenic granuloma over Hidradenitis Suppurativa in a child - Removed with CO2 laser

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Pyogenic granuloma (PG) is a benign cutaneous and mucous membrane vascular lesion. It manifests as a rapidly growing, rosy-pink, scaly nodule or papule with a smooth or lobulated surface. The head and neck, limbs, and feet are typical locations for PGs. It can develop in pregnant subjects, where the gingiva is the most common location. (1) Hidradenitis suppurativa (HS) is a chronic inflammatory skin condition characterised by excruciating nodules, abscesses, and sinus tracts in the axilla, inguinal, and anogenital regions. (2)A case of pyogenic granuloma that developed over an axilla with hidradenitis suppurativa is described here.

A 12 year-old female presented to our dermatology clinic with a rapidly growing lesion on his left axilla. He had a history of hidradenitis suppurativa in the same area for the past 2 years, for which he was receiving treatment intermittently. The lesion was initially small, but it rapidly grew in size over the past few weeks. The patient gave history of frequent bleeding from that site. Even minor rub initiated bleeding episodes which took half an hour to stop. There was no history of fever, chills, or any other systemic symptoms.

On examination, there was a 2 cm x 3 cm nodular, friable, erythematous lesion on the left axilla with overlying crusts and erosions. There was tenderness on palpation of the lesion. The surrounding skin showed multiple scars and sinus tracts of hidradenitis suppurativa. The rest of the systemic examination was unremarkable.A the lesion was removed using ablative carbon dioxide laser therapy from its base and the mass was sent for histopathological examination. The histopathology showed proliferation of capillaries with endothelial cells, accompanied by a mixed inflammatory infiltrate consisting of lymphocytes, neutrophils, and eosinophils. No malignancy was seen. The histopathological findings were consistent with pyogenic granuloma. The lesion healed in 6 days time without recurrence in 6 months follow up.

Common locations for pyogenic granulomas include the head, neck, hands, and feet, all of which have a high vascularity. They can also develop in areas of chronic irritation or inflammation. The pathogenesis of PG is unknown, but it is believed to be caused by a local vascular reaction to injury or irritation. (1) Additionally, patients with HS who are treated with retinoids are more likely to develop PG. (3) (4) Surgical excision, electrocautery, cryotherapy, laser therapy, or topical application of silver nitrate may be used to treat PG. The choice of treatment depends on the size, location, and individual preference of the affected area.

In areas of chronic irritation or inflammation, such as in hidradenitis suppurativa, pyogenic granuloma can develop. To exclude the possibility of malignant transformation, we advise physicians caring for HS patients to perform a biopsy or excision if a chronic lesion is accompanied by a tumour that bleeds. The treatment options for PG are very simple, and the resolution has a very brief recovery period. The choice of treatment depends on the size, location, and individual preference of the affected area.

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Legend

**Figure: A: Right axilla showing a lobulated reddish mass over hidradenitis suppurativa lesion.
B: The site after removing the lesion with CO2 Laser**

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