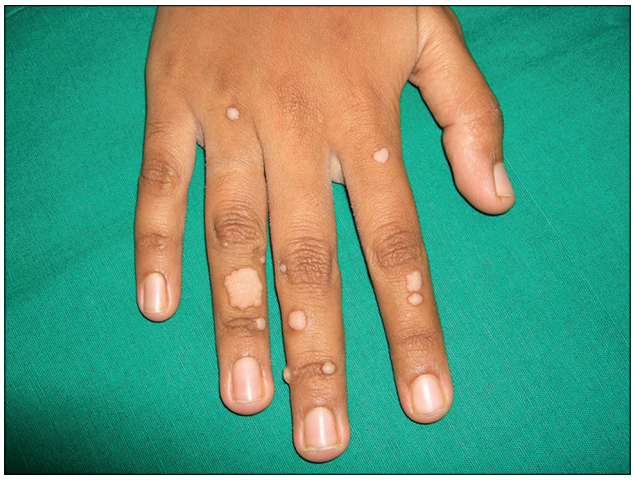
Residents' Corner

Viva questions from the IJDVL

SPOTTER



Spot the diagnosis and enumerate the treatment modalities

What are the morphological variants of warts?

- Common warts
- Periungal warts
- Palmoplantar warts
  Variants - myrmecia, mosaic
- Plane warts
- Filiform and digitate warts
- Anogenital warts.

What is the clinical guide to resolution of warts following treatment?

The best clinical guide to cure is the restoration of normal epidermal texture, including the epidermal ridge pattern where appropriate.

What are the histological features of verruca vulgaris?

The salient histological features of verruca vulgaris are:

- Hyperkeratosis, acanthosis, papillomatosis
- The rete ridges are elongated and are bent inwards at the periphery of the verruca
- Foci of vacuolated cells called koilocytes are located in the upper stratum malphigii and in the granular layer. The koilocytes possess a small round deeply basophilic nucleus surrounded by a clear halo and pale staining cytoplasm. They contain no keratohyaline granules
- Vertical tiers of parakeratotic cells are seen often located at the crests of papillomatous elevations of rete malphigi overlying a focus of vacuolated cells
- Granular cells are absent overlying the papillomatous crest but are increased in number and size in the intervening valleys and contain heavy, irregular clumps of keratohyaline granules
- Dilated capillaries in the papillary dermis and small areas of hemorrhage in the thickened horny layer at the tips of vertical tiers of parakeratotic cells may be seen.

How do you differentiate a plantar wart from a corn/callosity?

- Paring the surface with a scalpel demarcates the abrupt separation between wart tissue and the protective horny ring as the epithelial ridges of the plantar skin are not continued over the surface of the wart. If paring is continued, small bleeding points which are the tips of the elongated dermal papillae are noted.
- Dermatoscopy can also help to distinguish a plantar wart from a corn or callosity, where a wart would show homogenous red dots and globules accompanied with red linear vessels with interrupted skin lines that become prominent after trimming.

What are the modalities of treatment of verrucae?

The treatment modalities are outlined in Box 1.

What are the indications of *Mycobacterium indicus pranii (Mycobacterium w)* vaccine?

Mycobacterium indicus pranii (previously known as *Mycobacterium w*) is a nonpathogenic, rapid growing atypical *mycobacterium* belonging to group IV of the Runyon classification. Killed *Mw* vaccine has a strong antigenic response and is capable of generating a robust cytokine response (interleukin-2 [IL-2], interferon-γ and T-cell responses). Its uses in dermatology are:

- It is approved as an immunotherapeutic adjunct to multidrug therapy of multibacillary leprosy in India

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Box 1: Treatment modalities for warts

Continuous duct tape occlusion

Topical modalities
- Salicylic acid
- Tretinoin
- Podophyllin
- Caustics such as monochloroacetic acid, trichloroacetic acid, cantharidin, phenol, silver nitrate
- Formaldehyde
- Glutaraldehyde
- 5 fluorouracil
- Photodynamic therapy

Surgical/cytotoxic therapies
- Cryotherapy
- Laser ablation
- Intralesional bleomycin
- Electrocautery
- Surgical excision

Topical immunotherapeutic modalities
- Contact sensitizers-dinitrochlorobenzene, squaric acid dibutyl ester, diphenylcyclopropenone
- Imiquimod
- Intralesional interferon

Oral immunotherapeutic modalities
- Zinc suphate
- Levamisole
- Cimetidine

Intralesional antigens
- MMR
- Skin test antigen - mumpus, candida, trichophyton
- BCG vaccine
- Candida antigen

Auto-inoculation therapy

Hypnotherapy

BCG: Bacillus Calmette-Guerin, MMR: Measles, mumps, rubella

- It induces a strong nonspecific inflammatory response against human papillomavirus (HPV) infected cells and has been used intralesionally for the management of genital warts and has been tried with good results in the management of long standing refractory extragenital cutaneous warts.

How does auto-inoculation therapy help in the treatment of warts?

Auto-inoculation activates a delayed hypersensitivity response to the wart tissue antigens, aiding clearance of both local and distant warts.

This therapy has shown to be associated with the production of Th1 cytokines. Th1 cytokines tumor necrosis factor-α and IL-1 downregulate the transcription of HPV genes whereas IFN-γ and IL-2 stimulate cytotoxic T-cells and natural killer cells to eradicate HPV-infected cells.

PITYRIASIS ROSEA

What are the etiological factors in pityriasis rosea (PR)?

Pityriasis rosea has been etiologically associated with viral infections, especially human herpes virus-6 and -7, recent upper respiratory tract infections and several drugs, including barbiturates, clonidine, captopril, omeprazole, imatinib, isotretinoin, D-penicillamine, terbinafine, arsenicals, bismuth, and gold compounds.

What is "atypical pityriasis rosea"?

Atypical PR refers to cases presenting with atypical morphology or distribution of lesions and may be seen in approximately 20% of patients. The various atypical types include:

- Atypical morphology of lesions: Vesicular, purpuric, papular, hemorrhagic, urticarial
- Atypical size of lesions: PR gigantea of Darier
- Atypical distribution of lesions: Inverse PR (lesions on the extremities, flexural areas, and face), limb-girdle type (the eruption is restricted to the shoulders or hips), unilateral or localized (limited to a small area, such as the axilla or breast)
- Atypical number of lesions: Pityriasis circinata et marginata (fewer and larger lesions often localized to the axillae or inguinal region)
- Atypical site of lesions: Involvement of rare sites: finger/toe tips, eyelids, penis, oral cavity
- Atypical severity of symptoms: Severe itch, pain, and burning sensation
- Atypical course of the eruption: Recurrent or relapsed cases. Recurrent episodes have been estimated to occur in up to 3.5% of cases.

PHOTOTHERAPY

What is the mechanism of pigment induction by psoralen plus ultraviolet A in vitiligo?

The exact mechanism of pigment induction by psoralen plus ultraviolet A (PUVA) in vitiligo is still speculative. Psoralen may stimulate melanogenesis by the following mechanisms:

- Photoconjugation of psoralens in melanocyte DNA leads to mitosis, replication and proliferation of melanocytes, increased number

of melanosomes and their further transfer to keratinocytes
- Stimulation of cyclic AMP activity by PUVA leads to increased synthesis of tyrosine
- PUVA may induce a suppressor T cell population and release IL-10 which is important for differentiation and activation of T regulatory cells which may suppress the auto-immune stimulus responsible for melanocyte destruction
- PUVA induces basic fibroblast growth factor and hepatocyte growth factor which may aid in regrowth and migration of follicular melanocytes to the basal layer of skin.

Enumerate the techniques by which psoralen plus ultraviolet A can be administered.
- Ultraviolet A (UVA) treatment units are available in clinics and hospitals and are equipped with fluorescent bulbs (wavelength: 320-380 nm).
- PUVASOL (psoralen and UVA from sunlight) is advised for those patients who cannot visit the hospital for phototherapy. The best time of the day for PUVASOL is between 9.15–11.15 a.m. and 2.30–3.30 p.m.
- Modifications of PUVA include clothes-on PUVA, PUVA soaks, bath PUVA, turban PUVA and PUVA combs.

What specific precautions should be taken to prevent eye toxicity during psoralen plus ultraviolet A therapy?
Wrap around ultraviolet-blocking glasses which give complete UVA photoprotection like B2 Toric and Green 80 should be worn when the patient is exposed to sunlight, from the time methoxsalen is ingested until sunset on the same day.

FINASTERIDE AND DUTASTERIDE

What are the differences between finasteride and dutasteride?
- Finasteride selectively inhibits the type II isoenzyme of 5α reductase (the enzyme that is responsible for converting testosterone to dihydrotestosterone [DHT]). Dutasteride inhibits both type 1 and type 2 5α reductase.
- Dutasteride is about 3 times as potent as finasteride in inhibiting type II 5α reductase and more than 100 times as potent at inhibiting the type 1 5α reductase enzyme. Hence, finasteride is capable of reducing the serum DHT level by about 65% while dutasteride is capable of reducing the same by about 90%.

MASTOCYTOMA

What is the etiopathogenesis of mastocytosis?
- Increased local concentrations of soluble mast cell growth factor in lesions of cutaneous mastocytosis are believed to stimulate mast cell proliferation, melanocyte proliferation and melanin pigment production
- Upregulation of the apoptosis preventing protein Bcl-2 is postulated to cause impaired mast cell apoptosis
- Activating mutations of the proto-oncogene c-kit have been identified.

What are the types of cutaneous mastocytosis?
- Cutaneous mastocytoma
- Urticaria pigmentosa
  Variant – pseudoxanthomatous mastocytosis or xanthelasmoidea
- Diffuse cutaneous mastocytosis
  Variant – erythrodermic mastocytosis
- Telangiectasia macularis eruptiva perstans
- Bullous mastocytosis.

What is the Darier sign?
In cases of mastocytosis, the increased number of normal functioning mast cells degranulate in response to gentle rubbing leading to erythema and edema at the site which occurs within 2–3 min and disappears within 15 min to hours. This response is called Darier sign.

What are the special stains for mast cells?
- Toulidine blue
- Giemsa
- Leder's chloroacetate esterase
- Immunohistochemical stains including CD2, CD25, CD117 and mast cell tryptase.

What are the criteria for diagnosis of systemic mastocytosis?
One major and 1 minor criterion or 3 minor criteria are needed to establish a diagnosis of systemic mastocytosis.
- Major criteria: Multifocal dense infiltrate of mast cells in the bone marrow and/or other extracutaneous organs

- Minor criteria:
  - Baseline total tryptase level of greater than 20 ng/ml
  - Greater than 25% of mast cells in the bone marrow aspirate smears or tissue biopsy sections having spindled atypical morphology
  - Mast cells in bone marrow, blood, or other lesional tissue expressing CD25 or CD2
  - Detection of a codon 816 c-kit point mutation in blood, bone marrow or lesional tissue.

CHROMHIDROSIS

What is chromhidrosis?

Chromhidrosis is secretion of colored sweat.

- Apocrine chromhidrosis: It has been demonstrated that lipofuscin pigment that is produced by apocrine glands is responsible for coloured sweat. Colour of the apocrine sweat can be brown, yellow, green, blue or black depending upon varying concentration and oxidation states of lipofuscin granules. Darker color is because of higher states of oxidation
- Eccrine chromhidrosis: It occurs due to excretion of water-soluble pigment from the eccrine glands which may occur after ingestion of certain dyes and drugs
- Pseudochromhidrosis: It is said to occur when colorless eccrine sweat becomes coloured on the surface of the skin as a result of extrinsic dye. Clothing, paints and chromogenic bacteria.

BULLOUS PEMPHIGOID

What are the clinical variants of bullous pemphigoid?

- Localised variants
  - Pretibial pemphigoid
  - Around stomas
  - Vulvar region in women and perianal region in male
  - At the site of irradiation or confined to a paralysed limb
  - Isolated palmoplantar involvement (dyshidrosiform pemphigoid).

- Generalized variants
  - Dermatitis herpetiformis like (vesicular pemphigoid)
  - Erythrodermic BP
  - Lichen planus pemphigoides
  - Gestational pemphigoid
  - Pemphigoid incipiens (elderly patients with itchy BP lesions but a negative direct immunofluorescence in the presence of circulating anti-BP 180 antibody)
  - Prurigo nodularis-like pemphigoid (nodular pemphigoid).

LICHEN PLANUS PIGMENTOSUS INVERSUS

What is the differential diagnosis of pigmentary abnormalities that can be localized to the flexural areas?

- Acanthosis nigricans
- Erythrasma
- Pityriasis versicolor
- Reticulate pigmented anomaly of the flexures
- Postinflammatory hyperpigmentation
- Candidal intertrigo
- Fixed drug eruption
- Lichenoid toxic dermatosis
- Ashy dermatosis
- Lichen planus inversus.

DISSEMINATED CUTANEOUS GLOMUVENOUS MALFORMATION

What are glomus tumours? Enumerate the types.

Glomus tumors are thought to represent neoplastic proliferations of glomus cells, which are modified smooth muscle cells located in the walls of the Sucquet–Hoyer canal, a specialized arteriovenous anastomosis found most often in the fingers. They play an important role in thermoregulation. Glomus tumors can be subdivided into localized, disseminated and congenital plaque type forms.

What are the common sites for glomus tumours?

The commonest site is the hands, particularly the fingers, followed by other sites on the extremities, the head, neck and penis. Tumours beneath the nail are particularly painful and the affected nail has a bluish-red flush. Glomus tumours may also involve internal organs.

Enumerate treatment modalities for glomus tumours.

Simple excision can be done for symptomatic lesions. Other modalities include argon and carbon dioxide laser therapy, electron beam radiation, and sclerotherapy with hypertonic saline or sodium tetradecylsulfate.

REVERSAL OF PSEUDO-AINHUM WITH ACITRETIN IN CAMISA'S SYNDROME

What is ainhum and pseudo-ainhum?

The term ainhum is applied to a painful constricting band, classically around the base of the fifth toe, in adults, which may involve only the skin, or may extend more deeply involving the fascia or bone, and can result in spontaneous amputation. It is most common in black Africans.

Pseudo-ainhum is the term applied to other constricting bands around digits or limbs which may be congenital or secondary to other diseases like infection (particularly leprosy), recurrent trauma, cold injury, neuropathy (especially congenital sensory neuropathy), systemic sclerosis or chronic psoriasis. Pseudo-ainhum may also occur in association with certain hereditary diseases such as Vohwinkel's palmoplantar keratoderma, pachyonychia congenital, erythropoietic protoporphyria, and Olmsted's syndrome.

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