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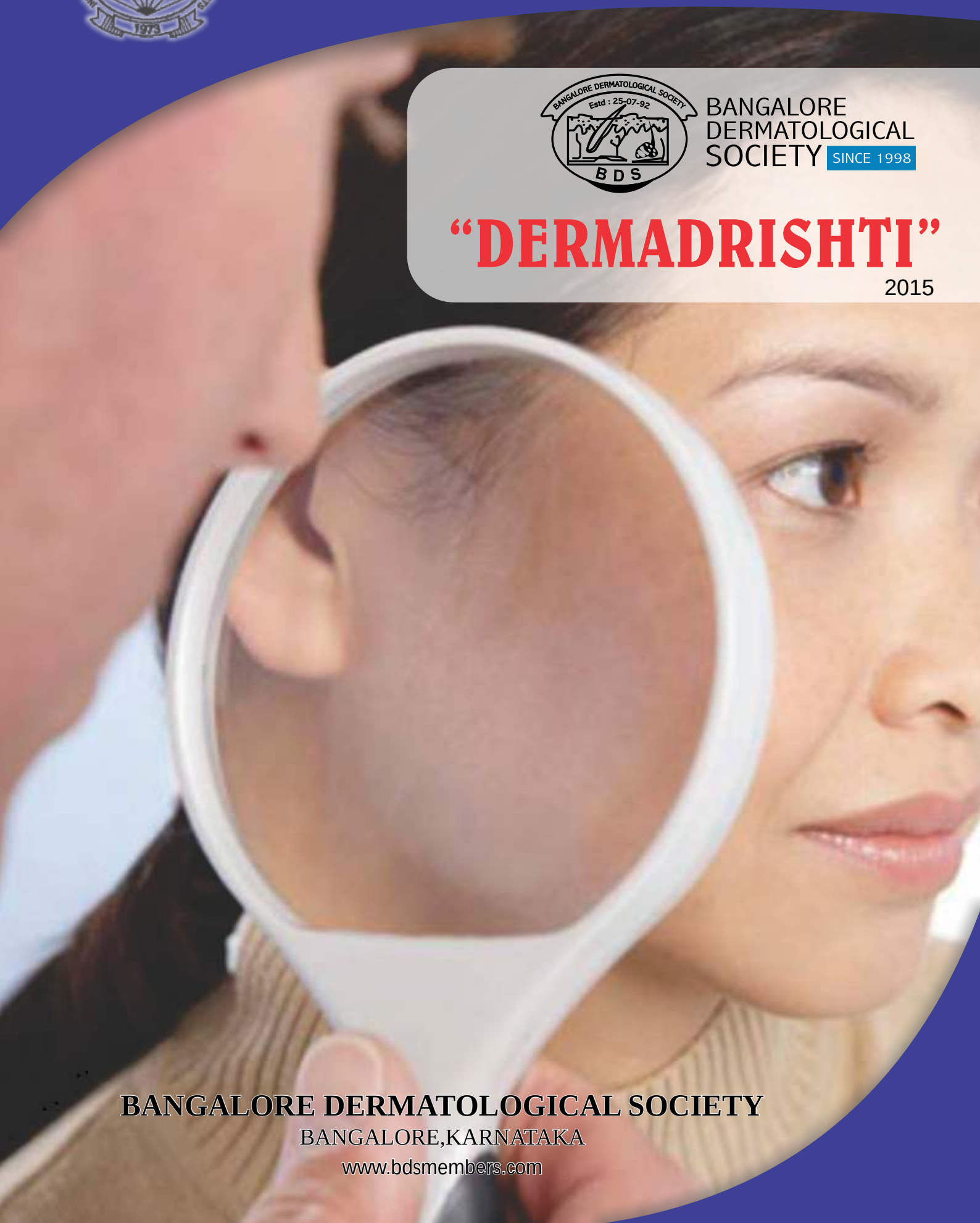

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Diaper Dermatitis - An update

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Introduction

Diaper dermatitis (napkin dermatitis, nappy rash) includes all eruptions that occur in the area covered by diaper. It is one of the most common cutaneous disorder seen in infancy and early childhood affecting 20% of all childhood dermatology visits. The term diaper rash is not a specific diagnosis as it encompasses many dermatoses affecting this region and is best viewed as a symptom complex. [1] These conditions are caused directly by wearing of the diaper or diaper environment, some of them may be worsened by diaper and there are dermatoses that occurs independent of the presence of diaper.

Diaper dermatitis was first described by Jacquet in 1905. Later Zahorsky in 1915 described that ammonia released from urine was the primary irritant in napkin dermatitis. [2] But few decades later investigations have shown that etiology of napkin dermatitis is complex. The incidence is between 7% and 35% with a peak incidence at 9 to 12 months of age. Most cases are mild, severe forms occurs in 5% of cases. [3]

Etiopathogenesis

Infant skin has distinct skin physiological features that affect skin barrier function and water handling properties. Skin pH and skin hydration is higher in diapered area as compared to non diapered region in infants. Factors that are involved in the pathophysiology of diaper dermatitis are excessive wetness, friction, high pH, high enzymatic activity due to faeces and urine. [4] Skin wetness and maceration increases the susceptibility to friction between skin and the diaper fabric causing physical damage. Urine and use of napkin itself may contribute to skin hydration. Napkin may prevent the evaporation of moisture and compromise epidermal barrier function.

Prolonged contact with faeces is the most irritant factor on the diaper area. Faecal ureases catalyse the breakdown of urea to ammonia which increases the pH of the skin. [5] Increased activity of faecal enzymes, proteases, ureases and lipases produces high irritation to the skin producing erythema. These enzymes increase the permeability to other potential irritants. Some studies have shown that there is no relation between faecal enzyme activity and napkin dermatitis. [6] Diarrhoea, increased bowel movements and developmental abnormalities of urinary tract predisposes to diaper dermatitis.

Microorganisms gain entry through the damaged skin. Most common organisms isolated are candida albicans and staphylococcus aureus.

Role of napkin in diaper dermatitis

It is a misconception that allergy to napkin causes napkin dermatitis. Allergic contact dermatitis to napkin occurs due to components like rubber, detergents, lanolin, neomycin, mercurial compounds and the wet wipes that contain methylisothiazolinone. Absorbency effectiveness in diapers has improved significantly in recent years with the advent of new ingredient combination and advanced design features. [7] There are no allergic reactions reported so far due to acrylate gel containing diapers. In view of the above reasons, modern disposable napkin is better than the traditional cotton napkin.

Clinical features

The different dermatoses that can present in diaper region are listed in Table 1.

Table 1: Differential diagnosis of diaper dermatitis

1. Primary irritant contact dermatitis	12. Bullous impetigo
2. Allergic contact dermatitis	13. Acrodermatitis enteropathica
3. Intertrigo	14. Cystic fibrosis
4. Candidiasis	15. Multiple carboxylase deficiency
5. Seborrhoeic dermatitis	16. Perianal pseudoverrucous papules and nodules
6. Atopic dermatitis	
7. Psoriasis	17. Child abuse
8. Kawasaki disease	18. Congenital syphilis
9. Granuloma gluteale infantum	
10. Perianal streptococcal disease	
11. Lamgerhans cell histiocytosis	

Primary irritant contact is the most common form of napkin dermatitis. It is characterized by erythema, edema, papules, scaling on the convex surface of the pubic area, buttocks, medial aspect of thighs, scrotum, labia majora reflecting the areas of the body in contact with the diaper. It spares the inguinal folds. There are two morphological subtypes of irritant contact dermatitis: tide mark dermatitis where there is a band like erythematous eruption confined to the margins of the napkin areas on the thighs. [8] Jacquet's dermatitis is a severe erosive form of irritant contact napkin dermatitis. They are also called as 'ammoniacal ulcers'. Clinically presents as papuloerosive and nodular lesions, superficial erosions or punched out ulcers with raised erythematous border.

Diaper dermatitis can be secondarily infected with candidiasis which involves deep inguinal folds associated with satellite pustules. Granuloma gluteale infantum is characterized by painless reddish brown to purple nodules on the buttocks and inner thighs. This is most commonly seen due to use of fluorinated topical steroids. [9] Staph aureus infection presents as bullous impetigo. Perianal pseudo-verrucous papules and nodules is a rare condition attributed to chronic urinary incontinence. Characterized by red, flat-topped papules and papulonodules on the perianal region. Langerhans cell histiocytosis can involve diaper area. Clinically presents as infiltrated or eroded papules, purpuric lesions on the inguinal folds resembling seborrhoeic dermatitis.

Diagnosis

Diaper dermatitis is a clinical diagnosis. Detailed history and morphology of the lesion is important for diagnosis. Skin biopsy is not required routinely. It is considered only when other differential diagnosis should be ruled out.

Management

Prevention is the cornerstone in the management of diaper dermatitis. There are no standard guidelines for prevention of diaper dermatitis. Care of the napkin area is outlined in Table 2.

Table 2: Preventive measures for diaper dermatitis

1.	Use of highly superabsorbent acrylate gel napkins is recommended
2.	Non disposable cloth napkins must not be rinsed in antiseptics or fabric conditioners. Adequate rinsing should be done after washing
3.	Frequent napkin change is very important. For newborns with high wetting frequency diaper change should be done every 2 hours. For older infants napkin change should be done 7 times/day or every 3-4 hours.
4.	Harsh soaps and scrubbing of diaper region should be avoided. Lukewarm water or mild cleansers (acidic to neutral PH) can be used. [10] Cleansing using cotton wool or formulated wipes are safe.
5.	Alcohol and fragrance free wipes formulated with emollient cleansers and acidic PH protect the barrier function
6.	Skin in the diaper area should be gently patted and not rubbed.
7.	Application of barrier creams after each diaper change is recommended. Zinc oxide or petrolatum based products are used. This prevents contact with urine and faeces and reduces irritation. Use of baby powders should be discouraged [11]
8.	Breastfeeding has a preventive role in diaper dermatitis. This is due to the lower PH and lower enzymatic activity of faeces than that of formula fed babies. [12]

Treatment

Counselling about diaper rash to care takers is very important in the management. Stepwise approach in the treatment of diaper dermatitis is outlined in Table 3.

Basic preventive care of the diaper area as mentioned in Table 1

↓

Rash not improved by barrier creams Mild topical steroid can be used (1% hydrocortisone) for 5 -7 days once at night along with barrier creams

↓

Treat if secondary infection is present (bacterial/candidiasis) Topical antibiotics/ imidazoles/oral nystatin suspension/oral fluconazole

↓

Diaper rash not resolving with above management/persistent, to consider other differential diagnosis and investigate

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PSORIASIS & EYE: AN UNCOMMON ASSOCIATION – A REVIEW.

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Psoriasis is a multi-system chronic inflammatory skin disease targeting 2% to 3% of the general population. It is a prototype of immune dysregulation mediated by TH1 proinflammatory cytokines, with far reaching systemic effects. There is growing evidence that with severe skin disease portends a serious risk for development of other comorbidities and are found to have a higher association of extracutaneous disease manifestations. The causal relationship between the eye and psoriasis has been a topic of discussion since decades, but the precise eye manifestations in patients with psoriasis and psoriatic arthritis are only recently findings. Psoriatic eye findings may include conjunctivitis, dry eye, episcleritis, and uveitis, all of which may precede articular changes. Uveitis, seen in 7% to 25% of psoriatic arthritis patients, may be recognized by the presence of conjunctival injection, photophobia, pain, lid swelling, or otherwise unexplained visual changes. Early recognition and prompt treatment is important because its natural course may lead to vision loss. This report highlights the importance of psoriasis and eye involvement, and further need for interdepartmental association between dermatologists and ophthalmologists.

The relationship between the eye and psoriasis has been recognized for decades, but the precise eye manifestations in patients with psoriasis are only recent findings.1-4 Psoriatic eye findings may include conjunctivitis, dry eye, episcleritis, and uveitis. Eye findings in conjunction with psoriatic arthritis were reported in 1976 by Lambert and Wright, who noted the presence of ocular inflammation in 31.2% of 112 patients with psoriatic arthritis, with conjunctivitis the most common lesion (19.6%), followed by iritis (7.1%).5 Psoriatic arthritis has traditionally been thought to precede psoriatic eye manifestations, but a minority of cases are seen in the reverse order.6 Uveitis may manifest solely in the eye, or it may be associated with a systemic disease. Multiple studies quote the prevalence of uveitis in psoriasis and Psoriatic arthritis 4,5,8 the highest of which is three of seven patients with psoriasis.5,9

For patients with psoriasis, uveitis had been commonly thought to occur only in conjunction with psoriatic arthritis; however, there have been many case reports of psoriatic uveitis presenting independent of joint disease.3,10,16 Furthermore, the temporal relationship of these two entities has been disputed. Some recent studies suggest that for most spondyloarthropathies (SpAs), inflammatory joint manifestations precede uveitis.11-13 Nevertheless, some cases of uveitis have been reported to occur even before psoriatic skin disease,6 and uveitis has been reported as the first presenting sign of SpAs in 0% to 11.4% of cases. The severity of ocular inflammation does not necessarily correlate with extent of joint findings but may correlate with skin disease.14-16

Acute uveitis attacks typically present with pain, intense photophobia, red eye, blurred vision/miosis (pupil constriction), and varying degrees of lid edema.17 Conjunctival injection in acute anterior uveitis begins at, and is most intense around, the edge of the cornea. Eyes affected by uveitis may have smaller pupils than on the unaffected side because inflammation may trigger muscle spasm of the iris sphincter, or the pupil could be distorted by posterior synechiae. However, the actual predictive value of symptoms in diagnosing uveitis is unknown. In fact, the only warning sign may be unexplained poor vision.18 Thus, patients who show no evidence of inflammatory changes should nevertheless be referred to an ophthalmologist if symptoms worsen.

Psoriatic uveitis is most commonly anterior, although it can be associated with posterior uveitis as well.10,19 It is also more likely than other forms of spondyloarthropathy-associated uveitis to be insidious in onset, bilateral, with periodic flares.5,10,19, 20 All complaints should be referred to an

ophthalmologist for evaluation. Nonophthalmologists can assess a patient's visual acuity and examine the external eye for circumcorneal injection. Physicians may evaluate with a direct ophthalmoscope for evidence of decreased corneal transparency, keratic precipitates (inflammatory cells on the cornea), and posterior synechiae (adhesions of the lens and iris).18 However, the diagnosis of uveitis must be confirmed with a slit-lamp examination performed by an ophthalmologist. HLA-B27, as noted, is not currently considered diagnostically useful.10

Although the exact underlying mechanisms contributing to the link between psoriasis and uveitis remain poorly understood, there are common etiologic pathways involved in the pathogenesis of both entities. More research on the relationship between uveitis and psoriasis is needed. In particular, a greater understanding of the frequency of psoriasis-specific uveitis may shed light on the importance of surveillance. Current experimental eye models for the study of uveitis do not specifically address the pathophysiology of psoriatic uveitis.17 Long-term follow-up of psoriasis patients with eye manifestations would provide more insight into pathogenesis and treatment methods.

Psoriatic eye manifestations, uveitis in particular, can lead to serious consequences, including vision loss. These manifestations have been reported more frequently in psoriasis patients with arthritis, but they have also been reported in psoriatic patients without arthritis. Psoriatic eye manifestations may precede articular changes. Referral to an ophthalmologist is essential for definitive diagnosis and treatment. Corticosteroids are the primary treatment modality. However, increasing emphasis has been given to immunomodulators and TNF blockers for the more intractable cases. TNF blockers may be promising for the prevention of induction and recurrence of uveitis in psoriasis patients.18

This report highlight the importance of thorough evaluation of any pre-existing psoriasis and involvement of eye, and coordination between dermatologists and ophthalmologists prior to any interventions. Hence we recommend regular surveillance of psoriasis patients for visual changes and eye symptoms.

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STASIS DERMATITIS

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"It's the stasis that kills you off in the end, not ambition".

SYNONYMS:

Stasis eczema, Gravitational eczema, Venous eczema, Varicose eczema, Hypostatic dermatitis, Liposclerosis dermatitis.

INTRODUCTION:

Stasis dermatitis is a cutaneous marker of Chronic Venous Insufficiency [CVI]. Cutaneous manifestations of CVI include lower extremity oedema, varicose veins, varicose eczema, atrophie blanche, Lipodermatosclerosis (LDS) and venous ulcers. Stasis dermatitis is often associated with varicose veins, however they are not present always. Stasis dermatitis is a misnomer because high blood flow, not stasis, which occurs in the capillary bed is the prerequisite. Stasis dermatitis is one of the most common causes of secondary dissemination of dermatitis.

DEFINITION:

Eczema secondary to venous hypertension of the lower extremities.

EPIDEMIOLOGY:

The prevalence is different among various races and societies. Prevalence rates increase with age. Females are affected more than males, this is most likely due to the fact that pregnancy results in significant stress on the lower-extremity venous system, with many women experiencing earlier and more severe derangement of lower-extremity valvular function. The epidemiological data of the prevalence rates is scarce.

PATHOPHYSIOLOGY:

The pathophysiology of cutaneous manifestations of CVI is not clear. It involves chronic ambulatory venous hypertension and resulting micro-angiopathic and inflammatory changes, which lead to classic clinical and histologic changes. Venous hypertension of lower extremities result from calf pump failure or abnormalities in venous system, such as valve dysfunction, venous outflow obstruction or combination of these.

Venous hypertension slows the blood flow in the microvasculature, distends the capillaries and damages its permeability barrier, allowing the passage of fluid and plasma proteins into tissue and extravasation of erythrocytes. The fibrinogen that leaks out into dermis forms layer of fibrin around the capillaries (pericapillary fibrin cuffs), which acts as a barrier for diffusion of oxygen and other nutrients that are essential for skin viability and this leads to hypoxia and poor skin nutrition.

Slow blood flow induces upregulation of ICAM-1 and VCAM-1 on endothelia, expression of L-selectin on neutrophils, and activation of neutrophils and macrophages. Neutrophils are attracted into and trapped within the affected areas (specifically the medial supramalleolar region). Release of inflammartory mediators, free radicals and proteases by neutrophils leads to pericapillary inflammation. Matrix metalloproteinases may be important in lesional skin remodeling. It is very likely that chronic inflammation and microangiopathy are responsible for stasis dermatitis. Stasis dermatitis typically occurs in the same regions where microangiopathy is most intense (i.e. the medial supramalleolar area), and the patches of dermatitis arise preferentially over dilated varicose veins. Also, dermal inflammation is known to induce epidermal dysfunction (hyperproliferation, barrier impairment, desquamation).

CLINICAL FEATURES:

Early clinical findings of CVI include lower extremity oedema, a condition that frequently presents at the ankles, worsens towards the end of the day, and improves overnight. Over months to years, as venous hypertension continues, distal red brown hyperpigmentation due to extravasated

erythrocytes, hemosiderin-laden macrophages, and melanin deposition occurs. These changes tend to be localised to the gaiter area. At this stage of disease, xerosis and pruritus may appear and can develop into venous eczema. It is often, but not always, bilateral. It may begin as sharply demarcated erythematous papules and vesicles; however it eventually becomes diffuse, poorly defined, and may demonstrate serous exudate and crust. It usually seen around the ankle and lower leg, may extend to just below the knee: this is sometimes referred to as stocking erythroderma. The dorsal part of the foot may be involved in severe cases. It often occurs as a late result of deep vein thrombosis. Occasionally, similar changes occur at other sites of venous hypertension such as the pendulous skin over an obese abdomen or in association with an arteriovenous ?stula in the upper limb. The eczema may develop suddenly or insidiously.

Factors that worsen peripheral oedema (eg, congestive heart failure, long standing hypertension with diastolic dysfunction) are often found in patients with stasis dermatitis. Some anti-hypertensive medications (such as amlodipine) may increase leg edema and trigger the onset of stasis dermatitis.

Although not always present, varicosities range from thin telangiectasia to submalleolar venous flares to larger tortuous vessels The eczema is often accompanied by other features of venous hypertension like purpura, atrophie blanche, lipodermatosclerosis and venous ulceration. Stasis dermatitis can lead to fat

Table 1 Classification System for Chronic Venous Disease (CEAP)[3]

Grade	Description
C: Clinical Manifestations	
C_0	No visible or palpable signs of venous disease
C_1	Telangiectasias or reticular veins
C_2	Varicose veins; distinguished from reticular veins by a diameter of 3 mm or greater
C_3	Edema
C_4	Changes in skin and subcutaneous tissue secondary to chronic venous disease: 4a (pigmentation or eczema), 4b (LDS or atrophie blanche)
C_5	Healed venous ulcer
C_6	Active venous ulcer
E: Etiologic Factors	
E_C	Congenital
E_P	Primary
E_S	Secondary (post-thrombotic)
E_N	No venous cause identified
A: Anatomic Distribution of Disease	
A_S	Superficial veins
A_P	Perforator veins
A_D	Deep veins
A_N	No venous location identified
P: Pathophysiologic Findings	
P_R	Reflux
P_O	Obstruction
$P_{R,O}$	Reflux and obstruction
P_N	No venous pathophysiology identifiable

Abbreviation: LDS, lipodermatosclerosis.

necrosis with the end stage being permanent sclerosis (lipodermatosclerosis) with "inverted champagne bottle" legs. Patients with lipodermatosclerosis may also have acute inflammatory episodes that present with pain and erythema (these episodes can be mistaken for cellulitis).

Based on cutaneous findings, etiologic factors, anatomic distribution of disease and pathophysiologic findings, the chronic venous disease is classified as follows.

In these patients of venous eczema, secondary infection and contact dermatitis are common. Secondary infection can occur and should be suspected when the skin barrier has broken down and other signs of impetiginisation exist. Inciting agents of contact dermatitis include topical antibiotics such as neomycin and bacitracin, lanolin products, fragrances, parabens, corticosteroids, rubber components, and epoxy resin. Secondary patches of eczema may develop on the other leg, even when it is not affected by obvious venous insuf? ciency. Generalized secondary dissemination may occur, and occasionally this can progress to erythroderma. In long standing lesions, lichenification and hyperpigmentation may occur as a consequence of chronic sctratching and rubbing.

PATHOLOGY:

Histopathology reveals an acute or subacute dermatitis. Acute lesions may exhibit a superficial, perivascular lymphocytic infiltrate; epidermal spongiosis; serous exudate; scale; and crust. Chronic lesions may show epidermal acanthosis with hyperkeratosis. The dermis is characterised by deep dermal aggregates of siderophages due to uptake of hemosiderin from degraded erythrocytes. Dermal capillaries are frequently dilated; long standing lesions show intimal thickening of small arterioles and venules along with dermal fibrosis. Fibrosis is variable and a mononuclear cell inflammatory infiltrate is somewhat inconspicuous.

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis includes other papulosquamous conditions such as nummular eczema and psoriasis. In psoriasis, additional body sites are affected with nail changes or joint complaints. Xerosis and asteatotic eczema can resemble venous eczema but are frequently more diffuse. Venous eczema is commonly misdiagnosed as cellulitis. Both entities can present with pitting edema, erythema, serous drainage, and even desquamation. However, cellulitis is usually unilateral, tender, and may be associated with systemic symptoms such as a fever. Venous eczema is commonly bilateral and tends to be itchy, non-tender, and more chronic. Allergic and irritant contact dermatitis should also be considered and the patient should be questioned about topical applications to the skin. In cases where allergy is suspected, patch testing is a valuable diagnostic tool. Other differential diagnosis include pigmented purpuric dermatitis, pretibial myxedema, necrobiosis lipoidica and cutaneous T-cell lymphoma.

INVESTIGATION:

Biopsy should be avoided as the histologic changes are not specific to this entity and the biopsy site may fail to heal. In chronic stasis dermatitis, biopsy may be necessary if acroangiodermatitis (pseudo-Kaposi sarcoma) has developed. Duplex ultrasound should be performed to confirm the suspected diagnosis of venous insufficiency and can specifically identify incompetent veins. Dermoscopy shows a scaly surface and red globules at low magnification(x10) & glomerularlike vessels at high magnification(x30). In patients with suspected venous thrombosis a thorough hematologic workup is to be done to rule out underlying hypercoagulability states.

TREATMENT:

Management of venous eczema must address the underlying venous insufficiency, primarily by compression therapy. Compression is a mainstay of treatment for CVI. Given that venous eczema and LDS also improve with compression, this should be the first line treatment recommendation for patients. In particular, below-the knee, open-toe, graded

compression is ideal. Pressure at 20-30 mm Hg may be sufficient for less severe cases, however, patients with any history of ulcer disease should employ 30-40 mm Hg. Compression can be accomplished by means of specialised stockings that deliver a controlled pressure gradient, which should be applied early in the morning, before the patient rises from bed, in order to facilitate application when leg oedema is at its lowest point. High-level compression can be performed by using elastic wraps, unna (compression) boots, and more sophisticated devices, such as end-diastolic compression boots. It is important to note that up to two thirds of patients may be non-adherent in the use of compression stockings for reasons including a binding sensation, presumed ineffectiveness, sensation that they are too hot to wear, limb soreness, poor cosmesis, contact dermatitis, pruritus and cost. Prior to attempting more aggressive interventions, clinicians should attempt to elicit an honest report from patients regarding their compliance with compression. Other simple interventions focus on lifestyle changes such as weight loss, increased leg elevation, and increased exercise to improve calf muscle pump function.

In addition to treating underlying venous insufficiency with compression, venous eczema is managed topically with emollients and immune-modulators, including corticosteroids and calcineurin-inhibitors. For severe venous eczema, a short course of oral corticosteroids can be helpful. Weeping lesions can be treated with wet to damp guaze dressings soaked with water or with a drying agent, such as aluminium acetate. Mild topical steroids may be used to relieve irritation, but the use of potent steroids should be limited to short periods of a few days as they may cause cutaneous atrophy and increase the risk of ulceration. In a study by Maroo et al, who used oral doxycycline with topical tacrolimus for treatment of stasis dermatitis due to venous insufficiency. The authors site the anticollagenase, anti-inflammatory and immune-modulatory effects of doxycycline and the T-cell inhibitory effects of tacrolimus as important mechanisms for disease modification. Because the calcineurin inhibitors do not carry the risks of skin atrophy or tachyphylaxis, they have the potential to become valuable agents in the treatment of chronic dermatoses such as stasis dermatitis. If secondary infection is present, it is better to use a systemic antibacterial rather than a topical one because of the risk of contact sensitization. Sedative antihistamines help to relieve itching. Oral pentoxifylline 400mg three times a day may be used to improve the venous circulation. Calcium dobesilate 500mg twice a day has been reported as an effective adjuvant therapy in patients with venous ulcers and stasis dermatitis.

Stasis dermatitis related to an arterio-venous fistula or incompetent perforators may respond to ligation of the vessels.

CONCLUSION:

Though CVI originates with abnormalities of venous system, the target organ of CVI is skin thus producing several cutaneous manifestations. A through history and physical examination is necessary to arrive at diagnosis of stasis dermatitis. Compression therapy remains the corner stone in the management of stasis dermatitis.

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5. CROSSWORD ON SIGNS

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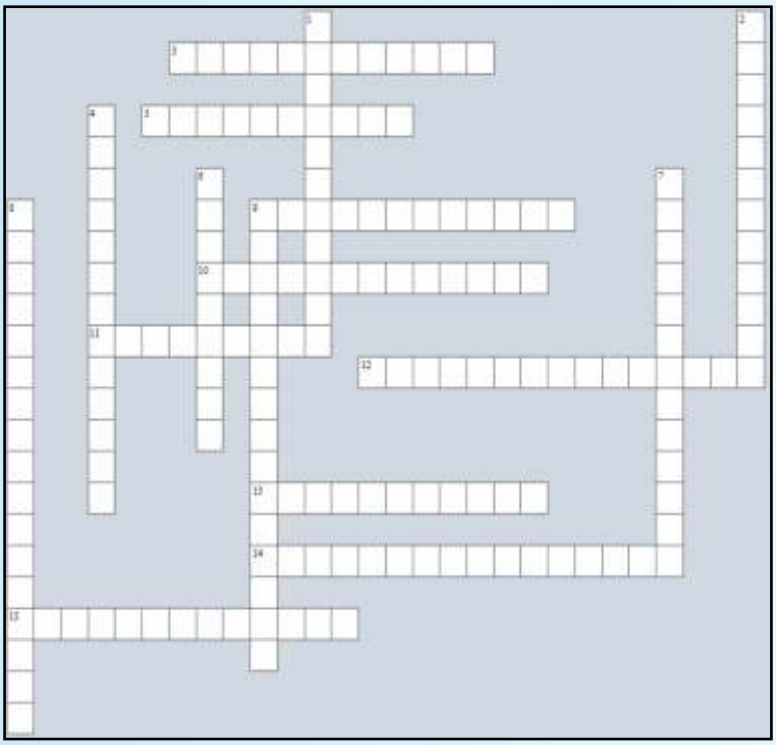


Across

3. Subungual corn (12)
5. Pain in the nail unit (10)
9. Biting of nails (12)
10. Longitudinal striations on the nail plate(13)
11. Absence of nail unit (9)
12. Thickened and upturned nail (15)
13. More than one nail on a digit (11)
14. Destroying the nails by some method (16)
15. Superficial splitting of nail plate (13)

Down

1. Spooning of nail plate (11)
2. Localized or diffuse hyperkeratosis of nail folds (12)
4. Proximal nail separation (13)
6. Through and through pit in the nail (9)
7. Rough nails (13)
8. Abnormally placed nail on the digit (17)
9. Ingrown nail (15)

(Answers to Crossword on Page No.14)

4. Case report with Quiz

MILIA EN PLAQUE: A RARE ENTITY

Dr. Praveen Kumar S[1], Dr. K.N. Shivaswamy[2], Dr. Sreedhar[3]
1, 2 Department of Dermatology, 3 Department of Pathology
M.S. RAMAIAH MEDICAL COLLEGE

Case Report:

A 60 year old male presented with raised, asymptomatic lesions over both sides of the cheeks since 4 years. On examination, the lesions were skin coloured plaques, seen distributed bilaterally symmetrically over the cheeks, firm in consistency, non-tender, mobile. On applying lateral pressure, cheesy material was seen extruding from surface of the lesion.

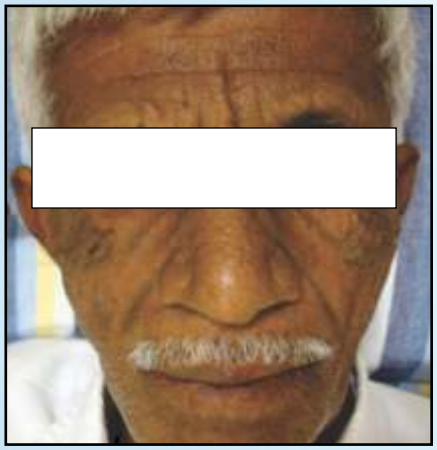


Figure 1

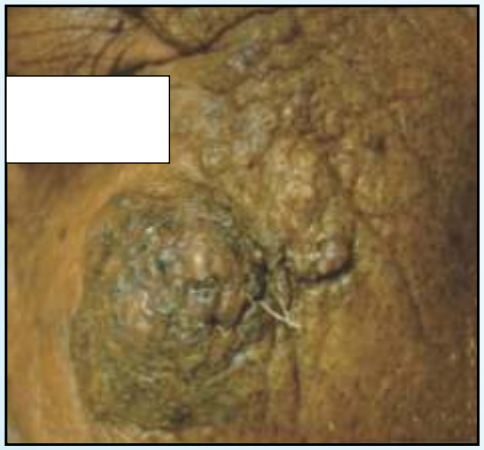


Figure 2

Histopathological examination revealed presence of epidermal cysts with layers of keratin and the cysts were surrounded by inflammatory infiltrate(Fig 3,4).

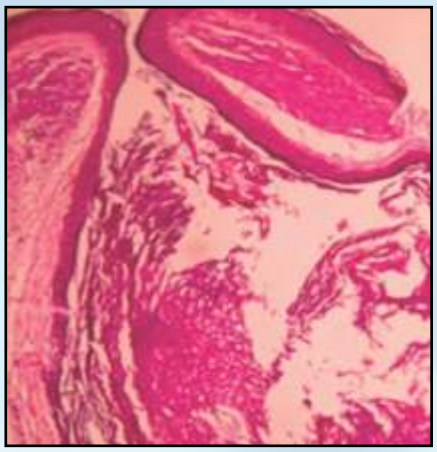


Figure 3

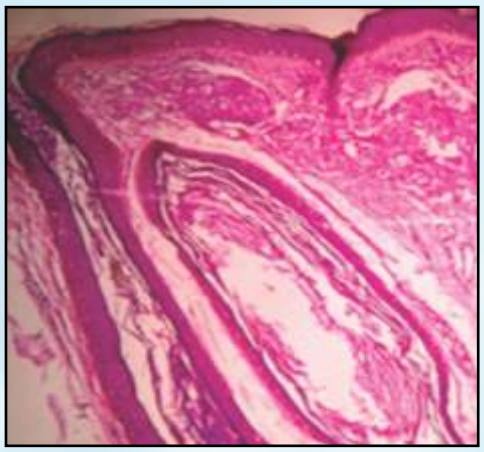
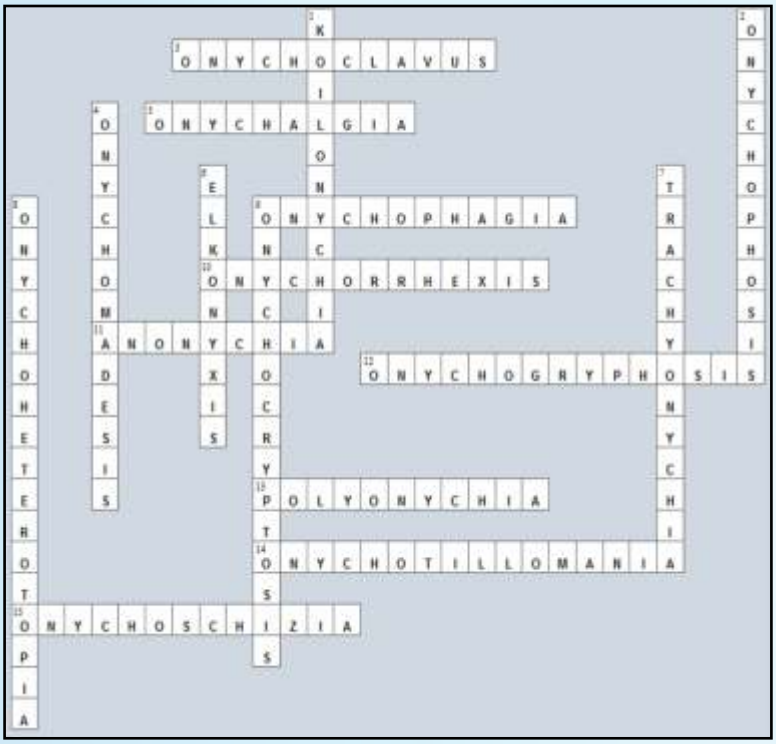


Figure 4

(Answers to Quitz on Page No.15)

8. ANSWERS TO CROSS WORD



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7. ANSWERS TO CASE REPORT & QUIZ

MILIA EN PLAQUE: A RARE ENTITY.

DISCUSSION:

Milia are small keratin-filled cysts that result from obstruction of hair follicle or eccrine sweat duct. Milia en plaque(MEP) is a form of primary milia characterized by the presence of milia on an erythematous infiltrated plaque. The condition was first described by Balzer and Fouquet in 1903 and was denominated by Heubler in 1978.

The present case highlights the presence of bilaterally symmetrical lesion under eyelids. The various differential diagnosis to be considered are nevus comedonicus, Favre-Racouchot disease, lichen planus follicularis tumidus and folliculotropic mycoses fungoides. Histopathological evaluation is a must in differentiating the entities. The histopathological presentation in MEP includes presence of multiple epidermal cysts filled with keratin and they are surrounded by an inflammatory infiltrate.

The lesions usually spontaneously regress but may remain unchanged if left untreated. Treatment options include manual extraction, topical tretinoin, oral minocycline (in cases with inflammatory infiltrate), electrocauterization, cryotherapy, dermabrasion and surgical excision.

This case is presented to highlight the diagnosis of a benign entity which can be ruled out by a histopathological examination and also the unique presentation of lesions under eyelids with a bilaterally symmetrical distribution.

REFERENCES:

1. Hallaji Z, Akhyani M, Jamshidi S, Modabbernia A et al. Bilateral retro-auricular milia enplaque: A case report and review of the literature. Dermatology Online Journal;16(1):12

2. Sharma R, Kumar B. Milia en Plaque. Indian J DermatolVenereolLeprol 1995;61:365-6

NEWS & EVENTS

1.BDS CME at 25 th January 2015 (Video CME on cosmetology)

2. DERMACON 2015 from 12th to 15th February at TMA Pai International convention Centre,Mangaluru

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