BIODERMA
LABORATOIRE DERMATOLOGIQUE

INTERNATIONAL MEDICAL

BIODERMA Congress Reports
RADLA 2023

Reports written by

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Dermatologist, Brazil

RADLA - This is the Annual meeting of dermatologists from Latin America that took place in the end of May, in Curitiba, Brazil.
It is with deepest pride and greatest pleasure that I welcome you to join us in the highlights of this great meeting!
Lilia Guadanhim, MD PhD – Dermatologist from São Paulo, Brazil

Pediatric Dermatology

Speakers: Johanna Peceros, Maria Cecília da Matta Rivitti Machado, Maria del Carmen Boente, Ana Maria Mósca de Cerqueira, Margarita Larralde, Felipe Velásquez Valderrama, Vania Oliveira de Carvalho, Rosalía Ballona, Kerstin Taniguchi Abagge, Rosa Inés Castro Rodríguez and Reynaldo Alberto Pomar Morante

Mastocytosis

Clinical manifestations:
· Pigmented Urticaria
· Lonely Mast cell tumor
· Eruptive macular telangiectasia perstans
· Diffuse cutaneous mastocytosis
· Mast cell activation syndrome – facial flushing and systemic symptoms (rash, fainting, seizures and cognitive decline), no cutaneous lesions

Diagnosis:
· Darier sign – pathognomonic
· Cutaneous biopsy with mast cell staining
· In case of doubt: immunohistochemistry CD2, CD 25, CD 30, CD 117. Treatment:
· Mainly antihistamines H1 and H2.
o H1: cetirizine
o H2: famotidine, omeprazole – always associated to H1.
· Mast cell membrane stabilizers (sodium cromoglicate)
· Leukotriene inhibitors

· Others: aspirin, UVA, corticosteroids, laser, epinephrine Prognosis:
· Excellent in 90% of the children, with spontaneous regression.
· 10% of the cases might be persistent or associated to systemic mastocytosis.
· Investigate systemic mastocytosis:
o adolescents with cutaneous lesions.
o Persistent elevation of serum Tryptase> 20ng/mL and hepatosplenomegaly

Non-Infectious Neonatal pustulosis

Evaluation:
· History: Perinatal, Family and maternal
· Clinical examination, associated lesions or anomalies
· Work up: Cutaneous biopsy, Direct smear, Culture
· Infectious causes: Candidiasis, impetigo, scabies, varicela, herpes, listeriosis, dermatofitosis.
· Non-infectious causes: toxic neonatal erythema, pustular transient melanosis, benign cephalic pustulosis, neonatal acne, miliaria, infantile acropustulosis, pustular eosinophilic pustulosis, super IgE syndrome, pigmentary incontinence, histiocitosis, transient myeloproliferatie disorder associated to Down Syndrome, DIRA.

Toxic Neonatal Erythema
· Erythematous macules, 2-3cm, with central papules or pustules
· Does not affect palms and soles
· Diagnosis: clinical, Giemsa with many eosinophils
· Treatment: not needed, spontaneous resolution within 1-2 weeks.

Pustular transient melanosis
· 0,2 – 4% term newborns
· Fragile pustules since birth, single or multiple lesions
· 3 stages: pustules, scaly collar, residual pigmentation
· Diagnosis: clinical, histology with many neutrophils
· Treatment: not needed.
· Neonatal sterile transient pustulosis: overlap between Pustular transient melanosis and Toxic Neonatal Erythema

Benign cephalic pustulosis
· Very frequent (10-66%)
· Associated to *Malassezia sympodalys* , M. furfur, M. globose
· Multiple erythematous papules and pustules in face and scalp.
· Starts in 3 or 4 weeks of life.
· Diagnosis: clinical, KOH shows spores
· Treatment: spontaneous resolution in a couple of weeks, topic imidazoles if needed.

Neonatal Acne
· More frequent in male babies (5:1)
· Since birth and during the first year of life
· Comedones, papules and pustules.

Infantile Acropustulosis
· Chronic and recurrent dermatosis.
· Unknown etiology
· First weeks or months of life.

- Pruritic, papules and pustules in palms and soles
- Lesions last for 1 week and recur after 15-30 days, and after with longer intervals.
- Spontaneous resolution after 2 or 3 years.
- Diagnosis: clinical, recurrence patter is key, Giemsa with multiple PMN
- Treatment: topical steroids, oral anti histamines

Pustular eosinophilic pustulosis
- Recurrentand pruritic dermatosis
- Perifollicular papules and pustules over erythematous basis.
- Face and scalp, upper trunk and extremities
- Evolves in crisis.
- Spontaneous resolution in months
- Diagnosis: Clinical, smear with eosinophils
- Treatment: topical steroids, oral anti histamines.

Hiper IgE syndrome
- Immunodeficiency: recurrent infections (mucocutaneous candidiasis, otitis media, pneumonia with pnematocele), dermatitis, high IgE levels, eosinophilia
- Genetics: either STAT3 (autossomic dominant) or PGM3, DOCK8 (autossomic recessive)
- Starts during neonatal period
- Eczematous lesions, papules, vesicules and pustules, intensively pruritic
- Persistent evolution.
- Associated to dental anomaly, characteristic facies.

Transient myeloproliferative disorder associated to Down Syndrome
- Rare, associated to GATA1 mutation.
- Anormal transient Myelopoiesis
- Almost exclusive of patients with Down Syndrome and patients with mosaicism for 21 trisomy.
- Since the first days of life.
- Asymptomatic vesicles and pustules that evolve with a crust.
- Diagnosis: histology with immature myeloid infiltrate, peripheral blood with blasts.
- Treatment: not needed, spontaneous resolution in 1-2months.

DIRA
- Autoinflammatory disease due to deficiency of the receptor antagonist of IL-1
- Cellular hypersensitivity to this pro-inflammatory IL – systemic inflammation.
- Clinics:
  - Sterile Pustules, ichthyosiform desquamation, nail pitting
  - periostitis and osteomyelitis, osteolytic lesions, pain and swelling.
- Diagnosis: : Anakinra (antagonist of the receptor of IL-1).

Dermoscopy

Speakers: Nuria Ferrera, Bollea Garlatti, Luis Agustin, Lídice Dufrechou, Alejandra Larre, Borges Renato, Marchiori Bakos, Maria Sofia, Nicoletti Carolina, Spinelli Lídice, Dufrechou Blanca, Carlos Ortega and Giselle Claros

- Sptizoid lesions: mimic melanoma in clinic and histology.
- Spitz and Reed: same spectrum, reed is pigmented.

· Nodular Spitz nevi: excision.
· Spitz/Reed nevi with starburst pattern: always excision in children over 12yo.
· Spitz/Reed nevi with symmetric starburst pattern in children <12yo: observe.
· Acral nevi in children:
o Size is not criteria for malignancy in congenital nevi.
o Pigmentation Does not affect acrosyringes
· Melanonychia in children:
o Nevi are the most common cause: 48-94%
o Lentigo are the second cause: 30-67%
o Melanoma: really rare
o If multiple nails: evaluate extrinsic or systemic causes
o If one nail: history + examination, dermoscopy + photography.
§ If red flags (rapid growth, big anatomy change, extensive periungual dissemination and pigment progression, bleeding or pain): biopsy of nail matrix.
§ if not, re-evaluate in six months
· Melanomas in childhood:
o 50% de novo
o 50% on preexisting nevi (80% congenital nevi).
o Mostly mistaken for banal benign lesions, specially pyogenic granuloma (always look at histology of excised pyogenic granuloma in children!)
· Nevi in every age is a risk factor for melanoma.
· Genital nevi in children are of greater risk if associated to lichen esclerosus
· Scalp nevi: special patterns – eclipse, cocarde, regression is possible.

The second day of RADLA was cloudier and colder but the science keeps on! Let's dive in the subjects of the day!.

Alopecia Areata (Plennary)

Antonio Massa (Portugal)

· Topical treatment options:
o Tretinoin
§ Massage until complete absorption
§ Avoid eyelids and lips
§ If irritation, stop using in the area and associate topical steroid.
§ Adapalene – increased specificity of retinoid acid receptor, off label use in Alopecia areata.
o Anthraline
§ Anthraline 3% + salicylic acid 5% + petrolatum
§ Keep the formula in fresh environment
o Topical steroids – clobetasol
§ Considered first line: low cost
o Minoxidil
o Topical calcineurin inhibitors
o Cryosurgery
o Bexarotene

· Oral treatment options:
o Steroids – prednisolone up to 20mg

§ Tapering 20-15-10-5mg early morning
o Finasteride, Dutasteride
o Methotrexate + Folic acid
§ 7,5 to 20mg once a week
§ Folic acid one day before and after MTX.
o Oral minoxidil 0.5 to 5mg
§ 18-82% objective clinical improvement
§ Adverse events: hypertrichosis, postural hypotension.
§ Safe and successful treatment
o Anti depressants

· What's new:
o Baricitinib:
§ Good efficacy and safety profile
§ High cost
o Platelet rich plasma
§ Unclear whether PRP is better than triamcinolone alone.
§ Very low quality of evidence – inconsitency, imprecision and risk of bias.
§ No serious adverse events.
o Expert consensus:
§ 50 hair experts from 5 continents.
§ First round: agreement >66% in only 5% of the questions.
§ Greater consensus for intralesional treatment and topical treatment.

The challenges of acne treatment in Latin America

Marco Rocha (Brazil), Patricia Troiellli (Argentina), Leonel Fierro Arias (Mexico)

Adult female acne:
· Topical treatment from beginning to maintenance:
o retinoids, PBO, antibiotics, antiandrogens
o dermocosmetics: sensitive skin, skincare, photoaging.
§ Higher satisfaction and adherence
§ Actives that target inflammation, seborrhea and pigmentation
§ Make up is beneficial
§ Sunscreen always – 81% of reduction of pigmented spots with use of sunscreen for 8 weeks
§ Skin barrier matters.
§ Prefer syndets
§ Moisturizers are key.
· Hormonal workup is NOT necessary in 80% of cases. Only with other signs of hyperandrogenism.
· Systemic treatment:
o Start with antiandrogen and oral contraceptives, Spironolactone.

· Male transgender patients:
o Always have prolonged hormonal therapy in mind!
o Supraphysiological levels of testosterone
o Active ovaries
o Higher risk of hepatotoxicity and dyslipidemia.

- Dysphoria.
- More frequent blood exams
- Don’t forget pregnancy tests.

· Oral Isotretinoin:
- Low dose <= 0.5mg – lower adverse events
- No reason for stopping at 150mg/kg – patient improvement is the key for suspension, not cumulative dose
- Always recommend topical maintenance treatment for at least 1 year - reduces relapse.

· Diet:
- Negative impact of high glycemic load food, dairy
- Vegetables and fruits are beneficial

· Probiotics:
- Still low evidence for prescription
- Probably promising to be part of daily routine

· Scarring:
- Always: inform, prevent and avoid causes of prolonged inflammation.
- Investigate: family history, duration of acne, skin picking, presence of atrophic scarring.
- Early treatment even in mild cases reduces the risk of scarring

· Hyperpigmentation:
- Higher risk: acne severity, prolonged inflammation, higher phototypes
- Stigmatization prolongs the impact of the disease.
- Patients with hyperpigmentation are seen as less confident, successful and happy.
- Alternatives:
  - Retinoids – alone or in fixed combination
  - Azelaic acid 15-20%
  - Hydroquinone– alone or in fixed combination
  - Niacinamide
  - Thiamidol

Hemangiomas

· Usually grow up to 24 months and start to involute.
· Prolonged proliferative phase: deep and segmentary hemangiomas, head and neck.
· Prolonged Involutionary phase: deep hemangiomas
· Complications:
- Ulceration:
  - Mostly 4months
  - 16-29% of cases
- Functional compromise:
  - Periocular: astigmatism, lacrimal duct obstruction, amblyopy, squint
  - Beard area: airway obstruction
  - Perioral: dentition delay, lactation alteration.
- Hypothyroidism:
  - Hepatic or extensive hemangiomas
  - Overexpression of 3-iodine-thyronine-deionidase : consumption hypothyroidism
- Cosmetic Sequelae:
  - Risk factors: mixed, centrofacial, thoracic, mammary, abrupt boarders, cobbled surface.
  - Abrupt boarders: 80% of sequelae

§ Cobbled surface: 62% of sequelae
§ Telangiectasia (36-86%), anetoderma (47-68%), excessive skin (16%), scarring (5-13%), fibroadipose tissue (36-86%).

Off we go to the third day of RADLA – Annual meeting of the dermatologists from Latin America, with focus sessions on important topic! Come on aboard!

New Advances in Vitiligo Management

Jose Luis López Estebaranz (Spain)

· Latest guidelines (2022) include topical/oral corticosteroids, topical calcineurin inhibitors, phototherapy and targeted light therapy.
· Pathogenetic causes:
o Genetic background: polygenic (6% risk if 1st grade relative, 23% if twin affected).
o Dysfunctional biochemical pathways: IFN-□-CXL10-axis.
o Autoimmune processes: innate and adaptive immune system.
o Melanocyte adhesion deficits
o Nervous system imbalances: segmental vitiligo.
· Vitiligo induced by drugs:
o Immune checkpoint inhibitors: BRAF inhibitors.
o Biologics: TNF-□, Il-17, IL-12/23 inhibitors, dupilumab.
· Autoimmune comorbidities:
o 23% of patients had at least 1 autoimmune condition in a 10-year retrospective study of patients with vitiligo.
o Thyroid disease, psoriasis, rheumatoid arthritis, alopecia areata, inflammatory bowel disease, systemic lupus erythematosus, diabetes mellitus type 1.
· Quality of Life impact:
o Impaired QoL regardless of the location.
§ 29% of patients have depression.
§ Vitiligo patients are almost 5x more likely to be depressed than healthy controls
§ 91% of patients report stigmatization.
§ 1/3 of patients are at risk for suicide.
o DLQI is associated with the extent of disease.
§ Face and extremities: self-consciousness
§ Genital, chest and back: sexual dysfunction.
o Itching and/or burning of skin occurs in 1/3 of patients and is associated with BSA >25%.
o Therapeutic Targets:
§ Melanocyte stress – Antioxidants?
§ Autoimmune destruction:
· Topical immunosupressants
· Phototherapy
§ Melanocyte regeneration:
· Surgery
· Phototherapy
o All combination therapies ranked higher than NB-UVB monotherapy. Preferred therapeutic approaches:
§ NB-UVB + Er:YAG laser+ topical 5% 5-FUI
§ NB-UVB + needling/microneedling

§ Tacrolimus ointment + NB-UVB
· could activate both hair follicle and dermal melanocyte precursors.
· More effective than phototherapy alone, especially in facial and proximal limbs lesions.
§ Fractional laser + UVB/MEL
§ Afamelanotide:
· Synthetic analog to MSH
· Stimulates melanocyte production and function.
· Works better in combination with phototherapy
§ Bimatroprost and Latanoprost
· Analog to prostaglandin F2□
· Stimulates melanocytes and tyrosinase.
· Bimatoprost 0.03% twice daily + UVB-NB
§ □5□1 and MIA:
· MIA = melanoma inhibitory activity
· Vitiligo seems to be caused by the interaction between MIA and the integrins □5□1 in the melanocyte.
· MIA is able to break the linkages between integrins and melanocytes, what leads to detachment of melanocytes, that desquamate with keratinocytes – no inflammation and little to no immune system activation.
· Topical alternatives with this target.
§ Janus kinase inhibitor
· Tofacitinib :
o requires light exposure to stimulate melanocyte regeneration - low-level light.
o Monotherapy may lead to repigmentation.
o Suppresess T cell mediators in vitiligo
· Baricitinib:
o Jak1/2 inhibitor.
o Combined with NB-UVB.
o Good alternative and supplementary to treat vitiligo
· Ruxolitinib:
o Low nanomolar potency selective for Jak1 and 2, without significant inhibition of non-JAK kinases.
o Melanocyte stress leads to immune cell activation of CD8+ T-cells that target and destroy melanocytes. These T-cells also release IFN-□,
which activates the JAK-STAT pathway in keratinocytes, leading to production of CXCL-9 and CXCL-10.
o Continued signaling by IFN-□ and CXCL-9/10 attracts more autoreactive T-cells, perpetuating an inflammatory cycle of melanocytes.
o Ruxolitinib 1.5% cream BID.
o Acne and pruritus are the most commom adverse events
· Ritlecitinib:
o Selectively inhibits JAK3 and the members of TEC family of kinase3.
o 50mg daily

Light and Laser in Rosacea

Jose Luis López Estebaranz (Spain)

- Ethiopathogenesis: genetic + environmental componentes.
- Mechanisms:

- Deviant neurovascular signaling
- Dysregulation of the innate immune system
- Colonization of microorganism that stimulate innate immune system.

- Comorbidities:
- Depression – OR 4.81
- Migraine – 12% (general), 59% (in patients with ocular rosacea)
- Cardiovascular – be aware of atherosclerosis, hypertension, hyperlipidemia.

- Topical treatments:
- Ivermectine 1%
- Metronidazole 0.75%
- Azelaic acid 15%
- □-adrenergic agonists: Brimonidine and oxymetazoline
- Novel therapies: minocycline foam, timolol, tranexamic acid, botulinum toxin.

- Systemic Treatment:
- Doxycicline 40mg
- Doxycicline, Minocycline 100mg.
- Limecycline 150-300mg
- Oral isotretinoin 0.25-0.5mg/kg

- Laser and IPL:
- Target vascular component: telangiectasias, vessels, erythema and flushing.
- Anti-inflammatory properties.
- Dermis collagen remodelling
- Alternatives: PDL, KTP, Diode, Pro-yellow(577nm), Nd:YAG, IPL.

- IPL:
- Decrease periannexial inflammatory infiltrate
- Blue UV light has anti-inflammatory effects
- 83% improvement in flushing, erythema and skin surface
- 64% reduction in flares
- 1-7 treatments.

- 577nm pro yellow:
- Reduces density of demodex.

- Fluorescent light energy:
- Biophotonic platform with a multi light LED lamp + chromophore containing gel.
- Useful in burning/stinging symptoms.

- Ocular rosacea:
- Marginal lid telangiectasias, “honey crusts” and collarette in the eyelashes margin.
- Treatment: doxycycline 40mg/day, topical azithromycin, cyclosporin drops, IPL (field effect, meibomian eye disfunction).

- Phymatous lesions:
- Ablative lasers: Co2, Erbium.

- Skincare:
- Integrity of skin barrier
- Gentle moisturizer
- Amphoteric cleanser, micellar water, termal spring water.
- Sun protective creams
- Make up: cover up, color correcting powders

Today was the last day of RADLA but there was still time for some pearls! Hope you’ve enjoyed this journey!

Duel of Titans

Case 1

· History: 5-year-old patient with papulonodular lesions that ulcerate and heal in 2 to 10 days leaving atrophic scars. The lesions started when the patient was 2 years old. When new lesions happen, they are accompanied by fever, lymphadenopathy and malaise.
· Histopathology: perivascular and periannexal band infiltrate with moderate atypia and epidermal ulceration. Positive for CD3, CD4, CD30 (focal). Negative for CD8.
· Lab: mild lymphocytosis and anemia, IgM and IgG for EBV positive (0.96 / 62.98)
· Diagnosis: EBV infection with hypersensitivity to mosquito bites.

Hypersensitivity to mosquito bites as the primary clinical manifestation of a juvenile type of Epstein-Barr virus associated natural killer cell leukemia/ lymphoma.
· Described mainly in Japanese patients within the first 2 decades of life.
· Skin lesions at bite sites are typically a bulla that develops into necrosis.
· Patients exhibit fever and general malaise and might have lymphadenopathy and hepatospenomegaly.
· The natural killer cell, infected with monoclonal EBV seems to be involved in the pathogenesis of the hypersensitivity.
· The bite area develops blisters, bullae, indurations, necrosis and ulceration, leaving a scar that heal in 2-3 weeks.
· Patients have high levels of IgE and EBV infected large granular lymphocytes in peripheral blood. Some patients also exhibit hydroa vacciniforme-like eruptions in sun-exposed area.
· Pathogenesis: After a mosquito bite, the Lymphocytes T CD4+ react to the saliva of the insect and induce the reactivations of the latent EBV infected NK cells and lead to inflammation. The immune dysregulation with predominance of the Th2 response causes high levels of serum IL-13 and IgE. The basophils activation may also be envolved in the skin reaction.
· Mortality is around 53%,

Case 2

Erythema Multiforme minor around nevi associated to herpes simples and COVID. There's also description of **Nevocentric erythema multiforme after SARS- Cov2 vaccine**:

· Most cases of nevocentric EM were described as a postherpetic. Phenomenon.
· The reaction itself differs form the well-known phenomenon described by Meyerson which consists of an eczematous halo surrounding a preexisting melanocytic nevus, but it is not clear whether and interaction between CD4 T lymphocytes and increased expression of intercellular cell adhesion molecule 1 might be involved in both these nevocentric processes.
· The course of nevocentric EM does not differ from the classical form of EM, and no alterations of the affected nevi were reported previously.
· In conclusion, nevocentric EM is a benign, limited condition that can be added to the list of possible skin reactions associated with SARS-COV-2 vaccines.

Reports written by

Dr. Marcela Cirigliano

What´s new in itch and it’s management?

Dr Gil Yosipovicth – USA

Dr Yosipovitch's conference covered various topics related to itch and its management and new treatment options. Here is a summary of some topics considered of relevance along his conference.

Pathophysiology of itch

Triggers:
- Visual and Auditory information (e.g., scratch sound)
- Mechanical and nociceptive simulation (e.g., heat, warmth, clothes)
- Inflammation (e.g., IL-4, IL-31, IL-17, IL-2, etc)
- Environmental factors (e.g., temperature, humidity, etc)
- Physiological factors (e.g., sweat)

Neural sensitization:
- Irregular habits (decreased melatonin secretion, increased body temperature)
- Imbalance in μopioid and κ opioid agonists
- Psychological stress
- Attenuated descending noradrenergic system (nighttime itch)
- Inflammation (e.g., secretion of NGF)
- Abnormal innervation (e.g., HGF, artemin and semaphorine 3A)

Itch can be caused by either an increase in excitation or a decrease in inhibition through the itch signaling pathway:
1. Primary sensory neuron: C nerve fiber and Aδ nerve fiber
2. Secondary Spinothalamic Neurons
3. Anterolateral spinothalamic tract
4. Laminar nuclei Thalamus
5. Brain

Antihistamines do not improve itching in most of the chronic itch.
The pruritogens (cytokines, non-histaminergic pruritogens and histamine) bind to their respective receptors in nerve fibers (peripheral nerves) and generate and action potential via activating TRP channels: TRPA1, TRPV1, Nav 1,7.

Cytokines and signaling pathways:
Cytokines→ receptors→ JAK activation→ STAT phosphorylation→ Gene transcription
Itch is a behavioral extension of type 2 inflammation.
Type 2 cytokines directly stimulate sensory neurons in sites of inflammation.
- Neuropeptides released by dysregulated sensory neurons act on immune cells.
- Immune cell activation and release of type2 cytokines and other inflammatory mediators directly stimulate and enhance neural function in inflammatory sites.

Th2 immunity is a major driver of chronic pruritus.
IL31 is an “itchy cytokine: cellular origin: Th2 cells, macrophages, eos, basophils, keratinocytes, fibroblasts, mast cells.
Receptor components: IL-31 RA and OSMR:

· Nerves ⇊ itch
· Epidermal Keratinocytes ⇊ barrier dysfunction, promoting nerve branching and dermal fibrosis

JAK/STAT pathway plays a key role in generating an action potential by cytokines.
Ion channels are receptors for both pain and itch.
Prurigo nodularis (PN) is highly linked with neural sensitization disorders of pain like fibromyalgia, chronic interstitial cystitis and irritable bowel syndrome and type 2 inflammation plays a role in the inflammation seen in PN
Dupilumab and Nemolizumab significantly improves itch and lesions of prurigo nodularis
JAK inhibitors show a broad anti-pruritic effect possibly trough inhibition of TRPV1 and TRPA1

Emerging topical medications for pruritus:
· Immunologic: Ruloxitinib (JAK 1/2 INH) , Roflumilast (PD4 INH) , Tapinarof 1% (Ahr agonist)
· Neural: Acetaminophen (target: Phospholipase) , MMP (target: PAR2) , KM-001 (TRPV3 antagonist), Detomidine (α2 agonist)

Treating itch addressing the neural system

BRAIN	SPINAL CORD	SKIN
IV ketamine	Gabapentin, pregabalin	Topical anesthetics
Mirtazapine	Kappa opioid agonists	Topical KAL
SSRIs, SNRIs	NK-1 INH	Topical capsaicin
TCAs	Thalidomide	Topical menthol
Gabapentin, pregabalin		Topical cannabinoids
Kappa opioid agonists		Botulin toxin
NK-1 INH		NK-1 INH
		Thalidomide

Antioxidants: systemic administration of N-acetyl-Cysteine (NAC) and N-tert-butyl-α-phenylnitrone (PBN) and Quercetin has shown to attenuate histamine dependent and independent itch

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Melasma

Peels and topical treatments for melasma
According to the talk given by Dr Ivonne Arellano – Mexico

Dr Arellano began her talk by explaining that when it comes to the therapeutic approach for melasma, it's important to take into account the following **pathogenic factors**:
1. Abnormal melanogenesis
2. Chronic inflammation
3. Hypervascularisation
4. Altered barrier function

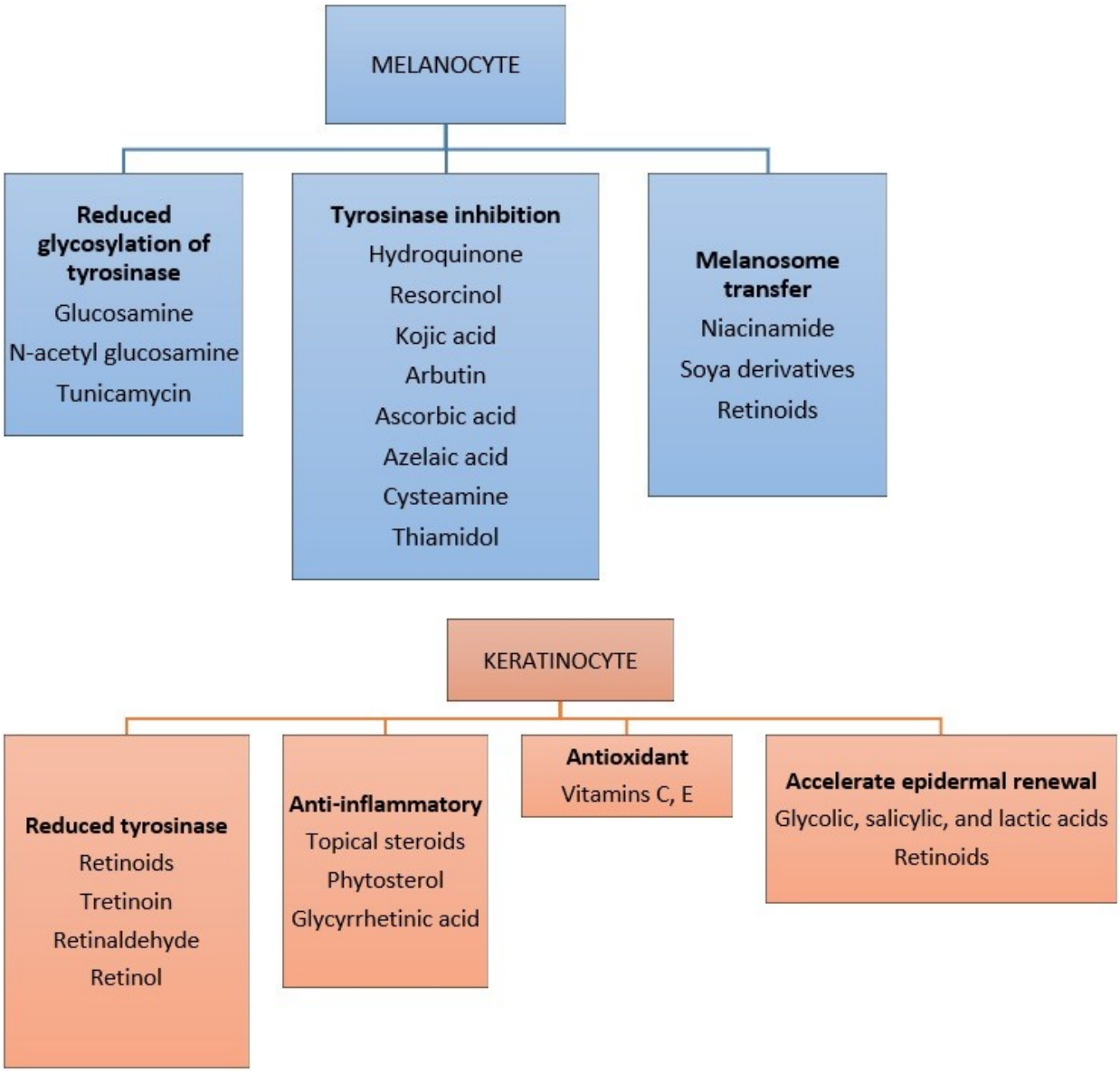
As for the **objectives of therapy**:
· Reduce the intensity of pigment
· Reduce the pigmented area
· Prevent recurrence

Melasma therapy is based on the following pillars:
· PHOTOPROTECTION

Effective, avoiding sunlight and using broad-spectrum sunscreen against UVR and visible light
· DEPIGMENTING AGENTS

Topical and oral
· ANTIOXIDANTS
Topical and oral
· PROCEDURES
Chemical exfoliation (series), microneedling, laser, OTC products/cosmetics

DIMINISHING PIGMENT
Monotherapy
Double or triple combination



Dr Arellano presented a very interesting algorithm for the treatment and monitoring of patients with melasma, in which she defined three phases:

1. **Intensive daily phase (8 weeks)**
2. **Additional daily phase (8 weeks)**
3. **Maintenance phase (16 weeks), tapering plan**

Topical treatment options:

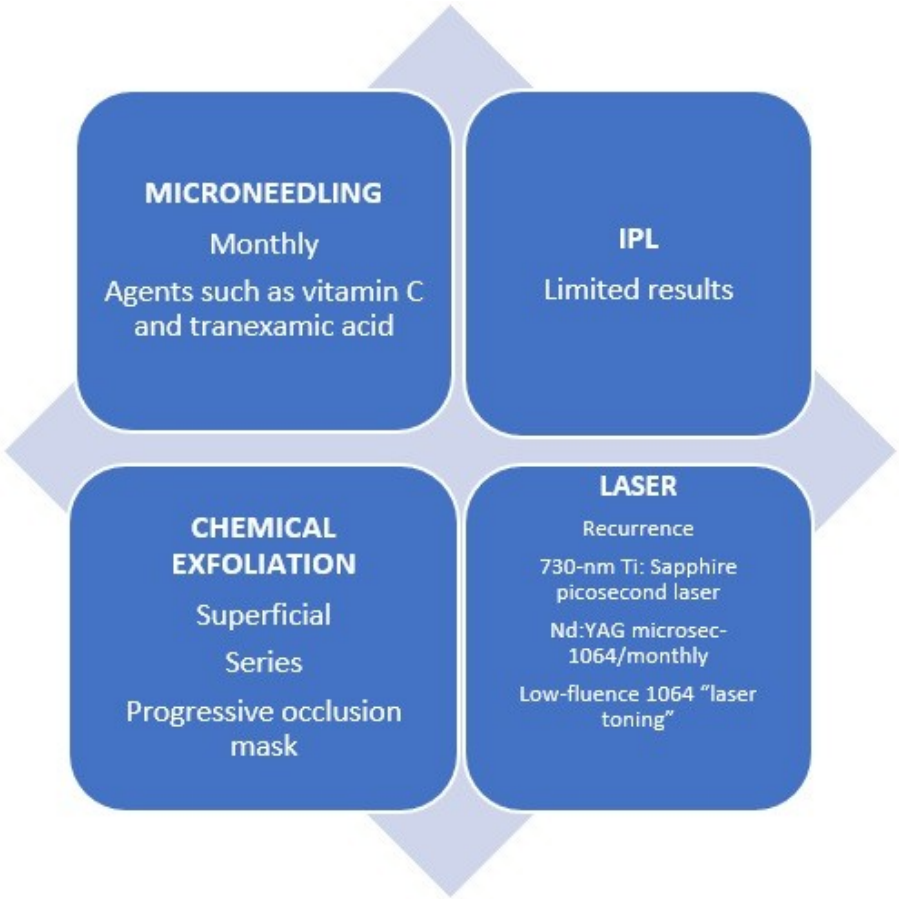
1. Broad-spectrum sun protection, including visible light. Education
2. First option: triple combination
3. Second option: double combination or hydroquinone 4%, in case the first option isn't possible

- If the treatment shows an adequate response: according to overall evaluation or reduction of MASI score by 30 or more, move on to phases 2 and 3.
- If the patient does not show a response or only a poor response:

1. First option: Triple combination + procedures (chemical exfoliation, microneedling)
2. Second option: Triple combination + lasers or light sources (IPL)

- In case the patient shows intolerance to the treatment, e.g., allergic or irritant contact dermatitis, treat the intolerance and then use a double or triple combination (non-phenolic).
- If that doesn't produce a response: re-evaluate the case, e.g., with biopsy, treatment adherence.
- If the response is adequate, move on to phase 3.

Maintenance phase (16 weeks), tapering plan: same topical treatments as in phase 1, plus review the therapy: consider alternatives (occasional adjustments or change of therapy).
Procedures should be used in combination, never as a monotherapy.



Antioxidants and melasma
According to the talk given by Dr Paula Torres – Mexico
Dr Torres reported that the use of antioxidants has increased thanks to a better understanding of melanogenesis and its alterations (oxidative stress), as well as the effects of UV and infrared radiation and visible light: photoageing and photodamage, inflammation, and hyperpigmentation.
The use of antioxidants can be complex due to variables such as stability, dosage, penetration, solubility, and absorption.

Primary antioxidants (Preventive)	Secondary antioxidants (Eliminatory)	Tertiary antioxidants (Reparative)
SOD Catalase Glutathione peroxidase Glutathione	Coenzyme Q10 Melatonin Oestrogens Carotenoids Flavonoids Phytochemicals (stilbenes and curcumin) Vitamins A, C, E	Minerals (enzyme cofactors) Proteases Telomerases

Oral tranexamic acid in melasma therapy

According to the talk given by Dr Fátima Agüero de Zaputovich – Paraguay

Dr Agüero highlighted the mechanism of action of tranexamic acid for the pathogenic targets of melasma:

- Decreased activity of AA, MSH, and tyrosinase
- Decreased activity of mastocytes
- Decreased release of VEGF and endothelin-1

She presented, among other studies, an interesting meta-analysis that indicated an optimum dose of 250 mg three times a day for 12 weeks, with the option of 250 mg twice a day. The preferred duration is 12 weeks, versus 8 weeks.

Dr Agüero stressed the adverse effects that can occur when using tranexamic acid to treat melasma, including nausea, diarrhoea, nasal congestion, muscle aches, oligomenorrhoea, abdominal pain, and eye problems such as blurred vision. She mentioned that there are other, more severe adverse effects, such as acute cortical necrosis, heart attack, and pulmonary embolism, that wouldn't be described at the doses used for melasma. She recommended that, in addition to taking an exhaustive clinical history, you should request for the patient:

- Biochemistry with hepatic and renal function
- Coagulation time
- Protein C
- Antiphospholipid antibodies and factor V Leiden

Light therapies for melasma: IPL and vascular lasers

According to the talk given by Dr Ricardo Galván García – Mexico

Dr Galván García gave a very interesting presentation concerning his vast experience with equipment and techniques that are effective for treating melasma. Some key points include:

Laser treatment of the pigmentary component: Q-switched Nd:YAG 1064 nm ns laser. Monthly sessions, with 4 to 6 in total.

He underscored that the ms pulse width (duration) should maintain a relation with the calibre of the vessel or structure to photocoagulate for the technique to be effective and safe.

Laser treatment of the vascular component:

+ IPL 555 nm, 3 to 7 J/cm2, 0.5-1.5 ms, SWT or SMT

+ Nd:YAG long-pulse 1064 nm, spot 5-6 mm, 10 to 18 J/cm2, 0.3 to 0.6 ms, 10 Hz

My best combinations against melasma

According to the talk given by Dr César González – Colombia

To begin, Dr González shared a few key points regarding the pathogenesis and histopathology of melasma:

The basement membrane is synthesised by fibroblasts and keratinocytes. Structural damage to the basement membrane facilitates the passage of dermal cytokines to melanocytes, which thus encourages the protrusion of melanocytes into the dermis.

The gene expression profile of fibroblasts in melasma includes proinflammatory and promelanogenic factors, as well as factors related to the tissue repair deficit. These factors can cause damage in the upper dermis and support the focal pigmentary phenotype.
Dr González highlighted the fact that using PLLA (poly-L-lactic acid) can improve not only colour, but also skin quality.
Sebocytes can encourage the development of melasma by exerting a paracrine effect, inducing an inflammatory process that contributes to proliferation, differentiation, and melanogenesis, since sebocytes can increase propigmentary factors, lipid mediators, and proinflammatory cytokines. Given these facts, the doctor highlighted the usage of low-dose isotretinoin for the treatment of melasma as a component of the process of ageing and photoageing.
With regard to another of the pathogenic factors, oxidative stress, Dr González noted that there appears to be a strong negative correlation between the levels of plasma glutathione and the severity of melasma; meanwhile, oxidative stress leads to depletion of plasma glutathione. Additionally, UVB radiation stimulates the expression of iNOS in keratinocytes, which leads to the activation of tyrosinase. In connection with that point, he underscored the action of melatonin as a potent eliminator of free radicals that acts indirectly by stimulating antioxidant enzymes such as SOD, glutathione peroxidase, and glutathione reductase. That stabilises the cell membranes and makes the cells more resistant to oxidative damage.

Rosacea

Systemic comorbidities with rosacea
According to the talk given by Dr Juan Carlos Diez de Medina – Bolivia

Dr Diez de Medina shared a series of general concepts relating to the link between rosacea and comorbidities:
- Given the complexity of rosacea's physiopathology, we might suspect that its phenomena are not limited to the skin and that there might also be a systemic expression of the disease.
- It should be considered a chronic inflammatory state, which makes sense when it's associated with cardiovascular diseases.
- The regulation of matrix metalloproteinases and antimicrobial peptides is the proposed link between rosacea and neurodegenerative disorders.
- Vanilloid channels are the proposed mediators of neurogenic inflammation and they contribute to the physiopathology of rosacea and migraine.
- A series of evidence suggests that rosacea may have a systemic origin or may be a marker of greater risk for an internal disease.

Following those points, Dr Diez de Medina shared the results of various studies pertaining to possible comorbidities in patients with rosacea. Based on the evidence presented, the possible associations described can be summarised as follows:

Malignancy	Gastrointestinal	Cardiovascular	Neurological	Autoimmune
Thyroid carcinoma Basal cell carcinoma Glioma	Crohn's disease Ulcerative colitis	Hypertension Hyperlipidaemia	Migraine Alzheimer's Parkinson's	HLA coincidence

Childhood rosacea
According to the talk given by Dr Ianina Massimo – Argentina

Dr Massimo presented the topographical and pathogenic characteristics of childhood rosacea.

- Skin: facial and extrafacial
- Mucosa: Ocular

Inflammatory component: erythema, papules, and pustules
Vascular component: erythema and flushing, telangiectasias, and oedema
Sebaceous component: fibrosis and phymas
In reference to a study carried out by Dr Massimo and her team, she reported that in the population studied, 84% of patients had the erythematotelangiectatic form of rosacea. In that clinical form, we find persistent erythema and telangiectasias, generally on the malar area, nose, chin, and glabella. On a histological level, we observe an inflammatory infiltrate that surrounds pilosebaceous units.
Other forms in childhood and adolescence:

- Pustular and ocular form: the patient presents papules and pustules that sit on an erythematous, pruriginous, or painful base.
- Nodular form: predominantly occurs in childhood and is more common in males
- Periorificial papular form: persistent erythema with papules and pustules located around the mouth, nose, and eyelids.
- Fulminans form: more common in males. Sudden exacerbation of multiple papules and pustules, and persistent nodules that may suppurate and resemble acne conglobata. Resolves with fibrosis. This form often presents with intense headache.

OCULAR: In the study presented, 45% of cases had ocular compromise.
The ocular pathologies that appeared in the group studied included:

- Dry eye
- Telangiectasias
- Chalazion
- Corneal ulcer
- Keratitis
- Loss of visual acuity
- Corneal empyema
- Corneal leukoma
- Blepharitis
- Meibomitis
- Epiphora
- Photophobia

78% presented demodex mites and 30% had a family history of rosacea.
With regard to therapeutics, Dr Massimo underscored the fact that the therapy must cover multiple factors, focussing on treating a combination of immune-mediated and neovascular responses, while taking into account environmental factors.

- We must always keep in mind that the skin and eye pathologies come together.
- First strategy: reduce the quantity of demodex mites. To do so, the first line of treatment is topical and systemic ivermectin.
- Effect against free radicals: Topical metronidazole
- Anti-inflammatory effect, for short-term use: Azithromycin at 10 mg/kg/day

Treatment of rosacea using lasers and other equipment based on energy sources
According to the talk given by Dr Jaime Piquero Casals – Venezuela

Dr Piquero Casals spoke about the laser treatment options for rosacea:

- Destroy the vascular component: haemoglobin + vascular walls and improve the adjacent tissue:

· IPL with dual filters: a larger spot is faster. The quartz should make contact with the skin, but without exerting pressure that would cause the emptying of blood vessels.
· Nd:YAG 1064 nm (very distinct telangiectasias)
· Pulsed-dye laser (cuperosis without inflammatory lesions, rosacea with inflammatory lesions)
· KTP-green light laser, 532 nm (cuperosis without inflammatory lesions; be careful with dark skin)
· Non-ablative laser - Er glass, 1550 nm (erythema caused by photodamage)
· Ablative fractional radio-frequency treatment
· MAL-PDT as an alternative: increases the expression of TLR, decreases the secretion of cytokines, has an immunomodulating effect in the dermis and an antimicrobial effect on Bacillus oleronius, Staph. epidermidis, and Demodex folliculorum.

Role of botulinum toxin (BT) in rosacea and flushing
According to the talk given by Dr Josaine Sanjinés Acuña – Bolivia

Dr Sanjinés Acuña highlighted the role of BT in rosacea and flushing, pointing to the various targets:
· Vascular, erythema, telangiectasias
· Flushing
· Innate immunity
· Neurovascular component
· Demodex
· Microbiome
· Alteration of the skin barrier
· Sebaceous glands
· Prevention
Dr Sanjinés Acuña noted that there is an increasing volume of evidence showing that botulinum neurotoxins have a biological effect on various types of cells, and as such, they can be used for the treatment of numerous non-cosmetic dermatological conditions.
The main angiogenic factor that produces the non-transitory erythema is VEGF, and BT suppresses VEGF through the inhibition of IL-8. It also inhibits TRPV1, which is the receptor that is activated by heat, spicy food, and alcohol. TRPV1 activation increases the release of substance P and CGRP, which are the mediators of the characteristic neurogenic inflammation we see in rosacea. Additionally, it has the ability to inhibit the degranulation of mast cells, reducing erythema.

Clinical cases we can learn from
According to the talk given by Dr César González – Colombia

Dr César González shared a series of case studies on erythematotelangiectatic rosacea with ocular compromise, treated with IPL + BT.
He commented on the ophthalmic treatment of dry eye with IPL and, specifically, the changes that occur with meibomian gland dysfunction (MGD), in which the destruction of fine telangiectasias along the eyelid inhibits the passage of inflammatory factors to the meibomian glands, reducing chronic inflammation. Focal heating enables better flow of the secretion, which favours the unblocking of the gland and reduces bacterial and parasite growth.

Updates on therapeutics: Interactive cosmetic dermatology

Skin care with rosacea
According to the talk given by Dr Jorge Moreno – Mexico

Dr Moreno highlighted the value of daily skin care with rosacea. That skin care regimen fundamentally relies on avoiding triggers, using gentle cleansers with an acidic pH, avoiding excessive and exfoliant cleansing, using emollients and moisturisers, selecting products that promote a healthy skin barrier, and, above all, strong photoprotection against UVR and visible light. He mentioned a few active ingredients found in OTC "cosmeceuticals" that are anti-irritant, anti-erythema, and antioxidant, such as licochalcones, ambophenol, neurosensine, caffeine, liquorice, and niacinamide, among others.
He presented evidence regarding the use of roflumilast to treat papulopustular rosacea. Roflumilast is a PDE-4 inhibitor that reduces the release of inflammatory mediators.

Cosmeceuticals for acne: do they work?
According to the talk given by Dr Jaime Piquero Casals – Venezuela

"Cosmeceuticals" are considered a complementary therapy to medical treatment. They include cleansers, moisturisers, and sunscreens. Dr Piquero Casals explained that the usage of cosmeceuticals specifically formulated for acne may:
- Improve the patient's adherence to their therapy
- Camouflage lesions (pigmented sunscreens)
- Reduce inflammation
- Reduce the incidence of post-inflammatory hyperpigmentation and erythema

SYNDET CLEANSERS	NON-COMEDOGENIC MOISTURISERS	TREATMENT COSMECEUTICALS	SUNSCREENS
In a foam, gel, or micellar water They lift off cellular detritus, grime, pollutants, and make-up	They counteract the irritant effect of BPO and oral and topical retinoids	Exfoliating action Adjuvant to oral pharmacological treatment AHA and BHA	Counteract and prevent sun damage Prevent PIH Camouflage of lesions

Dr Piquero Casals presented a few cosmeceuticals of interest for acne:
- Glycolic acid and buffered glycolic acid
- Retinol
- Bakuchiol
- Zinc PCA

Glycolic acid and buffered glycolic acid	Retinol	Bakuchiol	Zinc PCA	Salicylic acid	Biosaccharide gum-2	Niacinamide
Desquamation/exfoliation Bactericidal properties against P. acnes Anti-inflammatory effect	Cell proliferation and differentiation Assists penetration of other topical agents Anti-inflammatory effect	Anti-inflammatory effect Improves PIH Has a retinol-like anti-ageing effect	Sebum-regulating and antimicrobial agent	Exfoliant Keratolytic Bactericidal	Antimicrobial Calming	Sebum-regulating Reduces erythema Depigmenting agent

Photoprotection with acne

•Prevention (burns, skin cancer)
•Protection (photosensitivity associated with treatments)
•Prevents PIH
•Make-up/camouflage: colour pigments
•Faster recovery after a procedure

•Broad-spectrum (UVA/UVB), SPF ≥30
•Aqueous base and ultra-light texture

•Fast absorption, looks good
•Non-comedogenic, mattifying
•With antioxidants
•Sebum-regulating and anti-inflammatory properties

Regarding adjuvant procedures to acne treatment (peels, lasers, PDT), Dr Piquero Casals noted that they accelerate results and improve upon medical treatments.

Hair cosmetics
According to the talk given by Dr Maria Eugenia Capetta – Argentina

Damage to the hair fibre initially manifests as dry, dull, and brittle hair.

Dr Capetta explained that hair requires three levels of care:

Hair fibre
- Undamaged cuticles
- Intact cortex
- Minimise damage

Scalp
- Balanced microbiome
- Regulate sebum
- Maintain barrier function
- Photoprotection

Hair follicle
- Proper nutrition
- Euthyroidism

She stated that the frequency of washing may vary and she presented evidence that showed that greater frequency of washing is beneficial and preferable to a lesser frequency. The frequency of washing and the selection of the shampoo will depend on the conditions of the hair and scalp.

Shampoo trends:

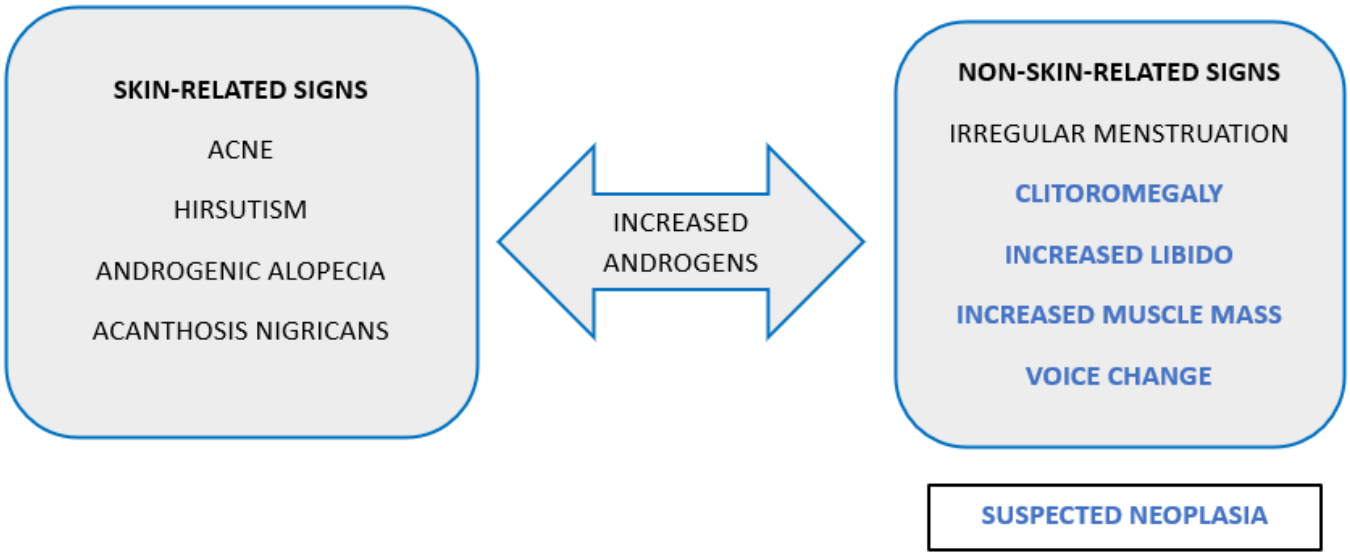
No-poo
- Washing without commercial products
- Co-washing
- Using tea tree oil
- Only rinsing with water

Low-poo
- Shampoos without sulphates
- Amphoteric surfactants

Acne

Controversies in acne hormone therapy
According to the talk given by Dr Patricia Troielli – Argentina

In her talk, Dr Troielli presented unresolved questions and controversies relating to two key themes: acne and hormonal alterations.
Acne has a peak incidence rate at 15 years old, declining in late adolescence; however, its prevalence continues into people's twenties. This incidence curve is associated with the slow decline of insulin and IGF-1 from its peak in late puberty.
Sebaceous glands have various hormone receptors (for growth hormone, melanocortin-5, CRH, and α-MSH, as well as for neuropeptides such as P substance). External stressors act on this hormonal level by stimulating the production of sebum and producing inflammatory cytokines. As such, we can say that the skin is a neuroendocrine organ, much like the HPA axis, and there is bidirectional interaction between stress and acne.
With regard to evaluating the hormones of patients with acne, Dr Troielli commented that there are several options. She also mentioned a few concepts to consider:
– 80% of patients do not show hormonal alterations.
– The AAD recommends laboratory testing for hormone levels in patients with acne and signs of excess androgens and suspected PCOS.



According to the International PCOS Network (2018-2021), the tests requested when PCOS is suspected are:

- Free testosterone
- Total testosterone
- DHEAS
- Androstenedione
- 17-OHP
- TSH
- Prolactin

Dr Troielli also presented evidence concerning biochemical anomalies in acne in adult women: in an observational study, 53% of subjects had persistent acne and 50% experienced premenstrual flare-ups. In the study population, 25% had a fasting glycaemia of ≥100 mg/dl, 10% had insulin levels >25 µIU/ml, and 55% had a HOMA-IR score >2.41. Additionally, 91% presented alterations in blood lipid levels.

Spironolactone
Average dose: 100 mg. Dosage range: 25 to 200 mg. Initial: 50 to 100 mg. Higher doses are not recommended. Taking it with food rich in fat or fish oil increases its bioavailability.

For patients 18 to 45 years old, rates of hyperkalaemia are very low, but the risk increases in women over 45 (physiological decline of kidney function in women >40 years old).
It may not be necessary to monitor potassium levels in patients with normal cardiac and renal function, independent of their age.
To summarise:

Healthy women ≤ 45 years old: DO NOT do K laboratory tests

Healthy women ≥ 45 years old: No consensus

You should do a K lab test the week after reaching the max. dosage and every 1 or 2 years after that.

If the max. dosage is 200 mg, do a K lab test annually.

Combined oral contraceptives (COC)
Oestrogens/Progestins
Approved (2012) by the U.S. FDA for the treatment of moderate inflammatory acne in women.
It can take six months to observe clinical improvement. After six months, they are as effective as oral antibiotics.
It is recommended that you push back the start of COC therapy to two years post-menarche to ensure adequate bone development, unless the treatment is clinically indicated.

Controversies around isotretinoin
According to the talk given by Dr Carlos Eduardo Montealegre Gómez – Colombia

Dr Montealegre began his presentation by explaining in which cases we should use isotretinoin:
· Severe acne
· Moderate acne
ü Body dysmorphic disorder
ü Lack of response to systemic therapy
ü As early as possible? This is a question being asked in current discussions about therapies.

The recommendations mentioned and explained by Dr Montealegre are:
When selecting the therapy, adequately educate patients: this means that they should understand the instructions for usage and possible side effects. Whenever possible, provide information in writing.
Educate patients about troubling symptoms and always order prior testing.
Potential risks: Monitor alcohol consumption and take special care when treating women of childbearing age and high-level athletes.
Review potential drug interactions.
Recommend the use of cosmeceuticals: lip balm, eye drops, and photoprotection.
Bidirectional communication: use teledermatology
Always ask about neuro-psychiatric symptoms, alcohol consumption, and contraception.
Employ a multidisciplinary focus: paediatrics, psychiatry, and other specialities.
Stay up to date with required tests and updates from academia (publications, consensus).
With regard to lab work:
Pay special attention to problematic values: triglycerides ≥500 and AST/ALT up to double the maximum.
In case of elevated transaminase levels: decrease the dosage, monitor alcohol consumption, and check in every month.

In case of elevated lipids: use statins or gemfibrozil.

Drug-induced acne: Then and now
According to the talk given by Dr Esperanza Meléndez – Colombia

Dr Meléndez began her talk by explaining that this type of acne appears suddenly and is connected to a history of taking medicine; it then improves after the medicine is stopped.

Among the possible acne-inducing drugs, she mentioned:
- Corticosteroids
- Vitamin B
- Anabolic steroids
- Lithium
- Iodides and bromides
- Targeted cancer therapy
- Medicines for autoimmune diseases

Below is a summary of some of the medicines discussed in Dr Meléndez's presentation.

Vitamin B
Anaerobic metabolism by C. acnes relies on vitamin B12. When a culture of C. acnes receives a vitamin B12 supplement, it increases the synthesis of porphyrins and produces inflammation. Additionally, excretion of vitamin B12 can irritate the follicular epithelium, resulting in inflammation.

Steroid-induced acne
This is seen with the usage of high levels of topical, inhaled, or systemic steroids. Anabolic steroids produce an increase in lipids on the skin's surface (free fatty acids and cholesterol). This stimulates the proliferation of C. acnes and produces hypertrophy of the sebaceous glands. The clinical presentation can range from a papulopustular form to acne conglobata or fulminans. Steroids can also exacerbate existing acne and may be accompanied by hirsutism and androgenic alopecia.

Iodine
Iodine can exacerbate existing acne or cause an acneiform eruption. It results in compromise of the face and upper trunk. In iodine patch tests, you may observe inflamed follicular pustules.

Whey protein
This is frequently used as a supplement by patients who visit the gym often. It increases levels of IGF-1, thus stimulating sebogenesis.

Testosterone pellets
These are used to improve libido, muscular strength, cognition, and cardiovascular function, as well as avoid the effects of ageing. The most commonly reported adverse effects associated with their use are acne and hirsutism.

Acne induced by JAK inhibitors
In biopsies of acne lesions, researchers have observed overexpression of JAK1 and JAK3 compared to skin free of lesions. As such, more studies are required to understand the mechanism by which acne is induced with JAK inhibitors.

Acneiform eruption with targeted therapy
This occurs in 45% to 100% of patients receiving EGFRi therapy and it always develops in the first four weeks. It is more severe with monoclonal antibodies than with kinase inhibitors. The severity of

the eruption is generally mild to moderate, but it can be severe. The lesions include comedones, papules, and pustules on the face and thorax, with sensations of burning, itching, and xerosis. It is the most common manifestation with targeted therapy. With regard to treatment, Dr Meléndez mentioned topical doxycycline foam, clindamycin, metronidazole, or mupirocin. The use of BPO is not recommended as it may cause xerosis. She also mentioned that administering vitamin K prevents this rash and decreases the intensity of the acneiform eruption.

Controversies around acne in adult women
According to the talk given by Dr Marco Alexandre Dias da Rocha – Brazil

Dr Dias da Rocha talked about the three acne phenotypes that tend to appear in women.
· Acne in adult women
· Hyperandrogenism: Polycystic ovary syndrome
- Menstrual irregularity and hirsutism (70%)
- Acne (30-50%)
· Metabolic syndrome
- Obesity
- Hypertension
- Dyslipidaemia
- Acanthosis nigricans

Dr Dias da Rocha explained several key points:
- Most patients have plasma androgens within the reference values (they are normoandrogenic).
- The psychological impact is significant, though generally underestimated.
- Stress in one's private and work lives, anxiety, and sleep quality all have an impact on susceptibility and the severity of the disease.
- The impact on quality of life is not dependent on the intensity of the acne.
- The increased incidence of acne can be explained by several factors: emotional and psychological stress, immunity, microbiome changes, diet, hormonal factors, tobacco use, and drug use, among others.

Case-based therapeutics

Difficult-to-treat acne
According to the oral presentation by Dr José María Azeñas – Bolivia
Dr Azeñas presented cases of acne treated with high doses of isotretinoin, a sign that these were cases that were more difficult to treat, whether due to their clinical severity, or due to poor previous treatment of the patient's pathology, or due to a lack of response to previous topical and/or systemic treatments. The doctor presented three cases of severe acne in male patients who were treated with isotretinoin at 1.5 mg/kg per day, for a cumulative total dose of 130 mg/kg. Dr Azeñas reported that these were patients who had a slower response, as shown by the fact that they started seeing improvement after about four months. These were patients who came to see the doctor with significant emotional distress due to the quantity and severity of lesions and resulting scars. After completing medical treatment with high-dose isotretinoin, for patients that need it, the doctor may proceed with treating the scars.

Combined treatment of mixed melasma
According to the presentation by Dr Héctor Cáceres – Peru

Dr Cáceres shared with the audience his experience with combined medical and laser treatments. He reported that today, a high percentage of women around the age of 70 years old have some degree of dyschromia or melasma, conditions in which there is dysregulation of the mechanisms that control pigmentation, resulting in greater production of melanin by melanocytes, greater transfer of melanosomes to keratinocytes, and ultimately, a very important fact, disruption of the basement membrane, with the appearance of “pendulous melanocytes,” which protrude into the dermis and are difficult to treat due to their depth. In addition to a genetic predisposition, light exposure is a critical factor in pathogenesis, and not just UVA, UVB, and infrared radiation, but also, particularly, blue light. Other factors involved in the pathogenesis are hormonal: oestrogens and progestogens, inflammatory mechanisms, and an increase in dermal mast cells, which produce angiogenic factors that increase vascularisation. As such, Dr Cáceres noted that, in his opinion, all cases of melasma have some grade of vascular alteration, whether visible or not, such that if you perform dermatoscopies on these patients, they all show some grade of vascular alteration.

When developing therapies for these patients, it is essential to bear all of these factors in mind. Firstly, Dr Cáceres noted the crucial importance of broad-spectrum photoprotection with pigments to protect against blue light. Additionally, he suggested melanin synthesis inhibitors: Hydroquinone (with the doctor emphasising its use as a first-line treatment, not for maintenance treatment), kojic acid, azelaic acid, arbutin, and ascorbic acid; you can also aim to inhibit the transfer of melanosomes to keratinocytes, for which niacinamide 10% is very effective. To remove pigment, if it’s in the epidermis, you can increase turnover, which can be done using glycolic acid, lactic acid, or retinoic acid. To treat inflammation and secondary vascularisation, the doctor recommends tranexamic acid.

With regard to equipment that can be used for treatment, he mentioned pulsed light and laser equipment. These devices remove melanic pigment, but they do not prevent its production; as such, Dr Cáceres proposed and underscored the value of mixed or combined therapy. He noted that when using lasers, it’s important to minimise excessive heat in the area around the treatment area. This can be done by using newer equipment that allows for short pulses (in picoseconds) that produce a photoacoustic effect rather than a photothermal effect, with selective destruction that prevents damage to surrounding tissue. These devices produce greater fragmentation of the pigment via pulverisation: thus, the pressure of the laser energy breaks up the melanin without generating heat that would inflame the healthy tissue around the treatment area. You must always choose the right wavelength, using very short pulses, which is what produces the effect of an acoustic shock wave. Additionally, Dr Cáceres mentioned that these procedures using equipment producing picosecond pulses are better tolerated by patients. The doctor presented several of his own clinical cases in which he combined treatments using equipment producing picosecond pulses: in these cases, not only was pigment removed, but also the treatment created microchannels in the tissue to allow for the application of medicine (laser-assisted drug delivery), in this case, tranexamic acid in a gel under occlusion. He also mentioned a new 9-mm fractional laser device specifically for the treatment of melasma, with larger spots and lower fluency, which produces a great distribution of energy and can be used with various wavelengths. These combined treatments prove to be particularly effective with mixed melasmas, achieving a very noticeable response after four or five sessions. Dr Cáceres also highlighted the importance of accompanying these procedures with maintenance treatment, applying SPF 50+ sunscreen with pigment every two hours, a vitamin C serum in the morning, and a niacinamide 10% plus tranexamic acid 5% cream at night.

Haemangiomas: Experience within my practice

According to the presentation by Dr Natalia Velásquez – Colombia

Dr Velásquez presented her experience with care and treatment for infantile haemangiomas, which are the most common tumours found in infancy, are of endothelial origin, and have an incidence rate of 4%. Among the risk factors, she mentioned pre-term birth, low birth weight, being female, being white-skinned, multiple gestation, and a family history of haemangiomas, among others. She noted that they are typically visible within the first few weeks of life and they follow a trajectory of

proliferation followed by involution. Most (50-60%) are superficial and consist of bright red papules, plaques, or tumours with a smooth or lobulated surface; 15% are deep and appear as bluish or skin-coloured tumours with telangiectasias on the surface; and 25-35% are mixed. Dr Velásquez presented two of her own clinical cases treated with timolol 0.5% drops (starting with one application per day and later two per day) with great results in terms of progressive reduction and complete elimination. She also presented a case of an ulcerated surface haemangioma treated with an intralesional injection of triamcinolone 50%, resulting in progressive reduction.

Combined treatment of keloid scars

According to the presentation by Dr Andrés Luque – Colombia

Dr Luque shared his experience with surgical treatment for keloid scars, with techniques combined with injections of bleomycin, as well as intralesional cryosurgery. Dr Luque highlighted the importance of maintenance treatment with pressotherapy for up to one year after surgery to prevent recurrence. With regard to the injections, he underscored the importance of seeing immediate whitening of the lesion as a sign that the injection was done properly, at the right level, which also avoids potential atrophy of the tissue. He also talked about the technique he uses for cryotherapy on keloids, using hydrodissection between the healthy tissue and the keloid tissue, which can be done with a saline solution or anaesthetic. He also shared his experience with the laser-assisted drug delivery technique for the application of topical corticosteroids. Additionally, Dr Luque presented the treatment of multiple keloids with CO2 laser in ultrapulsed mode, resulting in near-complete resection of the lesion, followed by contact cryotherapy. This technique, which combines the use of fractional laser with cryotherapy using a contact probe, in addition to offering great results, enables better recovery after treatment.

Scarring after burns: A therapeutic challenge

According to the oral presentation by Dr Susana Misticone – Venezuela

Around 70% of scars caused by second- or third-degree burns show abnormal healing, with alterations in thickness, texture, and pigmentation, as well as erythema. Dr Misticone explained that the earlier you detect pathological scarring, the easier it will be to treat. She noted that when any rigidity or hardness is detected at one to three months, the bandaging with silicone gel sheets should be exchanged for steroid tape, or you should start steroid injections. If the scarring doesn't improve, you should treat it with enzymes, 5-FU, or laser.

Laser treatments are effective, with low risk of adverse effects and fast recovery time. They can't replace surgery, but they can reduce its extent. Given that burn scars present alterations in thickness, pigmentation, and texture, the combination of several lasers enables more comprehensive treatment, especially using PDL or IPL combined with a CO2 laser. CO2 and erbium fractional lasers are the most common non-surgical option for improving the functional results of serious recalcitrant hypertrophic scars. Dr Misticone also mentioned the laser-assisted drug delivery technique, in which fractional lasers create lines in the epidermis in which the drug is delivered for better release and action.

With regard to the treatment of itching in keloids, she noted the action of collagenase, which reduces fibrosis, and injectable bleomycin, which inhibits collagen synthesis via suppression of TGF-β1. Finally, she mentioned radiotherapy for its anti-fibroblastic and anti-angiogenic action as an adjuvant treatment 24-48 hours post-surgery.

Hypertrophic and deforming facial scars treated with laser and injections

According to the oral presentation by Dr Carlos Echevarría Escribens – Peru

Dr Echevarría Escribens presented a case of a patient with hypertrophic and deforming scars on the face and neck, for which he combined treatments: first with enzymes and intralesional corticosteroids, then with surgery on certain hypertrophic scars—only those oriented in the same direction as the

skin’s tension lines—and later with sessions using a CO2 fractional laser, followed by treatment with clobetasol cream. The results were highly satisfactory after four sessions.

A therapeutic challenge in psoriasis

According to the presentation by Dr María Victoria Suarez – Brazil

Through the presentation of a case of severe psoriasis in a patient who also suffered from a psychiatric disorder, drug abuse, and alcoholism, Dr Suarez reviewed the evidence for the prevalence of addictions in patients with psoriasis. She also highlighted the fact that cocaine produces an inflammatory state, with activation of microglia and an increase in inflammatory cytokines, as well as an increase in the IL-6 to IL-10 ratio, which further boosts the inflammatory state. Through a strict and precise discussion of the case presented, Dr Suarez underscored the importance of a multidisciplinary approach for these patients, particularly involving collaboration between psychologists and dermatologists. She views this as crucial for this type of high-risk patient.