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"DERMADRISHTI"

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Executive Editor - Dr. Praveen Kumar S.

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A note from the presidential desk:

We are happy to continue with the BDS newsletter-“DERMADRISHTI”, which covers all aspects of the activities of our Bangalore Dermatological society, a vibrant branch of the IADVL. This news letter is brought out bianual by the editorial team comprising of Dr. Leelavathy B. and Dr. Praveen Kumar S. I wish all the success to this academic activity and request all the members of BDS to contribute actively to the journal. This is an opportunity for young dermatologists and postgraduates to showcase their talent.

I wish Dr. Leelavathy B. and Dr. Praveen Kumar S. all the very best to take this journal to greater heights.

Dr. R. Raghunatha Reddy

President

Bangalore Dermatological Society

Presidential Message

It is with immense pleasure that I accepted the role of the President ship of Bangalore Dermatological society for the year 2017- 2019.

First of all let me thank, all my senior colleagues and all the members of the BDS for having trust in me to hold this prestigious post and also to host the academically mind boggling CME's, workshops, conferences, etc. I also thank all my colleagues and executive committee members for their support and encouragement.

To continue the good work of all my predecessors, I would like to enhance our goals.

Integrity

1. To enhance professional cohesiveness & to encourage the spirit of fraternity and the collegial spirit

Academics

3. To provide a platform for the upgradation of professional information, standards and skills
4. To guard and enhance professional and ethical standards of the members and thereby enhance the honor of the profession.

Digital

5. To make BDS completely paperless. All communication will be through mails, web site, messages, whatsapp and through other digital media.

Rural camps

6. To improve quality of life of people living in the rural areas through camps and awareness .

Memberships

7. The strength of any organization lies in the membership. So to see to it that every practicing dermatologist in Bangalore becomes a BDS member

CME's

8. To make every CME unique and worth the change.

Postgraduates

9. To encourage every PG student to participate , present cases and papers, compete, research and discuss and win laurels .

Unique programmes

11. To continue the tradition of introducing something unique in every term , eg. Capsules, Hand outs, Research grants etc .

Non dermatology talks

12. To introduce non dermatology talks for the benefit of all members, eg. investment, spirituality, fitness, etc to lead a better life

All this can be done only with the active participation, encouragement and support of all my team members, senior colleagues and the BDS fraternity .

I would like to thank all members for their continued co-operation and to help me make BDS successful, an important arm of IADVL.

Thank you and regards ,

Dr. R. Raghunatha Reddy

President

Bangalore Dermatological Society

Message from the Secretary

I congratulate Dr. Leelavathy B. and Dr. Praveen Kumar S.- on bringing out the BDS NEWSLETTER, highlighting the activities and achievements of BDS. This should serve as an inspiration for those involved with BDS activities and as a motivation for those contemplating to be a part of the same and bring the best out of them.

Long live BDS

Dr. Jagadish P.
BDS Secretary

Chief Editor's message

This year, Bangalore Dermatological Society brought the dermatologists of Bangalore closer, as a family, whilst sparing no effort in nurturing our knowledge of the subject.

The post graduates, the newer members of this organisation, have benefitted from the monthly meets, which were directed at strengthening our understanding of topics such as "Adverse drug reactions" and "Biologicals in the management of Psoriasis" to name a few. The quiz competitions, for the post graduates and the undergraduates managed to keep us on our toes as well.

The BDS team successfully organised the "Back to the ROOTS -2" conference in August 2017, which was attended by more than a thousand delegates from all over India. We also organised a family get together on 26th November at "Clarks exotica resort and spa" to recharge and renew ourselves while, providing an opportunity for us to connect and build stronger relationships with each other.

The BDS team's perseverance, and it's massive presence in IADVL-KN", has been rewarded with the honour of hosting Dermacon in 2019.

I invite contributions to our biannual BDS Bulletin "DERMADRISHTI", from one and all, including innovative ideas, original research and, of course, the occasional, but essential write up that is the perfect amalgam of humour and subject matter. It is the perfect platform for the junior residents to gain visibility and hone their writing skills. I, especially, encourage them to contribute to this magazine.

I would like to sincerely thank the President, the Secretary and EC of BDS, for giving me the opportunity of being the editor of DERMADRISHTI.

Dr. Leelavathy B.
Chief Editor
Bangalore Dermatological Society

Executive Editor's message

Bangalore Dermatological Society is a close knit group of vibrant dermatologists. The IADVL BDS bulletin "DERMADRISHTI" was a concept of Dr. P.M. Sangolli with whom I was associated and we have brought out 9 issues in the past.

This bulletin is a platform which unearths scientific and writing talents amongst dermatologists young and old.

I am happy to be of service as the executive editor to the best of my ability under the able guidance of our Chief Editor Dr. Leelavathi and past Editor Dr. Prabhakar M Sangolli.

We are happy in bringing out the 10th issue of our bulletin and we hope that with continued patronage of all of you we would be able to index the journal in the near future.

I thank our president Dr. Raghunath Reddy, Secretary Dr. Jagadish and all our exective committee members for helping me to serve our Bangalore Dermatological Society.

I wish that this bulletin will reach greater heights in future.

Dr. Praveen Kumar S MD, DNB, MNAMS
Executive Editor
Bangalore Dermatological Society

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1. **Sahana M Srinivas DNB, DVD,FRGUHS (Pediatric dermatology)**
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Introduction

Infections acquired in utero or in the immediate postnatal period play a prominent role in perinatal and childhood mortality. Newborn infections can be congenital or acquired. A number of these infections are typically mild or subclinical for the mother; nevertheless vertical transmission can result in devasting consequences for the infant. Premature and low birth weight infants are particularly predisposed to fetal neonatal infectious disorders.

Congenital Neonatal infections

Infections acquired in utero are under congenital infections, while those acquired around the time of delivery and the immediate postpartum period are termed perinatal infections. The pathogens implicated in maternally transmitted neonatal infections is listed in [Table 1]

Table 1: Infections during pregnancy that can affect the fetus or infant

Viruses	Cytomegalovirus, rubella, herpes simplex virus, varicella zoster virus, parvovirus B19, hepatitis B, hepatitis C, HIV, enterovirus, papilloma virus
Bacteria	Treponema pallidum, mycobacterial tuberculosis, salmonella typhi, Campylobacter, borrelia burgdorferi, Group B streptococcus, E coli, Other gram negative bacteria
Fungi	candida albicans
Protozoa	Toxoplasmo gondii, plasmodium and trypanosoma cruzi

The original TORCH complex was a concept that described a group of clinically similar congenital infections caused by toxoplasmo gondii, rubella virus, cytomegalovirus and herpes simplex virus. [1] The most distinct cutaneous feature of congenital infections is blueberry muffin lesions. Blueberry muffin syndrome was first described in congenital rubella with thrombocytopenia. [2] It is characterized by blue red infiltrative papules and nodules associated with dermal erythropoiesis. [3] They are usually present at

birth or within 24hrs and new lesions rarely appear after 2 days of age. They are observed in association with variety of disorders; usually infectious or neoplastic [Table 2].

Table 2: Causes of blueberry muffin lesions
Dermal erythropoiesis
• Congenital infections
- Toxoplasmosis
- Rubella
- Cytomegalovirus
- HSV
- Coxsackie B2 virus
• Hemolytic disease of the newborn
• Hereditary Spherocytosis
• Twin-twin transfusion syndrome
Neoplastic disorders
• Transitory myeloproliferative disease
• Neuroblastoma
• Langerhans cell histiocytosis
• Congenital leukemia

Congenital infections cannot be diagnosed on the basis of cutaneous symptoms alone. The most clinical sign indicative of congenital infections is the general appearance of the child (decrease in vigour of crying and muscular tone, hepatosplenomegaly, lymph node enlargement, pale skin colour, neurological and other signs revealed by a thorough clinical evaluation).[4]

Table 3: Cutaneous signs of congenital infections

Infections	Time of maternofetal transmission	Age of onset	Cutaneous signs	Diagnosis
Toxoplasmosis (Toxoplasma gondii)	Intrauterine erythropoiesis	At birth or develop during the first few weeks of life	Maculopapular rash, petechiae, purpura, jaundice, blue berry muffin lesions, calcifications	Toxoplasma IgM ELISA
Rubella	Any time	At birth or within the first 24hrs	Blueberry muffin lesions, generalized brown macules, papules, persistent facial rash, recurrent urticaria	Maternal: fourfold rise in antibody(HI, CF, LA) Fetal: positive rubella IgM(ELISA)
Cytomegalovirus	Any time	At birth	Petechiae, jaundice, purpura, blueberry muffin lesions	Tzanck smear, culture(urine, saliva), PCR, cytomegalovirus IgM/ IgG
HSV	Any time Transplacental or ascending infection	At birth or within first 48hrs	Vesicles(single, grouped, disseminated), scars Widespread erosions	Tzanck smear, culture, DIF(vesicles, CSF fluid, conjunctivae, urine), PCR
VZV	Intrauterine, at birth	Onset of lesions between 5 and 10-12 days of life	Generalized varicelliform blisters or scars, grouped zosteriform vesicles	Tzanck smear, detection of viral DNA in PCR, ELISA, IIF, detection of IgA or IgM

Toxoplasmosis

Toxoplasmosis is caused by intracellular protozoa toxoplama gondii. Infection usually occurs through consumption of undercooked meats containing toxoplasma cysts excreted by cats. [5] Incidence of infection is directly and severity proportional to the gestational age of the fetus. Prospective studies that have monitored pregnant women with primary infection have reported fetal infection in up to 25-30% of cases despite treatment. [6] However the incidence of congenital toxoplasmosis in India is reportedly low. [7]

Most infants with congenital toxoplasmosis are asymptomatic initially, although 80-90% may develop eye and neurological disease in later life. [8] The classical triad of congenital toxoplasmosis are chorioretinitis, hydrocephalus and intracranial calcification. Other features include anaemia, jaundice, hepatosplenomegaly, lymphadenopathy and thrombocytopenia. Cutaneous features include a generalized maculopapular rash that generally spares the face, palms and soles. The diagnosis is based on typical clinical features [Table 3], serological studies and occasionally parasite isolation.
Treatment consists of pyrimethamine, sulphadiazine and folinic acid and spiramycin. The prognosis is variable, although infants with CNS involvement have poor prognosis. [9]

Congenital Rubella

Congenital rubella was one of the earliest described vertically infections in newborn infant in 1941. [10] It occurs following maternal rubella infections during the first 16weeks of pregnancy, and rarely when infection is acquired later in gestation. The classical triad of congenital rubella includes congenital cataract, deafness and cardiac defects (especially patent ductus arteriosus). Other features include microcephaly, mental retardation, retinopathy, hepatosplenomegaly, jaundice, and thrombocytopenia. [11] The characteristic cutaneous lesions are described in [Table 3].

Diagnosis is made by detection of rubella specific IgM in serum or oral fluid taken before 3m of age; or by demonstrating persistent IgG in serum taken between 6 and 12months of age. Treatment includes supportive therapy and recognition of potential disabilities. Congenital rubella can be effectively prevented by immunization with live rubella vaccine and universal vaccination is recommended. [12]

Cytomegalovirus

Cytomegalovirus (CMV) is a leading cause of congenital infections and long term neurodevelopmental disabilities among children. CMV affects 0.5-3% of all newborns worldwide. [13] The incidence of congenital CMV in India is estimated to vary between 1.8% and 2.1%. [14] Classical clinical features include prematurity, intrauterine growth retardation, jaundice, hepatosplenomegaly, seizures, microcephaly, intracranial calcification and bluberry muffin lesions on skin [Table 3]. Viral isolation or strongly positive serum IgM anti- CMV antibody is considered diagnostic. Prenatal diagnosis is done by amniocentesis and is most reliable after 21wks gestation or 7 weeks after maternal infection. [15]

Herpes simplex virus infection

Neonatal herpes simplex infection ranges from mild, self limited illness to potentially devasting condition. It is transmitted from the mother during one of the three distinct time intervals, intrauterine (5%), peripartum (85%) and post partum (10%). [16] Primary maternal infection results in a higher incidence of neonatal herpes compared to reactivation disease in the mother.

Intrauterine infection of the fetus i.e. congenital infection is rare (4-5%). Infection in utero may occur as a result of ascending infection through apparently intact membranes, or a potentially as a result of viremia occurring with a primary maternal infection. It is characterized by vesicles, scarring [Table 3], chorioretinitis, microphthalmia, microcephaly and abnormal brain CT findings. [17] Lesions characteristic of epidermolysis bullosa as well as aplasia cutis congenital- like lesions. [18] Perinatally acquired HSV infection presents with three clinical patterns [Table 4].

Table 4: Neonatal herpes simplex infection

	Skin-eye-mouth disease	CNS disease	Disseminated disease	Congenital HSV
Transmission	Perinatal	Perinatal	Perinatal	Intrauterine
Age of onset	1-2wks	2-4wks	1-2wks	At birth
Clinical features	Vesicles, keratitis, conjunctivitis	Letheragy, seizures, poor feeding	Jaundice, coagulopathy pneumonitis, sepsis	Microcephaly. chorioretinitis
Presence of vesicles	100%	50%	20%	Vesicles, scars
Sequelae*	0%	70%	13%	100%

Modified from (Kimberlin et al. 2001; 11483781)

The diagnosis is established by polymerase chain reaction (PCR) which has a sensitivity and specificity ranging from 75 to 100% and 71 to 100% respectively.[19,20] Intravenous Acyclovir is the drug of choice. It is given at the dosage of 20mg/kg 8th hourly for 14days for limited disease and 21days for disseminated infections. The detection of HSV DNA in CSF at the end of treatment is associated with poor outcomes. The acyclovir therapy should be continued in such patients till the CSF PCR becomes negative.

Varicella Zoster infections

Varicella zoster infection during pregnancy may follow three patterns: Fetal varicella syndrome, neonatal varicella and infantile herpes zoster. The manifestation of neonatal varicella during first 10-12 days of life suggests transplacental transmission of the disease. Post natally acquired neonatal varicella presents after 12 days of life. [21] The severity and mortality of neonatal varicella depends on the day of onset of rash in the mother and neonate. [22, 23] Fetal varicella syndrome presents with dermatomal cicatricial areas, limb hypoplasia, ocular defects, CNS abnormalities like microcephaly and seizures.

Maternal varicella in late gestational period may give rise to perinatal infection within 10days of birth, known as neonatal varicella. Risk of infection is greatest when maternal manifestation occurs less tha 5days prior to delivery and 2days after delivery. The infant presents with disseminated vesicular lesions, pneumonia, hepatitis and meningoencephalitis. [24] Maternal varicella acquired between 14-33weeks of gestation may result infant zoster manifesting in the first or second year of life. [25]

Diagnosis is mainly based on clinical features and investigations [Table 3]. Varicella zoster immunoglobulin can be given to prevent neonatal varicella to all infants whom the mother has onset of varicella within 5 days before or within 48hrs after delivery. Intravenous acyclovir is indicated in the treatment of neonatal varicella at the dose of 60mg/kg/day in 3 divided doses for 14-21days. [26]

Congenital syphilis

Congenital syphilis is rare now-a-days. Transmission occurs more frequently during primary or secondary syphilis in the mother than during latent disease. [27, 28] Early congenital syphilis presents with low birth weight, low birth weight, syphilitic rash with desquamation, rhinitis, hepatosplenomegaly, pneumonitis, bony involvement (osteochondritis, periostitis) and CNS involvement. The differential diagnosis includes neonatal sepsis and other congenital viral infections. Late congenital syphilis has several distinctive features including dental abnormalities (Hutchinson's notched teeth) interstitial keratitis, eighth nerve deafness, neurosyphilis especially paresis, bone and joint abnormalities such as frontal bossing, saber shin and painless knee arthritis. [29] Treatment of congenital syphilis is described in [Table 5].

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Table 5: Treatment of infants born to mothers with reactive serological tests for syphilis

Indications	Treatment
• Infant with confirmed diagnosis	Aqueous penicillin G, 50,000 U/kg IV, every 12 h (1 week of age or younger) + every 8 h (older than 1 week) for a total of 10 days.
• Symptomatic infant with presumptive diagnosis	
• Infant RPR/VDRL at least 4 times maternal RPR/VDRL	
• Maternal RPR/VDRL reactive and incomplete maternal treatment (none, or undocumented, or 4 weeks or less before delivery) PLUS infant evaluation abnormal or not done	

Indications	Treatment
• Maternal RPR/VDRL reactive and incomplete maternal treatment (none, or undocumented, or 4 weeks or less before delivery) PLUS normal infant evaluation PLUS infant RPR/VDRL non-reactive or less than 4 times maternal RPR/VDRL	Single dose of benzathine penicillin G, 50,000 U/kg IM
• Infant RPR/VDRL less than 4 times maternal RPR/VDRL PLUS infant evaluation normal PLUS adequate maternal treatment	
• Infant RPR/VDRL non-reactive, PLUS normal infant evaluation, PLUS maternal adequate treatment	No treatment required

Adapted from Report of the Committee on Infectious Diseases, American Academy of Pediatrics, Red Book, 28th Edition, 2009

Candidiasis in newborns

Candidiasis may be congenital or acquired in the neonatal period. Candida albicans is responsible for approximately 75% of neonatal fungal infections. [30] Congenital cutaneous candidiasis is acquired in utero and manifests as diffuse, erythematous papules and pustular eruptions. Palms, soles and nails may be involved but diaper area and oral cavity is usually spared. The lesions generally resolve with desquamation within 1-2wks. [31] Systemic candidiasis occurs in low birth weight infants manifests as burn like dermatitis followed by desquamation and isolated diaper rash with or without thrush. [32]

Neonatal candidiasis is acquired during passage through an infected birth canal or during postnatal period. It manifests after 7 days of life and is localized to oral cavity and diaper area or other intertriginous areas. In extremely low birth infants neonatal candidiasis may present as invasive fungal dermatitis. [33] Cultures of blood, urine and cerebrospinal fluid (CSF) should be done whenever systemic infection is suspected, and in all premature infants. Respiratory distress in immediate neonatal period should arouse the suspicion of systemic involvement. [32]

Intravenous amphotericin B is the first line of drug. Fluconazole 6 mg/kg/day intravenously can also be used if the neonate develops intolerance to amphotericin B. [34]

Acquired Neonatal infections

At birth, the new born is colonized with microorganisms of the birth canal and the outside world. The common acquired neonatal infections are described in Table 6.

Table 6: Common acquired infections in the neonate

Bacterial	Pustular staphylococcal infections Neonatal Impetigo and Staphylococcal scalded skin syndrome Omphalitis Cellulitis Neonatal abscess Circumcision infections
Yeast	Neonatal candidiasis Malassezia furfur infections Neonatal Pityriasis versicolar
Viral	Herpes simplex infections Cytomegalovirus infection Varicella Enteroviral infections
Parasitic	Scabies

Staphylococcal scalded skin syndrome (SSSS)

Potentially life threatening but treatable, toxin mediated manifestations of localized infection with certain strains of staphylococci producing exfoliative toxin A and B. Age of onset is usually between 3 and 16 days and very rarely occurs within 24hrs of birth. [35] It is characterized by tenderness, blistering, mainly over flexures and perioral area, superficial denudation of skin. Mucous membrane is spared. Nikolskys sign is positive.

Blood cultures are usually negative in SSSS. The first line of therapy is Oral/IV Flucloxacillin. In case of Methicillin resistant staphylococcal aureus parental vancomycin is used. SSSS responds well to antistaphylococcal antibiotics and supportive therapy. [36]

Other infections

Omphalitis neonatorum can occur due to improper severing of the umbilical cord, application of oily substances to the umbilical stump and other unhygienic practices during the neonatal period. In most cases gram negative organisms are responsible for omphalitis. [37]

Neonatal pustulosis is caused by Malassezia furfur. It is characterized by presence of non follicular pustules on face and neck in neonates, identification of Malassesia furfur by direct microscopy in pustular material and response to topical ketoconazole therapy. [38]

Scabies is rare in the neonatal period. Congenital scabies has not been reported. Pruritus is often subtle or non-existing. [39] Neonatal scabies presents with papules, vesicles, burrows and very typical vesicles on palms and soles. Mites, ova and faeces can be visualized in potassium hydroxide preparations from skin scrapings. The treatment of choice is 5% permethrin applied for 8 hours. [40]

Conclusions

Neonatal cutaneous infections vary from mild to severe skin involvement with or without complications. Early recognition of skin lesions is important for dermatologists to prevent complications. Counselling parents is important in management of neonatal congenital infections.

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AGEING AND SKIN

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Aging is a process of progressive decrease in the maximal functioning and reserve capacity of all organs in the body, including the skin. Senescence in the skin is a gradual process that ultimately results in the appearance and functional differences that we associate with old age 1. Elderly or old age consists of ages nearing or surpassing the average life span of human beings. 'Senior citizen' or 'elderly' is defined as a person who is of age 60 years or above 2. Elderly patients present to the dermatologists with a wide variety of skin problems: a few are more or less specific to the old age, but most are familiar skin disorders whose clinical expression, physical and emotional consequences and management may be altered by the age of the patient and the problems that increasing age bring with it. Skin also acts as a window for many serious internal diseases.

Skin aging appears as the result of two types of aging, intrinsic and extrinsic aging. Intrinsic structural changes occur as a natural consequence of aging and are genetically determined. Intrinsic aging is the rate of aging that occurs with the passage of time. Extrinsic aging, on the other hand, is the skin's response to external damage and is controllable to a very large degree by the lifestyle choices we make every day.[3,4]

Changing with time	Intrinsic or Chronological ageing	Extrinsic or Photoageing
Fine wrinkles	Yes	Yes
Skin laxity	Yes	Yes
Skin thinning	Yes	No
Xerosis	Yes	Minimal
Coarse wrinkles	Minimal	Yes
Solar lentigenes	No	Yes
Mottled pigmentation	No	Yes
Actinic keratosis	No	Yes

Morpholgical and functional changes in intrinsically aged skin 5

1. Thinning of the epidermis by 10 to 50% → Increased vulnerability, fragility
2. Atrophy of the stratum spinosum → Increased vulnerability, fragility
3. Increased heterogeneity in size of the basal cells → Increased vulnerability, fragility
4. Decreased mitotic activity, increased duration of cell cycle and migration time ↑ decreased desquamation, delayed wound healing
5. Slow replacement of lipids → Disturbed barrier function
6. Flattening of the dermoepidermal junction → Decrease in surface contact area, increased risk of separation by shearing forces
7. Decrease and heterogeneity of melanocytes → Graying of hair, guttate amelonosis, lentigenes
8. Decrease of Langerhan's cells → Diminshed cutaneous immune function
9. Reduction of dermis thickness, decrease of fibroblasts → Reduced strength and ResiliencyAtrophy of the extracellular matrix → Reduced strength and resiliency
10. Reduction and disintegration of collagen and elastic fibres, deposition of → exogenous substances
11. Reduction of cutaneous microvasculature → Reduction of cutaneous vascular responsiveness, disturbed thermoregulation and supply of nutrients
12. Decrease of skin appendages and their function → Decreased lipid and sweat production, disturbed reepi the lialization of deep cutaneous wounds
13. Thinning of subcutaneous fat → Reduced insulation and energy production
14. Reduction of nerve endings → Disturbed sensory function

Morphological changes in extrinsically aged skin5

1. Accumulation of abnormal elastic tissue in the dermis
2. Sparse distribution of collagen fibres, increased collagen degradation
3. Stellate phenotype of fibroblasts and increased biosynthetic activity
4. Increased levels of dysfunctional glycosaminoglycans and proteoglycans
5. Increased number of mast cells and neutrophils
6. Flattening of the dermoepidermal junction, reduction of anchoring fibrils
7. Thickening of the vascular walls of post capillary venules and of arterial and venous capillaries, increased number of veil cells, and marked regression and disorganization of small blood vessels
8. Impaired proliferation, differentiation, desquamation and apoptosis of Keratinocytes
9. Thickening of the epidermis

Glogau scale for grading the severity of photoaging

TYPE	VISIBLE INDICATIONS		
I	No wrinkles	Early photoaging	• Mild pigmentary changes • No keratoses • Minimal wrinkles • Minimal or no make-up
II	Wrinkles in motion	Early to moderate photoaging	• Early senile lentigenes visible • Keratosis palpable but not visible • Paralle smile lines beginning to appear lateral to mouth • Usually wears some foundation
III	Wrinkles at rest	Advanced photoaging	• Obvious dyschromia • Visible keratosis • Wrinkles even when not moving • Always wears heavy foundation
IV	Only wrinkles	Severe photoaging	• Yellow-gray skin tone • Prior skin malignancies • Wrinkled throughout, no normal skin • Cannot wear make-up-cakes and cracks

Throughout the world the population of people aged over 60 years is growing faster than any other age group. It is becoming increasingly important to cater to the health needs of older people.

The skin changes in the geriatric population merits greater study, not only because of the societal tendency to consider skin aging as an important indicator of aging itself, but also as the aging skin could be a marker of underlying, sometimes serious systemic illness.

A thorough knowledge of physiologic and pathologic skin changes in the geriatric population can strengthen the dermatologists ? hand in the management of such cases. Besides this, control of extrinsic factors such as exposure to sunlight and pollution, nicotine use and dietary factors can have significantly positive outcomes in the treatment of the elderly skin including prevention of malignancy.

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Cutaneous adverse drug reactions (CADR): Brief appraisal

Prabhakar M Sangolli, Consultant Dermatologist, Bangalore

Incidence of CADR with certain drugs: 1-3%. Nearly 1/3 need hospitalization.Mortality:1-2 %.ADRs are vastly under reported across health care setting. Nearly 1/3 to ½ of ADRs are preventable

Dose of the drug, **time** course of the reaction, **relevant susceptibility** factors (such as genetic, pathological and other biological differences) will help us in arriving at the correct assessment of CADR .

Maculopapular drug eruptions usually appear with 7-10 days after the administration of offending drug. They are usually itchy and involve palms and soles. Patients will have eosinophilia and abnormal LFT. Biopsy reveals epidermal necrosis with perivascular lymphocyte and eosinophil infiltration. Stoppage of culprit drug and topical steroid application is usually sufficient to manage these patients. Viral exanthema pose diagnostic difficulties in children.

SJS-TEN : Serial SCORETEN is preferable in the management of SJS-TEN. Serological markers like granzyme, perforin, granulysin, glutathione sulfate transferase (GST), lactic dehydrogenase may warn of impending TEN. Genotyping may help in prevention of SJS-TEN.

DICU: Multidisciplinary approach which includes skin care with biological dressings(xenografts and allografts), eye care with lubricants, adequate fluid and electrolyte balance along with care of esophageal and urethral mucosae. Amniotic membrane transplantation works well for eye complications
Early and aggressive therapy with cyclosporin is quite effective. Combination of steroid with cyclosporine,steroids with IVIG, steroids in combination with plasmapheresis and IVIG have been employed with reasonable success.

DRESS: SCAR with long incubation period. Common drugs include anticonvulsants, sulfonamides, DDS, antibiotics. Reactivation of HHV6 andHHV7 may worsen the condition .Drugs may exhibit specific organ involvement, like DDS affecting liver, minocycline heart etc .Multiple drug hypersensitivity is one more risk factor for DHS. One should always assess organ involvement before planning therapy. Steroids may need to be given for 8 to 12 weeks to prevent rapid relapse. Tests to detect delayed auto immune organ damage like AITD,SLE, IDDM,GVHD. may be necessary after 3 months. Genotyping may predict the risk in certain drugs

Erythroderma has rapid onset when is caused by a drug. The scales will be bigger in size. Histopathology reveals perivascular lymphocyte and eosinophil infiltration. The treatment remains the same, irrespective of etiology. Withdrawal of the suspected is the first step in the management

AGEP: Common agents include antibiotics,antimalarials,antifungals like terbinfine,fluconazole, antihypertensives.Viral,bacterial,protozoal infections also can trigger AGEP. Nearly 20 % of patients may have extra cutaneous involvement.

Withdrawal of the offending drug, topical steroids in mild cases and systemic steroids in patients with severe reaction is recommended.

Most of the CADR are of mild nature and can be managed conservatively. However one should identify impending SCARs early, so that prompt treatment is instituted to minimize morbidity and decrease the rate of mortality

Literature scan: JAAD – December 2018

Prabhakar M Sangolli, Consultant Dermatologist, Bangalore

DPP4I :(Gliptins) and DIBP : Outi Varpuoloma et al

Drug induced BP (DIBP) is seen more nowadays due to the introduction of newer therapeutic agents for the management of pain, infections, DM, malignancies etc.. There are 2 types of DIBP i.e, **DIBP proper** which resolves after the withdrawal of offending agent and drug triggered BP which persists as classical even after the suspected drug is withdrawn

Dipeptidyl peptidase 4 inhibitors (DPP4I)s, like sitagliptin, vidagliptin are commonly prescribed in patients with DM for glycemic control. They have been associated with 3 fold increased risk of DIBP. These patients exhibit increased mucosal involvement and known to have lesser eosinophil count. The situation gets complicated as systemic steroids still remain the most effective treatment modality.

Non DPP4Is can be prescribed in patients where DPP4I are implicated in inducing DIBP.

Incidence of ACD in patients with AD : Supriya Rastogi,Kevin R Patel, Vivek Singam, Jonathan I Silverberg

AD patients patients exhibited higher incidence of positive patch test reactions(PPTRs) to ingredient in their personal care products and topical medications.This included fragrances,lanolin,bacitracin, cinnamates,budesonide,tixocortol and chlorhexidine with more than 90% relevance.AD patients also had higher incidence of polysensitization as compared to non atopic controls

Impaired barrier function of skin, immune dysregulation and enhanced use of topical moisturizers,steroids and antibiotics could explain this phenomenon

CADM1 as diagnostic marker in early stage of MF: Akihiko Yuki et al
Cell adhesion molecule 1(CADM1) has been used as diagnostic marker to diagnose adult T cell leukemia and lymphoma. However recent study by authors revealed that CADM1 is also expressed in MF
.

Hence CADM1 can be employed to differentiate **early stage** MF from inflammatory dermatoses like pityriasis lichenoides chronica,lichen planus,drug eruption,discoid eczema and BCC with inflammatory infiltrates.

It was performed with the help of laser microdissection of frozen skin samples and RT PCR

JAAD October 2018

Ethnic differences and co morbidities of 909 prurigo nodularis patients: Emily Boozalis et al

Prurigo nodularis is an uncommon pruriginous dermatoses whose pathogenesis is ill understood. It is believed to be associated with various systemic co morbidities and infections

Literature review suggests following associations

CVS: Hypertension, ischaemic heart disease, Congestive heart failure

RS: COPD

Psychiatric disorders like depression whose incidence 3 and 2.5 times in comparison with AD and psoriasis respectively

Kidney: CKD is significantly associated with PN

Infections associated with PN include HIV and hepatitis C

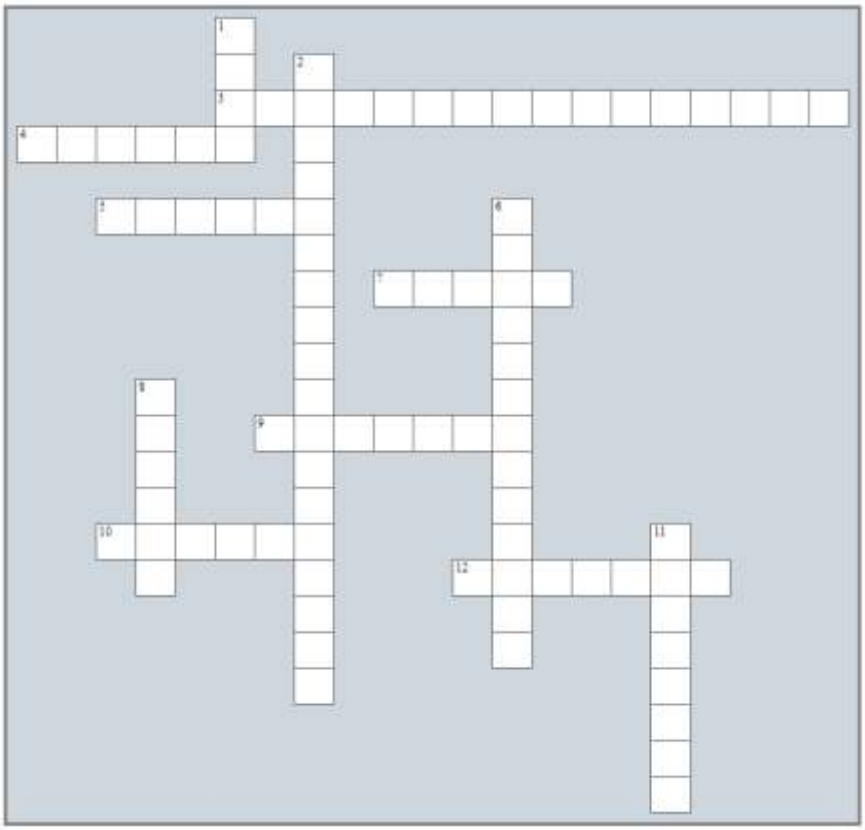
Wound care for Stevens-Johnson syndrome and toxic epidermal necrolysis: Brianna Castillo,Nora Vera, Alex G Ortega-Loayza, Lucia Siminario- Vidal

Dressings employed in SJS and TEN include simple dressings (Ointments or creams covered with bandages) and modern dressngs (Fiber,biologic,synthetic).Biosynthetic dressings offer advantage of changing dressings less frequently. However the efficacy remains the same. Other treatment modalities included systemic steroids and IVIG.

Increased incidence of autoimmune rheumatic diseases in patients with psoriasis: nationwide population based study: Hyun Jeong Ju et al

This study compiled the data generated across Korea on association of auto immune rheumatic diseases(ARD). There was significant co-existence of ankylosing spondylitis,rheumatoid arthritis,Behcet disease, Sjogren syndrome, SLE, systemic sclerosis ,dermatomyositis/polymyositis

Crossword puzzle: Dr. K N Shivaswamy



Across

Strap cells are charecterestic of this condition (16)

Macrophage containing protozoa in a "swarm of Bees" appearance in Leishmaniasis

Corps ronds are also known by these cells (6)

Possible precurssor of extramammary Paget's disease (5)

These elongated cells are seen in Spongiotic dermatitis (7)

Reiter's cells are synonymous with (6)

These cells engulf RBCs, WBCs,Platelets etc and seen in Histiocytic cytophagic panniculitis (7)

Down

Monocyte engulfing the nucleus of intact lymphocyte (4)

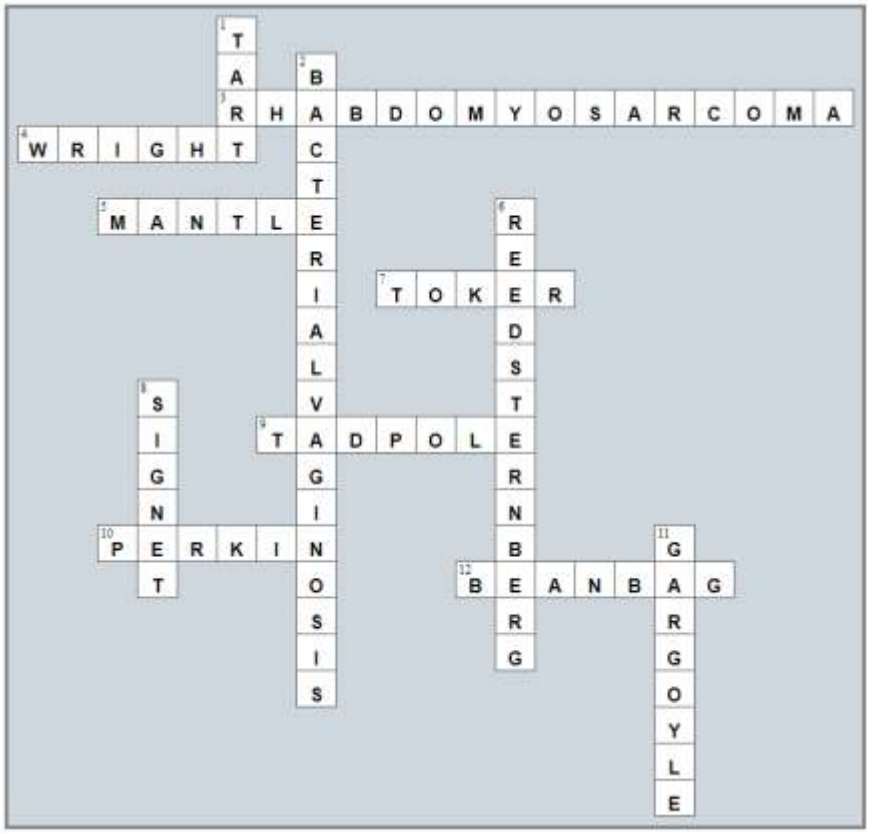
This clue cell gives clue to the diagnosis in (18)

'Owl eyed' cells of Hodkin's lymphoma (13)

These ring like cells are abundant in mucin producing adenocarcinoma (6)

Hurler's syndrome is characterized by this metachromatic cells (8)

Answers to crossword puzzle: Dr. K N Shivaswamy.



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