BIODERMA CONGRESS REPORTS

# JDP 2025

Reports written by **Déborah Salik** (dermatologist, Belgium), **Nicolas Kluger** (dermatologist, Finland) and **Ibrahima Traoré** (dermatologist, Guinea)

## LASERS IN DERMATOLOGY: PRINCIPLES, HANDLING AND SAFETY

Report written by Déborah Salik (dermatologist, Belgium)

Chair: Yvon Perrillat (France)

Speakers: Yvon Perrillat (France), Thierry Fusade (France), Samy Fenniche (France), Kawtar Zouhair (Morocco), Nathalie Gral (France), Sabrina Fourcade Roch (France)

### Introduction

The aim of this practical workshop was to introduce participants to different laser technologies. Each participant was able to familiarise themselves with how they work, handle the equipment in real-life conditions and master essential safety rules for day-to-day use.

## Techniques and safety

The session began with a reminder of the essential safety instructions. When using lasers in dermatology, you need to wear specific protective eyewear, tailored to the wavelength of the laser used. Patients are strongly advised to use eye shields to prevent any risk of error relating to the type of eyewear.

## Vascular lasers and intense pulsed light (IPL)

**Pulsed dye laser (PDL)** treatment operates at a wavelength of 595 nm, which corresponds to the absorption spectrum of haemoglobin. It can be coupled with a YAG laser for dual action. Its main indications are flat angiomas (face, trunk, limbs), rosacea, poikiloderma of Civatte and spider angiomas. It is suitable for infants.

Other vascular systems also exist:

- **KTP:** Used for facial telangiectasias.
- **Nd:YAG laser**: Operating at 1064 nm, it is indicated for cherry and spider angiomas and spider veins, but is less suitable for couperosis and erythrosis.
- **Intense pulsed light (IPL - Nordlys)**: indications: pigmented lesions, venous lakes, rosacea, scars, etc.

## CO2 laser

CO2 laser is a powerful ablative tool operating at 10600 nm. **UltraPulse** can reach a depth of 4 mm and is used for scars, facial resurfacing and the treatment of condylomas. The **Déka** system combines an ablative CO2 laser (10600 nm) with a non-ablative fractional laser (1540 nm) to prevent post-inflammatory hyperpigmentation, particularly in the resurfacing of atrophic scars.

## Lasers for pigmented lesions

They treat pigmented lesions and are also used for tattoo removal. **Q-Switched** systems use wavelengths of 1064 nm (infrared, for deep lesions such as naevus of Ota) and 532 nm (peak

melanin absorption, up to phototype IV). The work must always be carried out at the focal point.

## AviClear

This is an innovative laser for treating inflammatory acne at a wavelength of 1726 nm. It acts directly on the sebaceous glands, causing atrophy and reducing sebum secretion.

## Hair removal lasers

Alexandrite and Nd:YAG are the most commonly used. These types of lasers are also indicated for conditions such as pilonidal cysts and Verneuil's disease.

# PHOTODERMATOLOGY: WHAT DO I NEED TO KNOW ABOUT UV RAYS AND THE ENVIRONMENT? INTERACTIVE CLINICAL CASES

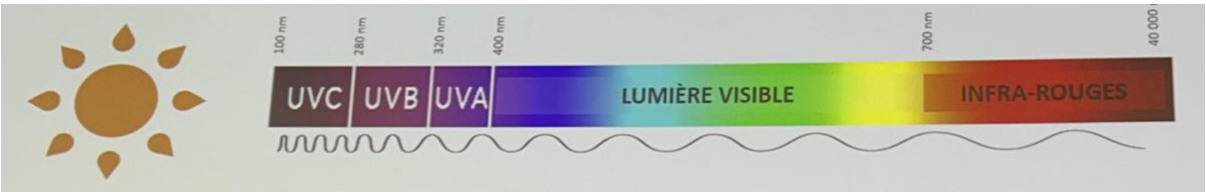
Report written by Déborah Salik (dermatologist, Belgium)

Chair: Henri Adamski (France)

Speakers: Henri Adamski (France), Martine Avenel-Audran (France), Chistophe Bedane (France), Christelle Comte (France), François Aubin (France), Laurent Meunier (France), Manuelle Viguier (France), Jean-Claude Béani (France)

## Solar radiation: Composition

Solar radiation consists of **UVA** and **UVB rays (7%)**, but also includes **visible light (40%)** and **infrared radiation (53%**).



## Radiation characteristics

- **UVA rays**: They penetrate down to the dermis and are present all year round, all day long.
- **UVB rays**: They are stopped in the epidermis, are predominant in summer and at hot times, and are partially stopped by clouds.
- **Infrared (IR) rays**: They account for 50% of solar energy, are perceived as heat and cause significant oxidative stress.
- **Visible light (VL)**: Its blue component can induce pigmentation (melasma) in darker phototypes (IV to VI) and contributes to photo-ageing. However, the blue light from screens is negligible compared with that from the sun.

## The effects of the solar spectrum on the skin

| Skin effect | Share of responsible radiation |
|---|---|
| **Solar erythema** | 90% UVB rays, 10% UVA rays |
| **Skin cancer** (carcinoma and melanoma) | 60-65% UVB rays, 35-40% UVA rays<br>- Basal cell carcinoma: intense and intermittent exposure in childhood.<br>- Squamous cell carcinoma: chronic exposure.<br>- Melanoma: sunburn during childhood. |
| **Photo-ageing** (wrinkles and fine lines, yellowish complexion, pigmentation disorders) | UVA rays, visible light and infrared radiation |

| | |
|---|---|
| **Photosensitisation** (sun allergy and benign summer light eruption) | UVA >>> UVB rays |
| **Photodermatoses** (aggravation of an existing dermatosis: rosacea, herpes) | UVA rays |
| **Hyperpigmentation** (solar lentigo, melasma) | UVA - UVB rays and visible light<br>Mainly in women (affects one out of three). The risk is increased nine-fold for phototypes IV to VI, with a multifactorial aetiology (sun, genetics, oestrogen, oral contraception). |

## Beneficial effects

Exposure to the sun has positive effects on seasonal depression and also on vitamin D synthesis (via UVB rays).

The amount of UV radiation needed to synthesise vitamin D is very low. According to the literature, a white child wearing clothes and exposing his hands and face requires 30 minutes to two hours of exposure per week for adequate vitamin D synthesis. For adults, five minutes of exposure per day, three times a week, is mentioned for a light phototype and 10 minutes for a dark phototype (PMID: 16164370). The use of adequate sun protection does not alter patients' vitamin D status, unlike their exposure habits (seeking shade, clothing).

AFSSAPS recommendations for anti-sun products stipulate that an anti-sun product should meet three criteria: persistence, photostability and protection against all the harmful effects of UV rays.

# 35 YEARS OF NAIL DISORDERS

Report written by Déborah Salik (dermatologist, Belgium)

Speaker: Bertrand Richert

Professor Richert gave an account of his most impressive cases over the last 35 years.

## Onychopapilloma

- Presence of longitudinal erythronychia, notching of the nail matrix (at the lunula) and subungual hyperkeratosis
- Women > Men
- Special case: lichen planus presenting as onychopapilloma
- Some cases of onychopapilloma are polydactyl, constitute a new cutaneous sign of loss of BAP1 function and are called “onychobapilloma”

## The most frequent diagnoses with nail alterations in children (PMID: 30835872)

**Table 2** Categories of diagnoses (the 10 most frequent diagnoses appear in bold)

| Categories | Diagnoses | *n* |
|---|---|---|
| Traumatisms<br>*N* = 77<br>25% | **Podiatric-related chronic trauma** | **23** |
| | **Acute trauma and *sequelae*** | **19** |
| | **Juvenile ingrown nail** | **15** |
| | Traumatism of unknown origin | 7 |
| | Onychotillomania | 7 |
| | Onychophagia | 6 |
| Congenital and/or hereditary disorders<br>*N* = 53<br>17.2% | **Congenital malalignment of the great toenail** | **25** |
| | **Congenital hypertrophy of the lateral nail folds** | **18** |
| | Other genetic syndrome | 5 |
| | Congenital pachyonychia | 2 |
| | Ectodermal dysplasia | 1 |
| | Periungual congenital naevus | 1 |
| | Supernumerary nail | 1 |
| Infections<br>*N* = 47<br>15.3% | **Onychomycosis** | **19** |
| | **Acute paronychia** | **17** |
| | Periungual wart | 6 |
| | Chronic paronychia | 5 |
| Inflammatory disorders<br>*N* = 40<br>13% | **Trachyonychia** | **26** |
| | Nail psoriasis | 8 |
| | Nail lichen planus | 3 |
| | Psoriasiform acral dermatitis | 1 |
| | Juvenile plantar dermatosis | 1 |
| | Lichen *striatus* | 1 |
| Tumours<br>*N* = 31<br>10% | **Longitudinal melanonychia** | **25** |
| | Subungual exostosis | 4 |
| | Inclusion body fibromatosis | 1 |
| | Onychopapilloma | 1 |
| Systemic disorders<br>*N* = 30<br>9.7% | **Fever-related Beau's lines or onychomadesis** | **30** |
| Physiological alterations<br>*N* = 15<br>4.9% | Physiological koilonychia | 12 |
| | Postdelivery Beau's Lines or onychomadesis | 2 |
| | Herringbone nails | 1 |
| Others diagnoses<br>*N* = 15<br>4.9% | Idiopathic pincer nail | 8 |
| | Nail fragility | 6 |
| | Calcinosis | 1 |
| **Total** | | **308** |

Nail disorders in children are generally benign and often self-limiting.

## Iso-Kikuchi syndrome

Congenital onychodysplasia of the index finger (MIM605779), or Iso-Kikuchi syndrome, corresponds to an abnormality of the nail of the index finger revealing underlying bone involvement. Various diagnostic criteria have been described: congenital brachymetacarpia and/or brachydactyly, uni- or bilateral hypoplasia of the index finger and onychodysplasia of variable appearance. X-rays confirm the diagnosis, showing Y-shaped bifurcation and hypoplasia of the distal phalanx of the finger concerned.

Various pathophysiological hypotheses have been put forward: iatrogenic (anticonvulsants during pregnancy), vascular (in utero ischaemic damage to arterioles that depend on the radial artery), or alterations in osteogenesis. None have been confirmed. Several familial cases have been reported, suggesting autosomal dominant transmission of a monogenic Mendelian disease, but no gene has yet been described.

## Squamous cell carcinoma

- The most frequent malignant tumour of the nail apparatus
- Bowen's disease: SCC in situ
- Mainly affects the first three fingers of the right hand (especially the middle and index fingers)
- Three types: verrucous (47%), verrucous + longitudinal melanonychia (12%) related to HPV, oozing type (28%)
- Exceptional metastasis, good prognosis
- Treatment: Conservative surgery with removal of the tumour (Mohs), amputation only if metastasis
- Role of HPV 16, 25% to 60% of carcinoma cases
- Proposed term: Nail apparatus intraepithelial neoplasia (NIN)

## Onychomatricoma

- Longitudinal pachyxanthonychia with a characteristic wood-worm-like appearance
- Usually monodactyl but described with two and six affected nails
- Search for a link between the clinical presentation and the operative outcome: tangential excision is ideal and allows for regrowth of a normal nail in 71% of cases. The risk of sequelae increases for lesions larger than 3 mm

## Retronychia

- Classic triad in retronychia
    1) Cessation of nail growth
    2) Paronychia
    3) Xanthonychia
- Disorder of adolescents/young adults, possibly involving podiatry and footwear problems
- Tends to develop into a chronic form
- Topical treatment with corticosteroids (no difference with or without occlusion), works well on early forms as it allows the plate to realign. Six to eight weeks of treatment are necessary
- Possibility of intralesional injection
- It is possible to perform proximal avulsion, with resolution in almost all cases, but in 10% of cases there is regrowth of a dystrophic nail

# NEUROFIBROMATOSIS: THE BASICS IN PRIVATE PRACTICE AND IN HOSPITAL

Report written by Déborah Salik (dermatologist, Belgium)

Chair: Laura Fertitta (France)

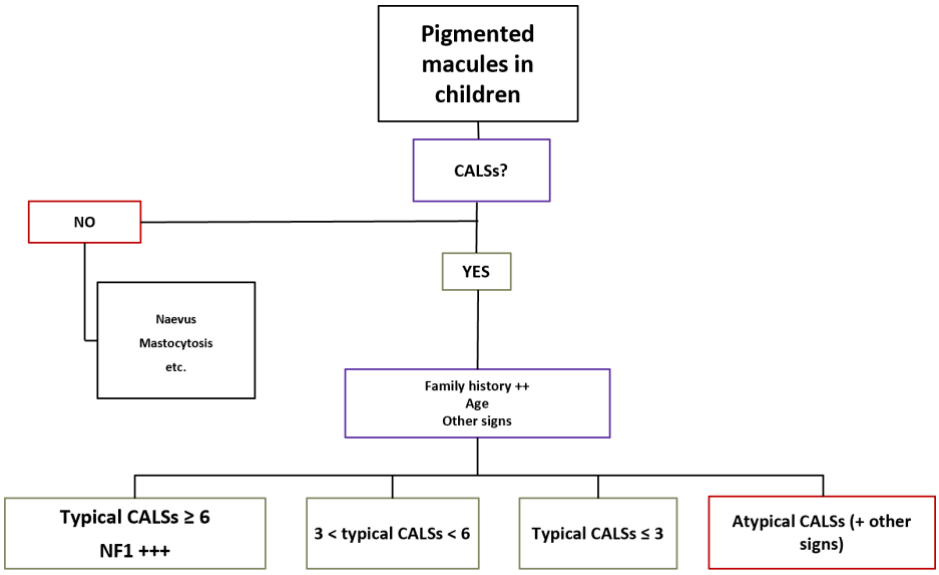
Speakers: Laura Fertitta (France), Aranaud Jannic (France), Sébastien Barbarot (France), Pierre Wolkenstein (France), Mona Amini-Adle (France)

## Subcutaneous neurofibromas and differential diagnosis

There are six questions to ask when faced with café au lait spots (CALSs)

1. CALSs or not?
2. CALSs typical of NF1?
3. Age?
4. Number?
5. Family history?
6. Other signs?

Algorithm for managing CALSs in children:




Patients with three CALSs represent 20% of the population.

Differential diagnoses for CALSs:

- Mastocytosis
- Clear congenital naevi or early naevus spilus. These are often single lesions. Clinical follow-up is useful in making the diagnosis. Confocal microscopy is an excellent tool for making the distinction
- Becker's hamartoma
- Pigmentary mosaicism (phyloid, blaschkolinear)

A CALS suggestive of NF1 is a rounded oval macule with a clear border, sparing the cephalic extremity. Lesions are generally present at birth and lentigines appear around the age of two to three years. Neurofibromas (NFs) appear later and are highly polymorphic. They are either superficial, maculopapular with skin atrophy, or subcutaneous, forming small, firm nodules along nerve pathways. Their presence is a factor for poor prognosis and the presence of internal neurofibromas.

Large CALSs can become progressively hairy and may be associated with the presence of plexiform NFs. Plexiform NFs can also be atrophic, but this is a rarer clinical presentation.

There is also a segmental presentation of NF1, which corresponds to type 1 (due to a postzygotic mutation) or type 2 mosaicism, based on the second-hit hypothesis.

There may be other associated clinical signs:

- Naevus anaemicus has a prevalence of 50% but is also observed in other RASopathies.
- Juvenile xanthogranulomas are present in ⅓ of cases, appear before four years of age and are transient.

Other conditions with CALSs:

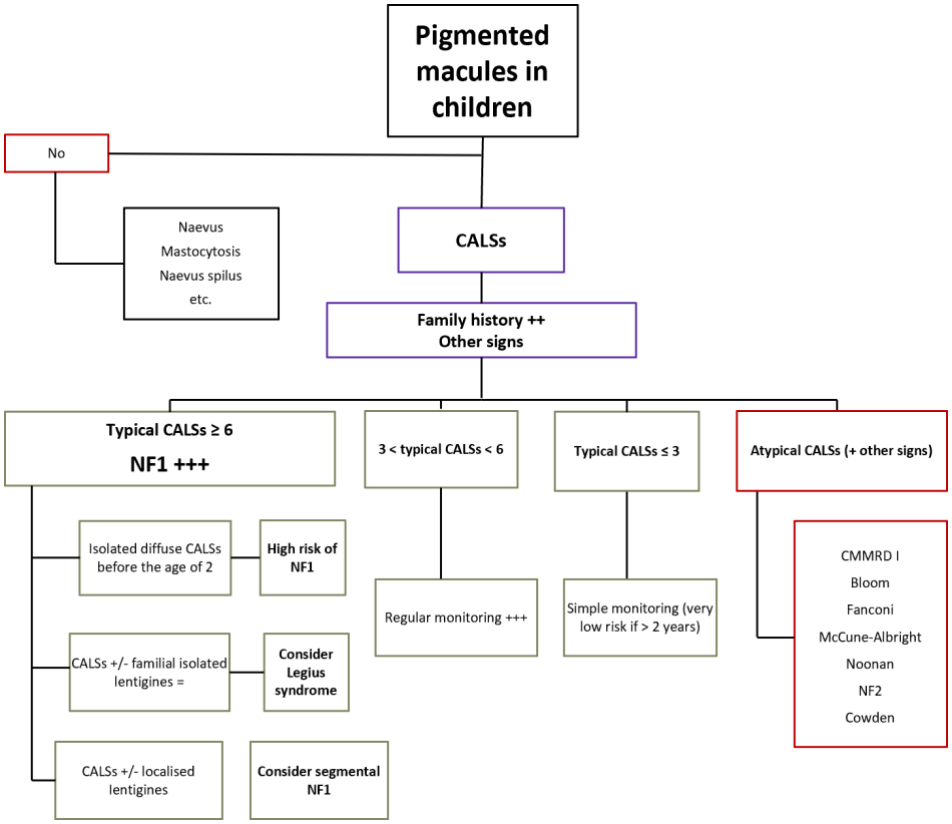
1) Legius syndrome
    - SPRED1 mutation
    - CALSs and isolated lentigines
    - 50% have at least two NF1 criteria

- Learning disability in 20% of cases
- Macrocephaly
- Lipoma
- No Lisch nodules, no NFs, no optic pathway tumours

2) Noonan-lentigines or LEOPARD syndrome
    - PTPN11 or NRAS mutated
    - CALSs and lentigines that are highly pigmented, like naevi

3) Non-PTPN11 Noonan syndrome
    - Presence of ulerythema ophryogenes
4) Bloom syndrome: scoliosis, chronic cheilitis, facial erythema (photosensitivity), “butterfly wing” telangiectasias
5) McCune-Albright syndrome
    - Large and irregular CALS
    - Precocious puberty, hyperthyroidism, hypophosphatemia
    - Somatic GNAS mutation
    - Perform X-ray and bone scan
6) Turcot syndrome: Childhood constitutional mismatch repair deficiency
7) CM-AVM: RASA1 - EPHB4

In short:




## Complications of NF1

Speaker: Laura Fertitta (France)

NF1 is a multisystem disease with complications that differ according to age.

**In children:**

1) Optic pathway glioma

- The most common intracerebral tumour in NF1
- In 15 to 20% of children
- Median age: Four years
- Slow and progressive course with a low risk of malignant transformation
- Good prognosis with a five-year survival rate of 90%
- Treatment discussed according to radiological progression or visual threat

- Should be screened for: visual function, OCT, fundus examination, presence of strabismus or exophthalmos
- Paediatric examination: neuromotor, signs of intracranial hypertension, early puberty, break in curve, measure BW
- Ophthalmological consultation every six months until the age of six, then once a year until the age of 18
- No systematic MRI of the optic pathways and brain – only if clinically indicated

2) Neurodevelopmental complications

- Frequency difficult to assess, probably underestimated
- Broad spectrum: ASD, ADHD
- Delayed psychomotor and/or language acquisition to be screened for
- Carry out neuropsychological assessments if necessary

3) Bone complications

- Scoliosis: common (10-25% of cases)
- Often associated with vertebral dysplasia
- Clinical evaluation is the rule and X-rays are necessary if there are clinical symptoms

4) Long bone dysplasia

- Remains rare (< 10% of cases)
- Congenital: curvature of a limb, generally the tibia or radius, with a risk of fracture and secondary pseudarthrosis
- Early detection

5) Growth and weight-related complications

- Shorter than the general population

- Sometimes associated with GH deficiency
- Precocious puberty in 3% of cases, often associated with OPG
- Request a brain MRI in case of suspicion

6) Cardiovascular complication: hypertension

- In children, represents 16% of cases, and increases with age
- Hypertension is primary or secondary to renal artery stenosis, coarctation of the aorta or pheochromocytoma
- Annual blood pressure monitoring
- Systematic work-up in cases of hypertension: CT angiography or MR angiography of the renal arteries

**In adults**

1) Ophthalmological complications

- Low risk of OPG
- Myopia, glaucoma, retinal hamartomas with risk of detachment
- Follow-up every two years

2) Screening for hypertension

- Annual
- If poorly controlled hypertension: repeat the work-up to look for secondary hypertension

3) Bone demineralisation

- Common: 45% osteopenia and 25% osteoporosis
- Phosphate-calcium metabolism disorder and vitamin D deficiency
- Fracture risk

4) Specific case of pregnancy

- Genetic counselling: 50% risk of transmission: Prenatal or preimplantation genetic diagnosis
- Hormonal impact of pregnancy on cutaneous neurofibromas?
- Close obstetrical monitoring
- Increased risk of hypertension
- Specifically in the 2nd-3rd trimester: Lumbosacral MRI allowing an epidural to be performed in the absence of localised NF at the puncture site
- Increased risk of prematurity and IUGR

5) Risk of cancer

- Increased risk of all types of cancer combined
- Life expectancy reduced by 10 to 15 years
- Main cause of death: risk increased four-fold compared with the general population
- By the age of 50, 20% of individuals with NF1 will have cancer

## Management of plexiform NFs

Speaker: Mona Amini-Adle (France)

**Plexiform NFs**

- Are one of the seven NF1 criteria
- Common: 30 to 50% of NF patients
- Often congenital
- Associated with considerable morbidity: disfiguring, functional limitation (blindness, deafness, respiratory, motor)

- 10-15% risk of degeneration into malignant peripheral nerve sheath tumour (MPNST)
- Two types: superficial and deep
- Surgery if possible
- Alternative to surgery: new treatment: MEK inhibitor

**SPRINT study: Selumetinib**

- Leads to significant volume reduction in almost 75% of patients
- Significant improvement in quality of life
- Poor tolerance, with toxicity leading to discontinuation in 10% of cases
- Duration of treatment: Six months to one year, but very rapid pain-relieving efficacy

**MIRDAMETINIB**

- From the age of two years
- As a drinkable suspension, making it a good alternative for children
- Improvement in pain
- Significant volume reduction

**There is mucocutaneous toxicity associated with MEK inhibitors, with acne, paronychia, aphthous ulcers and ulceration of the oral mucosa.**

# PAEDIATRIC DERMATOLOGY – GENETICS 2

Report written by Déborah Salik (dermatologist, Belgium)

Chairs: Thomas Hubiche (France), Sébastien Barbarot (France), Hamida Turki (Tunisia)

Speakers: Isabelle Luchsinger (Switzerland), Malek Cherif (Tunisia), Constance Deblock (France), Juliette Mazereeuw-Hautier (France), Rajaa Bousmara (Morocco)

## X-linked hypohidrotic ectodermal dysplasia: specific phenotype in female transmitters

Speaker: Isabelle Luchsinger (Switzerland)

X-linked hypohidrotic ectodermal dysplasia is the most common form of ectodermal dysplasia. The phenotype of female carriers is poorly characterised.

**Objective**: Describe the symptoms in women who transmit the disorder.

**Method**: Prospective study including women with a variant in the EDA gene.

**Results**: 36 women with:

Dental:

- Persistence of baby teeth (44%)
- An anomaly in shape: conoid teeth (42%)
- Hypodontia

Dermatological:

- Eyebrows: hypotrichosis in 78% of cases and fine hairs in 83%
- Eyelashes: partial hypotrichosis in 75% of cases
- Scalp: hypotrichosis and fine hair associated with slow hair growth
- Body hair: total alopecia and blaschkolinear hypotrichosis in 75% of cases. This reflects mosaicism
- 80% of women reported perspiration problems and regular fainting
- Dry skin and increased atopic diathesis (53%)
- Dry mucous membranes: xerostomia, xerophthalmia, hoarse voice, genital dryness

Breast abnormalities:

- Significant breast asymmetry (53%)
- Exceptionally flat nipples in 2/3 of women and absence of Montgomery glands, associated with breastfeeding difficulties

Conclusion: It is important to be able to identify female transmitters based on clinical criteria. These women deserve appropriate multidisciplinary support.

## Palmoplantar keratoderma in autosomal recessive congenital ichthyosis: clinical and genetic features

Speaker: Malek Cherif (Tunisia)

ARCI is a heterogeneous group of non-syndromic ichthyosis.

**Aim of the study**: to describe the different aspects of palmoplantar keratoderma (PPK) in ARCI and to establish a phenotype-genotype correlation.

**Method**: A 20-year retrospective study of patients with ARCI. Evaluation of the features of PPK in these patients according to their genotype.

**Results**: 41 patients

- PPK in 95.1% of cases
- NIPAL4 gene: Yellowish discolouration in 60% cases and diffuse involvement in 48% with plantar depression in 52%
- TGM1 mutation: Constant PPK but of variable intensity and constant palmar hyperlinearity. Brachydactyly in 87.5% of cases
- SULT2B1: Constant PPK; mild on the hands, moderate on the feet, sparing the plantar arches
- CYP4F22: Mild PPK with palmoplantar hyperlinearity
- CERSE: Aged appearance of the backs of the hands with palmoplantar hyperlinearity
- ABCA12: No PPK and no brachydactyly

## Effectiveness of a targeted high-throughput sequencing panel for the molecular characterisation of ichthyosis

Speaker: Constance Deblock (France)

- Inclusion criteria: Molecular analysis and clinical diagnosis
- Panel of 38 genes associated with ichthyosis
- Patients classified according to conclusive gene: class IV or V
- Inconclusive: No pathogenic variant or a single heterozygous variant
- Results: 90% of patients obtained a conclusive result
- Inconclusive results: 11% (n=33) had whole-genome sequencing: Five had a conclusive result

**Conclusion**: The panel was of interest because it was very effective, which was a quick step, but regular updating is essential. Whole-genome sequencing is useful for finding non-coding or structural variants.

## Ichthyosis linked to variants in the ELOVL1 gene: a rare and poorly understood syndromic form of ichthyosis

Speaker: Juliette Mazereeuw-Hautier (France)

Congenital ichthyosis is classified into three groups:

- Palmoplantar keratoderma (PPK)
- Non-syndromic ichthyosis
- Syndromic ichthyosis caused by various genes including ELOV1

ELOV1 is essential for the synthesis of long-chain fatty acids in the skin

ELOV1: AD form and AR form

Study of the c.494C>T variant (p.Ser165Phe)

Phenotype

- Cutaneous: erythematous and scaly plaques
- Neurological: delayed acquisition of walking or lack of acquisition associated with pyramidal signs
- Ophthalmological: Appearance of strabismus and nystagmus

Discussion: This ELOV1 variant is associated with a characteristic triad of dermatological, ophthalmological and neurological signs.

Neurological signs are at the forefront of the disease and are a major cause of its severity.

No correlation between cutaneous and neurological severity.

Pathophysiological hypothesis: dominant negative effect

Enables targeted therapeutic approaches to be considered by inhibiting the mutant mRNA using an antisense oligonucleotide approach.

## Efficacy and safety of topical gentamicin 0.1% in inherited epidermolysis bullosa

Speaker: Rajaa Bousmara (Morocco)

Inherited epidermolysis bullosa (IEB) is a genodermatosis that causes skin and mucous membrane fragility.

No curative treatment at present, but an emerging concept: stop-codon readthrough to enable synthesis of a complete protein.

Study: Evaluation of the effect of gentamicin 0.1% applied twice a day for 21 days.

Four patients had DEB and JEB.

The results showed very good, fairly rapid healing after one month, but this was marked by cases of recurrence over the longer term (three months on average).

Gentamicin B1 has strong nonsense mutation suppression activity and promotes collagen VII synthesis.

Gentamicin is a promising therapeutic option for IEB linked to nonsense mutations.

- Well tolerated
- Easy to apply
- Low cost

It is an alternative to gene and cell therapies, which are often inaccessible.

Intravenous gentamicin may provide improvement for up to six months and is well tolerated. Studies are under way to determine the appropriate doses for children in order to limit the side effects.

# VOLUNTARY COSMETIC DEPIGMENTATION (VCD) IN SUB-SAHARAN AFRICA: WHAT MANAGEMENT APPROACH SHOULD BE ADOPTED IN 2025?

Reports written by Ibrahima Traoré (dermatologist, Guinea)

Chair: Fatimata Ly (Senegal)

Speakers: Fatimata Ly (Senegal), Mariem Kebe Dia (Mauritania), Mame Thierno Dieng (Senegal)

## Voluntary cosmetic depigmentation (VCD): Background and motivations

Speaker: Mame Thierno Dieng (Senegal)

Mame Thierno Dieng introduced the issue of VCD by briefly tracing its historical background and socio-cultural roots. He explained how this practice, initially limited to certain social groups, has gradually spread in many countries in sub-Saharan Africa.

**Key points**

- VCD is based on an ancient phenomenon that has been influenced over time by aesthetic standards that value fair skin.
- The most common motivations include:
    - **The search for a beauty ideal associated with lighter skin**, perceived as a sign of modernity or social success.
    - **The influence of social pressure and social media**, reinforcing certain aesthetic standards.
    - **The impact of cosmetics marketing**, which is often aggressive and poorly regulated.
    - **Psychological motivations**, in particular self-esteem and the desire to belong.
- The most commonly used depigmenting agents: hydroquinone, potent corticosteroids (clobetasol propionate), mercury derivatives, injectable or oral glutathione, kojic acid.

Prof Dieng stressed the need to understand the underlying motivations in order to better guide prevention and management strategies, which should include an educational and socio-cultural component.

## Infectious complications of voluntary cosmetic depigmentation

Speaker: Mariem Kébé Dia (Mauritania)

Mariam Kébé Dia discussed the infectious complications associated with VCD, focusing on skin immunosuppression induced by the prolonged use of corticosteroids and other depigmenting agents.

**Key points**

- Dermatophytoses are the **most frequent** infectious complication, promoted by:

    - skin barrier impairment,
    - reduced local immune defences,
    - moisture and maceration associated with the products applied.
- Other possible infections (mentioned more briefly):
    - **bacterial**: impetigo, folliculitis, erysipelas;
    - **viral**: molluscum contagiosum, warts;
    - **parasitic**: secondarily infected scabies.
- Diagnosis is based on a careful clinical examination and, if possible, a mycological sample.

Prof Kébé Dia pointed out that management should combine anti-infectious treatment, discontinuation of dangerous depigmenting products and education about the risks of these practices.

## Non-infectious complications of voluntary cosmetic depigmentation

Speaker: Fatimata Ly (Senegal)

Fatimata Ly presented the non-infectious complications, which are often severe and largely underestimated, with a particular focus on the risk of cancer.

**Key points**

- **Squamous cell carcinoma**: a major complication linked to skin thinning, photosensitisation and prolonged exposure to depigmenting agents.
- Other non-infectious complications observed:
    - **Steroid dermatitis**: atypical stretch marks, skin atrophy, telangiectasias.
    - **Pigment disorders**: dyschromia, hypopigmented patches, exogenous ochronosis.
    - **Renal or neurological complications** due to mercury derivatives.

    - **Systemic effects** of corticosteroids in the event of massive use.

- The products concerned are the same agents frequently used in VCD: hydroquinone, mercury, fluorinated corticosteroids, glutathione and kojic acid.

Prof Ly stressed the urgent need to step up the regulation of depigmenting products, improve community awareness and develop appropriate management strategies, including rigorous dermatological monitoring.

## Dermatology on black skin: images to help you see more clearly

Report written by: Ibrahima Traoré (dermatologist, Guinea)

Chair: Émilie Baubion (Martinique University Hospital)

Speakers: Émilie Baubion (Martinique University Hospital), Emmanuelle Amazan (Martinique University Hospital), Gladys Ferrati-Fidelin (Martinique University Hospital)

The three speakers offered a session focusing entirely on clinical imaging, with the aim of improving the recognition of dermatoses on black skin, as this is an area that is still poorly represented in traditional dermatological literature. Rather than traditional theoretical teaching, they opted for an **interactive approach based on the projection and discussion of numerous clinical photographs**, promoting visual learning and diagnostic analysis.

The main objective was to improve diagnostic skills in the face of the multiple dermatological clinical presentations observed on dark skin. The speakers illustrated:

- common dermatoses,
- pigmentation disorders,
- autoimmune diseases,
- inflammatory dermatoses,
- skin infections,
- paediatric conditions,
- skin tumours,

- arboviral infections and other systemic diseases with cutaneous expression.

A secondary objective was to point out any particular therapeutic considerations when these diseases occur in black skin, particularly in cases where there is an increased risk of post-inflammatory dyschromia.

**Key points**

- A large number of clinical photographs were presented, covering a very wide range of dermatoses encountered on black skin in dermatological practice (without mentioning an exact number).
- The presentations stressed:
    - the importance of **recognising dyschromia**, which is essential in this phototype,
    - therapeutic precautions to avoid post-inflammatory hyperpigmentation,
    - the specific symptoms of certain dermatoses that present differently on black skin.
- The dynamic, visual approach covered a very wide range of clinical situations, making the session particularly educational.

It enabled participants to significantly improve their ability to recognise dermatoses on black skin thanks to the wide variety of clinical examples. The speakers demonstrated the need for specific training for this phototype, and enabled the dermatologists present to gain in diagnostic confidence and therapeutic relevance.

# PARADOXICAL SIDE EFFECTS OF IL-17 INHIBITORS

Report written by Nicolas Kluger (dermatologist, Finland) based on:

- Desjonqueres *et al.* Réactions paradoxales au bimékizumab dans l'hidradénite suppurée : série de cinq cas. P245
- Frédeau L *et al*. Intertrigos aseptiques sous-mammaires paradoxaux sous anti IL-1. P231
- Bertoli C *et al*. Anti-interleukines 17 : taux de maintien, efficacité, et tolérance dans le psoriasis de l'enfant. Cohorte internationale rétrospective en vie courante. CO 0022

The anti-IL-17 biotherapies currently available in dermatology in France include secukinumab and ixekizumab, two monoclonal antibodies specifically targeting IL-17A; brodalumab, which blocks the IL-17RA receptor and thus inhibits several IL-17 isoforms; and bimekizumab, which simultaneously neutralises IL-17A and IL-17F, offering broader inhibition of the IL-17 pathway. These treatments are used in moderate to severe forms of psoriasis requiring systemic management, after failure or intolerance of conventional treatments and hidradenitis suppurativa in the case of secukinumab and bimekizumab.

Paradoxical cutaneous reactions have been reported with all IL-17 inhibitors. The most common symptoms are psoriasiform (palmoplantar or pustular) and eczematous eruptions.

Desjonqueres *et al* (poster 245) reported several cases of psoriasiform reactions, particularly in the skin folds, in patients with HS treated with bimekizumab. Histology tended to support an eczematous and lichenoid reaction. Local corticosteroid therapy and methotrexate were proposed and discontinuation was not necessary.

Frédeau *et al* (poster 231) reported three patients with a similar eruption in the large folds under the breast, but also in the axillary and inguinal folds, for whom histology was again suggestive of eczema; they were taking various treatments for psoriasis (secukinumab, bimekizumab and ixekizumab).

It should be noted that in the SECU-Ped cohort of children with moderate to severe psoriasis treated with IL-17 inhibitors (Communication 022), a few cases of paradoxical eczema were observed with secukinumab (5/139, 3.5%) and ixekizumab (2/21, 9%).

| Paradoxical reactions to IL-17 inhibitors |
| --- |
| Psoriasiform reactions<br>• palmoplantar<br>• pustular<br>• skin folds<br>• ungual<br>Eczematous reactions<br>Behçet's disease<br>Sarcoidosis<br>Granuloma annulare<br>Pyoderma gangrenosum |

# ANIFROLUMAB IN LUPUS (1)

Report written by Nicolas Kluger (dermatologist, Finland) based on:

- De La Rochefoucauld *et al*. Facteurs prédictifs de réponse à l'anifrolumab dans le lupus cutané : une cohorte prospective de 57 patients. CO95

- Moschkowitz *et al*. Évaluation de la possibilité d'espacement des perfusions d'anifrolumab chez les patients lupiques répondeurs. CO94
- Le Bellour *et al*. Efficacité de l'anifrolumab dans les panniculites lupiques : étude rétrospective de 11 patients. CO96
- Mollet *et al.* Herpès vulvaire chronique résistant à l'aciclovir et induit par l'anifrolumab. P186
- Russo *et al*. Psoriasis paradoxal induit par l'anifrolumab chez une patiente atteinte de lupus cutané sévère : caractérisation par la médecine de précision. P236

Anifrolumab is a **monoclonal antibody** used to treat moderate to severe systemic lupus erythematosus by **blocking the type I interferon receptor**, an overactive pathway in lupus patients. It has had marketing authorisation since 2022 for systemic lupus with active skin involvement despite standard treatment.

Anifrolumab has **rapid and comparable efficacy across the different subtypes of cutaneous lupus**: discoid, subacute, acute and chilblain lupus. Efficacy appears to be better in patients with a moderate (rather than severe) CLASI activity score at treatment initiation. However, there are few predictive factors for response, apart from the level of interferon activity and the interferon signature (CO95).

It is **administered intravenously** (300 mg every four weeks) on an outpatient basis. This raises the question of **spacing out infusions** in patients in remission. A prospective study (still in progress) aimed to assess the relapse rate during dose spacing. Relapse was noted in a third of cases in a series of 78 patients with a median duration of 11.5 months after the start of spacing (22% at 12 months, with spacing at eight weeks) (CO94).

Le Bellour *et al.* reported significant efficacy with anifrolumab in a small series of patients with **lupus panniculitis** (LP). LP is a rare form of cutaneous lupus; it is often resistant to conventional therapies (hydroxychloroquine, oral corticosteroids, methotrexate, etc.). It may be isolated or associated with systemic involvement or other forms such as DL or chilblain lupus. The efficacy of the treatment was noticeable at six months, although some skin sequelae inherent in panniculitis were observed.

These results are encouraging in this form of cutaneous lupus, but the role of anifrolumab remains to be determined.

The **main infections** associated with anifrolumab are respiratory infections and viral skin infections, in particular herpes.

In the de La Rochefoucaud series, 23% of patients developed an infection, and in the Le Bellour series, 45% developed a viral infection as well as sepsis with a urinary origin.

Mollet *et al.* reported the case of a 34-year-old woman who developed valaciclovir-resistant chronic genital herpes on anifrolumab and valaciclovir (P186).

Lastly, **paradoxical reactions** such as psoriasis can occur with anifrolumab. By neutralising the interferon pathway, anifrolumab can disrupt a feedback loop, allowing uncontrolled Th17 hyperactivity and a permissive inflammatory environment for psoriasis, in people with genetic or epigenetic susceptibility to Th17 inflammation (P236).

# ANIFROLUMAB IN LUPUS (2)

Report written by Nicolas Kluger (dermatologist, Finland) based on:

- Lippert *et al.* Caractéristiques clinico-biologiques des dermatomyosites de l'adulte ayant des papules ou macules palmaires au diagnostic : cohorte rétrospective multicentrique française de 44 patients. CO92
- Ghorfi *et al*. Assocation exceptionnelle dermatomyosite - hypertrichose lanugineuse acquise lors d'un cancer du sein. P345
- Diop *et al*. Dermatomyosite juvénile : particularités cliniques et évolutives sur phototype foncé. P126
- Cordel *et al*. Érythème des paupières « en lunettes » : signe dermatologique de la maladie de Still. P343
- Le Clainche *et al*. Efficacité et tolérance de l'hydroxychloroquine dans l'atteinte cutanée de la dermatomyosite: étude rétrospective multicentrique de 102 patients. CO93

- Estenssoro-Alvarez *et al*. Réponse spectaculaire à l'anifrolumab d'une dermatomyosite juvénile avec atteinte cutanée sévère et réfractaire à de multiples traitements. P105

*Clinically,*

Palmar papules are most often associated with anti-MDA5 antibody-positive dermatomyositis (DM) but are by no means specific to this form. Could they be used as markers to identify patients at risk of developing interstitial lung disease? (CO92)

DM can be paraneoplastic and associated with other paraneoplastic syndromes, as in a case of acquired hypertrichosis lanuginosa with a poor prognosis (P345).

On melanin-rich skin, juvenile dermatomyositis (JDM) presents with classic symptoms such as erythema or oedema of the eyelids as well as Gottron papules on the backs of the hands. Emphasis should be placed on the hypopigmented poikilodermic appearance of the trunk, which is particularly noticeable on dark skin and can be disconcerting at first sight. Necrotic lesions appear achromic (P126).

Lastly, periocular/palpebral involvement in DM should not be confused with the fixed, painless, oedema-free periorbital erythema described by Cordel *et al.* in Still's disease (P343).

*Therapeutically,*

Hydroxychloroquine (HCQ) is considered to be effective against the skin damage caused by DM/JDM, either alone or in combination. However, there is little data in the literature and its efficacy varies widely from > 10% to 75%. A multicentre retrospective study compared 57 patients who had received HCQ and 45 who had not. In total, 46% of patients treated with HCQ achieved a complete skin response, compared with 75% in the group without HCQ. In multivariate analysis, only HCQ was significantly associated with a lower rate of complete response. Overall, HCQ appears to be associated with a lower rate of complete skin response. Prospective studies or studies involving larger numbers of patients are needed to confirm these results.

Anifrolumab could be a promising option in severe juvenile forms resistant to conventional therapies (P105).

# DOES DUPILUMAB INCREASE THE RISK OF CUTANEOUS LYMPHOMA IN PATIENTS WITH ATOPIC DERMATITIS?

Report written by Nicolas Kluger (dermatologist, Finland) based on

Neildez *et al*. Lymphomes T cutanés au cours d'un traitement par dupilumab : revue systématique de la littérature. CO116

Dupilumab is a human monoclonal antibody that blocks the action of the cytokines IL-4 and IL-13. It is indicated in particular for treating moderate to severe atopic dermatitis (AD), severe asthma, nodular prurigo, eosinophilic oesophagitis, etc.

A few clinical cases and retrospective series have pointed to a possible increase in the incidence of cutaneous T-cell lymphoma (cTCL) in AD patients on dupilumab.

Are these genuine dupilumab-induced cTCL cases, cases unmasked during AD or cases of cTCL misdiagnosed as AD?

A systematic review of the literature carried out by GFLEC and GREAT included 51 studies, consisting mainly of clinical cases (33) with six retrospective cohorts. A total of 547 patients were identified with lymphoproliferation, including 531 who were diagnosed with cTCL and were on dupilumab. The average age of the patients in this group was 58 (47% female), with AD in 95% of cases. cTCL preceded or was concomitant with the diagnosis of AD in 1.7% and 2.9% of cases respectively. A history of atopy was found in 56% of cases. Seventy percent had received systemic treatment, including cyclosporine in 61% of cases and methotrexate in 36%.

In 75% of cases, the cTCL appeared as an aggravation of pre-existing lesions, and in the remaining 25% as the formation of new lesions. The time to onset was nine months after initiation of treatment. In > 70% of cases, there was mycosis fungoides and in > 10%, Sézary syndrome. Retrospectively, five patients were found to have a diagnosis of cTCL prior to initiation of dupilumab, following re-examination of histologic slides. Clinically, the patients presented with erythroderma (23% of cases) and patches of mycosis (12%). Dupilumab was discontinued after cTCL was diagnosed in 75% of cases. For the patients with follow-up, 70%

went into remission after treatment and 13% died. There have been cases of spontaneous regression following discontinuation of dupilumab.

Based on this review, an expert consensus was established between the GFELC and GREAT members using the DELPHI method:

- Contraindication of dupilumab if there is a history of cTCL in a patient with AD
- Investigate/rule out cTCL before starting dupilumab when there is late-onset AD (> 40 years), absence of atopy and AD with an atypical clinical presentation. Carry out a skin biopsy to test for T-cell clonality. Erythrodermic AD or AD > 50% of body surface area should also be investigated by a specialist
- If there is atypical worsening or if new lesions appear during treatment with dupilumab, rule out cTCL
- If cTCL is confirmed, stop dupilumab, report the case to pharmacovigilance and treat the cTCL according to the aggressiveness of the clinical form
- If cTCL is not confirmed, close monitoring is recommended (repeat biopsies and blood tests if lesions persist), with a transition to methotrexate or phototherapy. Avoid cyclosporine, JAK inhibitors and other inhibitors of the Th2 pathway

All in all, this review of the literature points to a link with patients with late-onset adult AD, with no history of atopy, and with the aggravation of pre-existing lesions. When the bioavailability of IL-13 is increased on dupilumab, this cytokine could bind to the IL13Ralpha2 receptor, which is not blocked by dupilumab, but is over-expressed in certain types of cancer. This suggests that reported cases more likely represent unmasked or previously undiagnosed cTCL. The other anti-Th2 biotherapies used in AD (tralokinumab, lebrikizumab) have a different mechanism to dupilumab, but there is currently insufficient hindsight to rule out a risk.

# JAK INHIBITORS AND SERIOUS CARDIOVASCULAR EVENTS IN AD: REASSURING RESULTS

Report written by Nicolas Kluger (dermatologist, Finland) based on

Neildez *et al*. **Risque d'événement cardiovasculaire grave chez des adultes traités par inhibiteurs de JAK pour une dermatite atopique : une étude de cohorte nationale à partir du Système national des données de santé. CO117**

Several JAK inhibitors are currently available for the treatment of atopic dermatitis (AD). However, an increase in the risk of major adverse cardiovascular events (MACE) observed in patients over the age of 50 with at least one cardiovascular factor who were treated with tofacitinib for rheumatoid arthritis prompted the health authorities to issue recommendations regardless of the indication, including AD.

Using the French National Healthcare Data System, patients > 18 years of age who had received a JAKi (n = 4300) or a biotherapy (n = 23,574) for AD between 2018 and 2024 were identified and each cohort was compared to a general population cohort.

In the cohort of AD patients taking a JAKi, there was no increased risk of MACE (heart attack, ischaemic stroke) compared with the general population. This may have been due to the application of the health authorities' recommendations, with limited use of the treatment in at-risk patients and with appropriate patient selection. Other foreign studies also have found no link between MACE and the use of JAKis in AD.

An increased risk of MACE was noted in the biotherapy group before initiation of treatment and during the 1st year of exposure, which raises the question of the role of systemic inflammation in severe AD as an independent cardiovascular risk factor.

# RECOMMENDATIONS FOR THE MANAGEMENT OF ATOPIC DERMATITIS

Report written by Nicolas Kluger (dermatologist, Finland)

Speaker: Prof Marie-Sylvie Doutre (France)

During the Hot Topics session, Prof Marie-Sylvie Doutre presented the new French recommendations for the management of atopic dermatitis (AD), which were developed and adapted based on the European recommendations published in 2022. They were also published in full in the December 2025 issue of Les Annales de Dermatologie et de Vénéréologie - FMC.

| **Table 1. For all patients** |
| --- |
| **Daily use of emollients**<br>**Short, lukewarm baths and/or showers, cleansing products without allergens or irritants** |
| **Comprehensive management, considering the impact of AD. Offer psychological support if necessary. Discuss corticosteroid phobia at the 1st consultation.** |
| **Offer a patient education programme** |
| **Avoid smoking among patients and parents** |
| **Avoid irritating clothing** |
| **Food diversification at the age of four months for all foods. No food avoidance or preventive diet.** |
| **No systematic allergy testing. Allergy testing if immediate IgE-mediated reaction (urticaria, angioedema, flushing, digestive problems) or worsening of moderate to severe AD with suspected worsening six to 48 hours after food intake.** |
| **If allergic, limit exposure to known allergens** |

| **Table 2. Treatment of flare-ups** |
| --- |

| |
|---|
| **Potent topical corticosteroids for the body and moderate topical corticosteroids for the face**<br>**Once a day, until symptoms disappear without gradual tapering** |
| **Topical calcineurin inhibitors on areas at risk of atrophy: face, eyelids, skin folds, ano-genital region** |
| **Potent topical corticosteroids with wet wrapping if local care needs to be optimised** |

| **Table 3. Proactive treatment for frequent recurrences** |
|---|
| **Potent topical corticosteroids for the body and moderate topical corticosteroids for the face**<br>**Twice a week on sites usually affected** |
| **Topical calcineurin inhibitors applied twice a day to the sites usually affected and in particular to areas at risk of atrophy** |

| **Table 4. Treatment of infections** |
|---|
| **Bacterial infection (impetigo) Local or systemic treatment depending on the extent of the lesions. Continuation of TCs and TCIs subject to antibiotic treatment. Antiseptics are unnecessary.** |
| **Viral infection (herpes) Systemic antiviral treatment without waiting for virological results. Suspend TCs and TCIs for at least 48 hours after starting herpes treatment.** |

| **Table 5. Systemic treatment** |
|---|
| **In the event of poorly controlled AD (SCORAS > 50 and/or major impact on quality of life) despite appropriate and well-administered local treatment**<br>**or if unable to treat locally**<br>**or if > 4 tubes of 30 g (> 120 g) of TCs per month in adults are needed to control AD** |
| **Cyclosporine 4 to 5 mg/kg/day as a short course (18 years and over)***<br>**Or biotherapy*: dupilumab (six months and over), lebrikizumab (12 years and over), tralokinumab (12 years and over)** |

| |
|---|
| **Or JAK inhibitors*: abrocitinib (12 years and over), baricitinib (two years and over), upadacitinib (12 years and over) [take into account the ASNM recommendations of March 2023]**<br>***There is no hierarchy between treatments**<br>**Systemic corticosteroids are not recommended in the treatment of AD** |
| **Alternative options**<br>**Methotrexate (off-label)**<br>**UVB phototherapy** |

| **Table 6. In the event of remission on systemic treatment** |
|---|
| **As soon as remission is achieved on cyclosporine, reduce the dose but do not exceed a total duration of one year** |
| **After 16 weeks of treatment, administer lebrikizumab and tralokinumab by injection every four weeks** |
| **After six months of remission,**<br>**Reduce methotrexate to the minimum effective dose**<br>**Space out dupilumab injections**<br>**Reduce the JAKi dose to half of the original dose** |

# JAKIS: WHAT ARE THE SIDE EFFECTS FOR OUR PATIENTS?

Report written by Nicolas Kluger (dermatologist, Finland)

Based on Dereure O. Autres indications, tolérance et suivi. FMC01 Anti-JAK : les dermatologues aussi !

The side effects of JAK inhibitors (JAKis) are frequent, especially with first-generation broad-spectrum “pan-JAK” inhibitors.

| **Table 1. Complications of JAKis** | |
|---|---|
| **Specific to the mode of action** | **Not specific to the mode of action** |

| | |
|---|---|
| **Infection**<br>**Tumour**<br>**Inflammation**<br>**Haemostasis (thrombosis)**<br>**Various metabolic pathways (weight gain, etc.)** | Allergic: urticaria, angioedema, drug-induced dermatitis<br>Other: acne, headaches, gastroenterological (perforation, etc.) |

**Infectious complications** are the most common, particularly with broad-spectrum JAK inhibitors, and include bacterial, viral and fungal infections. However, they are **usually benign**.

**Serious infections** include urinary tract infections, digestive tract infections (colitis), skin infections (secondary infection of wounds) and viral infections such as **herpes and shingles**, which is why it is so important to keep up to date with shingles vaccinations. Live vaccines are not permitted during treatment, and it is strongly recommended that vaccinations be updated before treatment. For vaccination with a live vaccine on JAKis, treatment should be discontinued for three months and resumed one month after vaccination.

To minimise the **risk of serious side effects** (cardiovascular disease, thrombosis, cancer and serious infections), the Pharmacovigilance Risk Assessment Committee (PRAC) of the European Medicines Agency has issued the following general recommendations for JAKis and for all chronic inflammatory diseases; they also apply to atopic dermatitis and alopecia.

- **JAKis should be avoided in patients aged > 65** unless no therapeutic alternatives are available, and in subjects at increased risk of cardiovascular disease as well as smokers and former smokers
- **Use with caution** in patients at risk of venous thromboembolism
- **Dose reduction** in patients potentially at risk of cardiovascular disease, venous thrombosis or cancer

However, it should be remembered that, at present, patients with AD or alopecia generally do not have the same profile as elderly patients with inflammatory diseases such as rheumatoid arthritis. Furthermore, the literature relating to our populations is reassuring as to the risks of

serious morbidity associated with the use of JAKis. Nevertheless, these risk factors should be borne in mind for specific patients (smokers, patients on combined oestrogen-progestin contraception, obese patients, etc.). An article was published in 2024 in Les Annales de Dermatologie et de Vénéréologie - FMC (Staumont Sallé D, *et al*. Aide à la prescription des inhibiteurs de JAK en dermatologie : évaluation du risque cardiovasculaire et thrombotique veineux et conduite à tenir).

Lastly, in terms of tumours, there is an increased risk of **squamous cell skin cancer**, mainly in the head and neck region, only with ruxolitinib and exclusively in haematological indications (to date).

| **Table 2. Pre-therapy assessment of JAKis in dermatology** |
|---|
| **Blood count**<br>**Renal and liver function tests**<br>**Lipid profile**<br>**CPK**<br>**Blood tests: HBV, HCV, HIV +/- VZV**<br>**Screening for tuberculosis (Quantiferon)**<br>**Chest X-ray** |
| **Pregnancy test/contraception for women** |
| **Assessment of cardiovascular, thrombo-embolic and tumour risks** |
| **Patient information** |

# NAEVUS AND MELANOMA IN CHILDREN. THE KEYS TO REASSURANCE

Report written by Nicolas Kluger (dermatologist, Finland)

Based on **Boccara O. Naevus chez l'enfant. Quand faut-il s'inquiéter ? Forum FO13 Prolifération mélanocytaires de l'enfant**

Olivia Boccara gave a reassuring lecture on naevi in children. It is important to remember that the rules of surveillance as applied to adults in terms of the diagnosis and monitoring of naevi do not apply to children. In fact, childhood is a period when naevi physiologically appear (acquired naevi) and, by definition, are growing, sometimes even from birth. In addition, there is little or no cumulative sun exposure. The appearance of a new naevus is nothing to worry about and its growth, whether in appearance or size, is a normal phenomenon. It should also be remembered that trauma does not increase the risk of melanoma.

The incidence rate of melanoma in 0-14-year olds is 1.74 per 1 million people/year, i.e. 60 times lower than in adults and children aged over 15, according to an American study. SSM is virtually non-existent in children, even though it is "the" type of melanoma in adults. Melanoma in naevi is very rare, especially in children under 10, and melanoma in giant congenital naevi is also uncommon. On the other hand, a child with several dozen naevi needs more attention.

The ABCDE criteria should not be applied to children.

In the latter, melanomas are often raised and unpigmented. They are not irregular and not very wide. An ABCD acronym has been proposed for children: **A**melanotic; **B**ump, bleeding; uniform **C**olour; **D**e novo, any **D**iameter. In short, **we should be concerned about a recent achromic/erythematous nodule that is growing rapidly**. However, even with these criteria, a classic (benign) Spitz naevus can meet them.

Lastly, isolated longitudinal melanonychia in children is almost exclusively benign and simple monitoring is sufficient, with pigmentation that sometimes regresses.

# POTPOURRI: SOME RARE AND NEW DISORDERS IN DERMATOLOGY

Report written by Nicolas Kluger (dermatologist, Finland) based on:

- Benzaquen M and Rongioletti F. Nouvelles entités et dermatoses inhabituelles ou trompeuses à connaitre en pratique. FMC079
- Kluger N. Tout ce que vous avez toujours voulu savoir sur la dermatologie et la cosmétologie de la peau asiatique. FMC029
- El Lakkis *et al*. Une dermatose en or / une dermatose oubliée. Top 12 des Juniors
- Bois F *et al*. Quand la radiothérapie fait parler la peau. Laroche Posay Dermacademy symposium

Focal epithelial hyperplasia (FEH or Heck's disease) is a benign condition of the oral mucosa caused by HPV infection, mainly types 13 and 32. It mainly affects children and teenagers, but can also occur in adults. It is rare. In Europe, it is common in cases of HIV infection or organ transplantation. It manifests as multiple small, soft, painless, non-ulcerated pinkish or whitish lesions, mainly on the lips, cheeks and tongue. The disease is non-cancerous and often self-limiting, disappearing spontaneously within a few months or years. Diagnosis is usually clinical and is sometimes confirmed by biopsy with HPV PCR for typing. Treatment is not always necessary, and observation may be sufficient, but curettage, cryotherapy, imiquimod or CO2 laser ablation may be proposed for functional or aesthetic reasons.

Confluent and reticulated papillomatosis (CRP) of Gougerot and Carteaud is a rare, benign dermatosis that mainly affects adolescents and young adults. It presents as brownish, warty patches that are confluent in the centre and reticulated around the edges and are located mainly on the trunk and neck. Its origins are poorly understood, involving disordered keratinisation and sometimes abnormal bacterial flora. Diagnosis is clinical, ruling out mycosis. Treatment is based mainly on antibiotics (minocycline) or keratolytic agents, with good results.

| **Table 1. CRP diagnostic criteria according to Davis *et al.* (BJD 2006)** |
| --- |
| **Scaly brown macules and plaques, appearing at least partly reticulated and papillomatous** |

| |
|---|
| **Trunk and neck involvement** |
| **Negative mycological samples** |
| **Lack of response to antifungal treatment** |
| **Excellent response to cyclins** |

**Facial discoid dermatosis** is a rare chronic inflammatory condition mainly affecting the **face**; it was first described in 2010 by Ko *et al.* (Int J Dermatol 2010). It presents as **rounded or discoid plaques** that are erythematous, pink or orange, sometimes scaly, and well demarcated Lesions occur mainly on the **cheeks, forehead and chin**. They can be pruritic but are often not very symptomatic. Some 40 cases have been described to date. It seems to affect women more frequently. Histology is not very specific, showing psoriatic epidermal hyperplasia, hyperkeratosis with parakeratosis, lymphohistiocytic perivascular infiltrate, follicular plugs, spongiosis and demodex. The aetiology remains poorly understood. Diagnosis is clinical and is sometimes supplemented by a biopsy. Differential diagnoses include discoid lupus, superficial pemphigus and seborrhoeic dermatitis. Treatment is based on **topical corticosteroids** with or without calcipotriol and acitretin. The condition is generally chronic, with lesions remaining stable for years and with a disappointing therapeutic response. However, it is benign, with no systemic involvement. Its individualisation is not clear. Is this a distinct condition or a specific presentation of psoriasis, seborrhoeic dermatitis or demodecidosis?

**Linear lupus panniculitis of the scalp** is a rare form of cutaneous lupus that mainly (but not exclusively) affects young men from South-East Asia. It is asymptomatic and progressive, with a linear, annular or arciform distribution, following Blaschko's lines and affecting any region of the scalp.

It results in non-scarring alopecia with possible regression and regrowth of hair, and rarely with systemic involvement. Antinuclear antibodies are positive in 50-60% of cases. Treatments include hydroxychloroquine, local or oral corticosteroids and even methotrexate.

**Prurigo pigmentosa** is a chronic inflammatory and pruritic dermatosis that develops in flare-ups. It was described by Nagashima in the 1970s and the condition is well known in Japan and Korea. It tends to affect younger women (aged 11-30).

It has a particular proximal distribution, affecting the neck, trunk, back and shoulders. The initial papular lesions become crusted, giving way to hyperpigmented macules that last for several months. As the disease progresses in flare-ups, lesions of different ages coexist and some coalesce, giving a reticulated appearance. A ketogenic diet, diabetes and anorexia nervosa, as well as friction and stress, are factors that can trigger the disease. Treatment is based on antibiotic therapy with cyclins, macrolides or dapsone.

**Cutaneous plasmacytosis** is a chronic "haematological" disease that most frequently affects individuals from South-East Asia. It takes the form of macules or papules that are not very specific and are hyperpigmented, asymptomatic and chronic, appearing mainly on the trunk. Skin biopsy shows a perivascular infiltrate of mature plasma cells in the dermis and hypodermis. Biologically, there is an inflammatory syndrome with hypergammaglobulinaemia (more polyclonal than monoclonal). If a second internal organ is affected, with an infiltrate of mature, well-differentiated plasma cells without atypia, the disease is classified as "systemic" plasmacytosis. Obviously, "classic" causes of cutaneous plasmacytosis such as syphilis and borreliosis should be ruled out. In addition, every effort should be made to rule out lymphoma. Plasmacytosis may be associated with Castleman disease, IgG4-related disease, lymphoma, etc. Various treatments can be proposed, including oral corticosteroid therapy, thalidomide and PUVA therapy. This is a disease generally managed in a university setting.

Eosinophilic polymorphic and pruritic eruption associated with radiotherapy (**EPPER**) syndrome is a rare skin complication associated with radiotherapy. It manifests as a pruritic polymorphic eruption on all four limbs (sparing the head and beginning at the radiotherapy site) that is made up of papules, plaques or vesicles. **Blood eosinophilia** is frequently observed. Histology shows an eosinophilic perivascular infiltrate in the dermis. The exact mechanism is poorly understood, but an immuno-allergic reaction induced by irradiation is suspected. The syndrome generally appears during or after radiotherapy. It can sometimes be delayed by several months. Treatment is based on topical or systemic corticosteroids and antihistamines. The outcome is usually favourable after treatment, but it may resolve spontaneously.

**Accidental exposure to mercury** is still possible in France, as illustrated by the case of an auctioneer who developed acute generalised exanthematous pustulosis (AGEP) with

pulmonary involvement and an inflammatory syndrome. Exposure to mercury was probably occupational via the handling of antiques.

# TOXIN-MEDIATED DISEASES CAUSED BY *STAPHYLOCOCCUS AUREUS*

Report written by Nicolas Kluger (dermatologist, Finland) based on

Tristan A. **Quand *Staphylococcus Aureus* s'attaque à la peau. Le staphylocoque doré dans tous ses états FMC047**

Dr Anne Tristan from the French National Research Centre for Staphylococci outlined the epidemiology, bacteriology and different presentations associated with *Staphylococcus aureus* toxins.

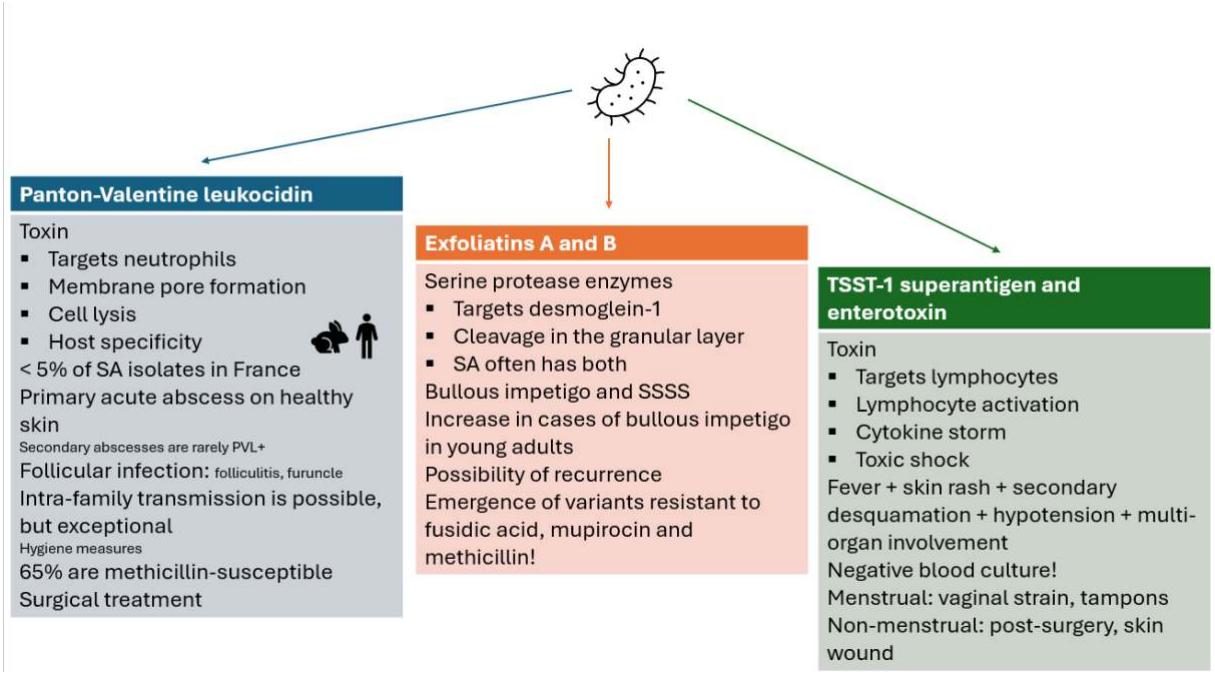



Figure 1. Toxin-mediated clinical presentations of *Staphylococcus aureus*