BIODERMA
LABORATOIRE DERMATOLOGIQUE

INTERNATIONAL MEDICAL

Bioderma Congress Reports
JDP 2022

Reports written by

Dr. Laura BOUCHARD

Dermatologist, Finland

All these small interventional actions on the nail apparatus that terrify you... As well as your patients!

Dr Villani presented training in preoperative consultation and nail apparatus injections.

As part of the preoperative consultation, he recalled the importance of **informing** the patient of possible **pain, infection, risk of nail dystrophy, definitively narrower nail and relapse** after the procedure. Thus, **postoperative care** must be explained and patients are sent a preoperative letter reminding them in particular to bring a scarf if they are to undergo finger surgery, to bring wide, open-toed shoes for toe surgeries, to be prepared to elevate the operated limb for 48 hours and walk as little as possible for 48 hours in the event of a toe surgery.

Anaesthesia of the nail apparatus with a 2-3 ml **Luer Lock syringe** and a **fine needle** (30 G) for gentle injection. Choice of anaesthetic: bupivacaine, ropivacaine, lidocaine with or without adrenaline; ropivacaine has the most lasting effect but its price is higher (3x that of lidocaine). Lidocaine combined with adrenaline should not be used in vasospastic patients and the speaker saw no merit in using the adrenaline version. The effect of lidocaine lasts for 2 hours, that of bupivacaine for 8 hours (but only acts after 30 min) and that of ropivacaine for 8 to 12 hours. Ropivacaine injection is less painful if concentration is less than 5mg/ml.

Anaesthesia by distal digital block is often favoured. Anaesthesia by matricial block can be carried out by spreading from a proximal injection site.

Local **injections of triamcinolone** 10 mg/ml every 3 to 8 weeks to treat **psoriasis**, mild to moderate **lichen planus, chronic paronychia**. For chronic paronychia, the **treatment of external causes** is crucial to successful treatment, i.e. limiting hand washing to 3-4 times per day, wearing gloves while peeling vegetables, etc. to combat maceration as well as the Koebner phenomenon while avoiding all external traumatic factors.

To treat **psoriasis**, injections of **methotrexate** (MTX 2.5 mg/ml – 0.1 ml/finger – every 6 to 8 weeks), **cyclosporin** and a case of **secukinumab** were used in addition to **triamcinolone**. A study compared MTX with triamcinolone and cyclosporin. A 75% improvement was achieved in 50% of the

triamcinolone group vs 56.7% of the MTX group and 33.3% of the cyclosporin group (Mittal et al. Indian j Dermatol Venereol Leprol 2018; 84: 419-423).

He pointed out the need to raise the issue of **mechanical stress** when analysing a nail and to examine shoes for signs of anterior, anterosuperior or lateral stress.

Poster session: Side effects of Covid-19 vaccination

Elkissouni et al. from the Casablanca University Hospital, reported 2 cases of **generalised morphea** after Covid-19 vaccination. In both cases, a 62 year-old man and a 59-year old woman, with no history of morphea, developed skin induration, initially locally, gradually extending to the trunk and limbs without affecting the face and extremities. The results for antinuclear (ANA) and anti-Scl70 antibodies were negative. The biopsy was consistent with deep morphea. The patients were prescribed oral corticosteroids, which led to a positive outcome.

Sueur et al. from the Besançon University Hospital reported the case of a 92-year-old female patient who experienced a **bullous fixed pigmented erythema** that progressively worsened and evolved into a widespread form with mucosal involvement further to Covid 19 vaccination. The first rash developed the day after the 2nd Pfizer/Comirnaty Covid19 vaccine in the form of isolated, round erythematous lesions on both hands and one leg. The day after the 3rd dose, similar lesions appeared, albeit more diffuse, with dysphonia. The Moderna vaccine was picked as 4th dose and, the following day, she experienced oval lesions over her entire body, maculopapular, purplish erythematous, sometimes bullous, and mediodorsal skin detachment in addition to mucosal involvement with sublingual aphthoid lesions, erosions of the inside of the cheek and lips.

The skin biopsy confirmed the diagnosis of fixed pigmented erythema determined through clinical examination. Patch testing for vaccines and their excipients was negative. Subsequently, the patient contracted the virus and experienced a further recurrence of rash, with reactivation of the older lesions and the appearance of new ones.

Ouni et al. from the Farhat Hached Hospital, Sousse, Tunisia described a case of **dermatomyositis** that appeared 2 weeks after the 1st dose of Pfizer/Comirnaty vaccine. The 19-year-old female patient experienced a heliotrope rash on the upper eyelids, forehead and cheeks combined with photosensitivity, erythematous stripes on the back of the hands with keratotic purplish erythematous papules opposite the extensor surfaces of the metacarpophalangeal and interphalangeal joints. Muscle weakness predominantly affecting the shoulder and pelvic girdles.

Antinuclear antibodies and auto-antibodies specific to myositis were negative. The electromyography revealed a myogenic involvement and the skin biopsy showed interface dermatitis with perivascular inflammatory infiltrate and an oedema of the dermis, while direct immunofluorescence was negative. The patient was prescribed oral corticosteroids, which led to a positive outcome after 3 weeks.

Manaa et al. from the Farhat Hached Hospital, Sousse, Tunisia, present a case of **psoriasis** and **cutaneous B-cell lymphoma** which occurred 10 days after Covid19 vaccination. An 82-year old male patient, with not prior history, was hospitalised for a widespread psoriasiform rash combined with nodules on the left leg that had grown for 2 months and had appeared 10 days after the ASTRAZENECA vaccine. The histological aspect was indicative of psoriasis of erythemato-squamous plaques and of large B-cell lymphoma, leg type, on a leg tumour with the following immunohistochemistry: CD20+, CD3-, CD30-, Mum1+, Bcl2-. A blood test confirmed

thrombocytopenia, microcytic anaemia, high LDH levels, and satisfactory liver and kidney function tests.

They speculated about the role of the Covid19 vaccine in the induction or worsening of psoriasis and said it could be linked to the production of interferon 1 and interleukin 6 and the recruitment of Th17 cells involved in the physiopathology of psoriasis. 3 cases of cutaneous lymphoma were reported further to the covid19 vaccine. In our case, the viral proteins introduced by the vaccine may induce immune dysregulation and chronic antigen stimulation, which could explain, in a predisposed subject, the origin of lymphoma.

Bailly-Caillé et al., from the Caen and Rouen University Hospitals, reported a case of **p200 pemphigoid**. A 74-year-old male patient experienced a vesicular rash on the wrists which occurred 10 days after the first dose of the Moderna vaccine; followed by, within 48 hours of the second dose, a bullous rash on the extremities with no associated mucosal involvement. The histological analysis showed a detachment of the dermal-epidermal junction with a deposit of IgG and C3 along the epidermal basement membrane highlighted by direct immunofluorescence. The detection of anti-BPAG1 and 2 antibodies was negative. Indirect immunofluorescence found marking on the dermal side. Immunoblotting revealed the presence of antibodies which recognise the 200 kDa protein, hence the diagnosis of p200 pemphigoid. A case of P200 pemphigoid following vaccination was described, after a pneumococcus vaccine. The patient was once again vaccinated against sars-cov 2 one year later by Nuvaxovid, a non-mRNA, recombinant protein vaccine with no adverse effects.

Walid et al. from the Ibn Rochd University Hospital, Casablanca, Morocco, have reported a case of **amyopathic dermatomyositis** resulting from the Covid 19 vaccine. A 60-year-old female patient was admitted for a pruritic rash which appeared 5 days after she received her second dose of the Astrazeneca vaccine, combined with photosensitivity and Raynaud's phenomenon. She exhibited facial erythroedema, flagellate erythema of the décolleté, Gottron papules and infiltrated erythematous plaques on the extensor surfaces of the forearms as well as a periungual erythema, trachyonychia and cuticle thickening. The nail dermatoscopy highlighted megacapillaries, a splinter haemorrhage, trachyonychia and unstructured avascular zones indicative of nail hypoperfusion. She was suffering from moderate myalgia and the blood test showed elevated levels of CK, AST and LDH, positive anti Mi-2 antibodies and ANAs, while the ENMG revealed a myogenic syndrome. The CT scan did not reveal any interstitial pulmonary involvement or any signs of malignancy. A skin biopsy was performed, showing keratinocyte necrosis. The pharmacological investigation exposed the responsibility of the Covid 19 vaccine in the induction of the disease.

Poster session: Use of spironolactone in acne in adolescent females

Canu et al. from the Bordeaux University Hospital assessed the efficacy and safety of spironolactone in the treatment of acne in **11 adolescent females** monitored from 2015 to 2022.

- Acne starting age (average): 9.5 (4-12)
- Age at which spironolactone is introduced (average): 13.6 (12-18)
- GEA grade:

- GEA 2 (1/11)
- GEA 3 (4/11)
- GEA 4 (4/11)
- GEA 5 (2/11)

- Dose of spironolactone (average): 125 mg (75-150 mg)

- Treatment duration (average): 15 months (3-51 months)

Associated systemic treatment
- None 5/11
- Antibiotic (cyclin, azithromycin) 3/11
- Oral corticosteroid therapy 2/11
- Oestrogen plus progestin pill 1/11 (during treatment)
- Isotretinoin (during treatment)

After 3 months
- Partial response < 50%: 1/11
- Partial response > 50%: 10/11

At the end of treatment
- Complete response 1/8
- Partial response < 50%: 1/8
- Partial response > 50%: 5/8
- No response 1/8

Why opt for spironolactone?
- Isotretinoin: risk of early growth plate fusion
- Antibiotics: no antibiotic resistance
- Oestrogen plus progestin pill:

- Refusal by some patients/parents
- No room before the beginning of menstruation

In conclusion
- Spironolactone is very well tolerated in their cohort
- Spironolactone is very effective in adolescent females
- Interesting alternative to other forms of treatment, in particular cyclins

---

Management of cutaneous ageing: personalised treatment plans

During the session on cutaneous ageing, **Doctor Martine Darchy** presented studies on the benefits of taking oral collagen and hyaluronic acid.

Oral collagen: a meta-analysis of 19 studies, with a total of 1,120 participants aged 20 to 70 and 95% of women aged 20 to 70, showed the positive results of hydrolysed collagen supplements compared with placebo on skin hydration, elasticity and wrinkles. The result was assessed after ingesting hydrolysed collagen for 90 days (De Miranda et al., Int J Dermatol 2021; 60: 1449-1461).

Oral hyaluronic acid (HA): 3 Asian studies showing some success on reducing wrinkles. In a randomised, double-blind placebo-controlled trial, 60 Japanese men and women aged 22 to 59 were treated with 120 mg per day of 2 or 300 kDa HA or placebo for 12 weeks. An analysis of skin imprints on the crow's feet wrinkles and a subjective assessment questionnaire (wrinkles, skin radiance and suppleness) showed a significant improvement in wrinkles in the 300 kDa HA group after 8 weeks, and slightly lower with 2 kDa HA compared with placebo. Oe et al., Clin Cosmet Invest Dermatol 2017; 10: 267-273.

Two more studies showed the same thing, one with 240 mg of 38 kDa HA per day for 8 weeks in a group of 14 Japanese subjects in the HA group versus placebo ($p<0.01$) (Watanabe et al., Jpn Pharmacol Ther 2015; 43: 57-64) and the other in 26 Korean subjects receiving 240 mg of 75 kDa HA for 8 weeks versus placebo ($p<0.05$) (Kim et al., Food Style 2007; 11:42–46). However, the effect of epidermal and dermal HA is much greater and oral HA is no substitute for injections, but could serve as adjunctive and maintenance treatment. Thus, it seems that the use of HA in combination with biotin, vitamin C, copper and zinc increases its absorption by 31.5%. (Göllner et al., J Evid Based Complementary Altern Med 2017; 22:816-823).

Poster session: Chronic radiation dermatitis, which treatment should be proposed in the event of failure of pulsed dye laser?

Baqays et al., from the Toulouse University Hospital, reported 3 cases of chronic radiation dermatitis resistant to pulsed dye laser (PDL) treated with PDL/Nd:YAG multiplex vascular laser

Introduction

- Chronic radiation dermatitis characterised by telangiectasias and changes in skin texture
- Primary treatment pulsed dye laser (PDL)
- No data available in the event of PDL failure

Objective

- Analyse the percentage of PDL-resistant radiation dermatitis
- Evaluate the improvement achieved by the treatment with PDL/Nd:YAG multiplex vascular laser

Methods and Results

- Identification of female patients treated for radiation dermatitis between 2015 and 2021
- Clinical photos used for comparison
- 5 patients identified
- Treatment initiated with PDL
- Only 2 patients (40%) treated with PDL showed satisfactory clinical improvement
- 3 patients did not respond to PDL (60%)
- Treated with PDL/Nd:YAG multiplex laser, with good results

Treatment settings

- Patient 1 (radiation dermatitis after breast carcinoma) 4 sessions

PDL 7 J/cm2, 20 ms, YAG 40 J/cm2, 20 ms, 7mm handpiece

- Patient 2 (radiation dermatitis after radiotherapy for mycosis fungoides of the thigh) 9 sessions

PDL 6-8 J/cm2, 20 ms, YAG 30-40 J/cm2, 20 ms, 7mm handpiece

- Patient 3 (radiation dermatitis after breast carcinoma) 10 sessions

PDL 12J/cm2, 40 ms, YAG 50 J/cm2, 40 ms, 7mm handpiece

Discussion and Conclusions

- They present 3 cases of PDL-resistant chronic radiation dermatitis successfully treated with a combination of PDL and Nd:YAG
- PDL/Nd:YAG multiplex vascular laser can be proposed in female patients with chronic radiation dermatitis if they do not respond to PDL

Comment

The addition of Nd:YAG to PDL improved the response to treatment. However, radiation dermatitis responds better to short pulses (0.45-1.5-3-6 ms) and PDL failure may be due to the settings. While Nd:YAG could be a good addition on limbs or skin with a higher phototype, it should be used with caution on irradiated tissue given its deeper penetration.

Poster session: Study of the prevalence of skin cancer in a retrospective cohort of homeless patients

Bergeret et al. from the Nîmes University Hospital reported the diagnoses of skin cancer in a group of 1,990 homeless men and women who were seen by a volunteer general practitioner with experience in dermatology for a one-year period independently of the reason for consultation.

Population
Male
Female
Median age
M/F ratio
Dermatological consultations
Skin cancer
No social security coverage
Addictions
Tobacco
Alcohol
Others

General
1,683 (83%)
347 (17%)
38
5
13.4%
105 (5%)
966 (44%)

1808 (90%)
733 (37%)
288 (14%)

Included
88 (84%)
17 (16%)
45
5

48 (42%)

52 (50%)
23 (22%)

12 (12%)

Skin cancers diagnosed
Basal cell carcinoma (n = 44; median age 40)
- 19 superficial BCC
- 19 nodular BCC
- 6 invasive BCC

Squamous cell carcinoma (n = 34; median age 45)
- 34 including 19 invasive

Melanoma (n = 7; median age 48)
Cutaneous metastases of visceral cancer (n = 8)
Cutaneous lymphoma (n = 6)
- 5 mycosis fungoides
- 1 Sézary syndrome

Other cancers (n = 5)
Tumour in photoexposed area 66%

Conclusions
First study of this kind in France
4% of the homeless had skin carcinoma
4‰ had melanoma
1% had rarer forms of skin cancer
The age of onset of skin carcinoma seems to be earlier than in the general population.
These results show the merit of screening skin cancer in this disadvantaged population who often have no social security coverage and are difficult to treat.

Facial dermatosis: appropriate management of a red face in atopic patients

Prof Delphine Staumont-Sallé, Lille Regional University Hospital

During the facial dermatosis session, Pr Delphine Staumont-Sallé presented a cavalcade of clinical cases of red faces in atopic patients.

Red Face patients: new appearance/worsening/resistance to AD treatment

1st patient
32-year-old male
Severe AD since childhood
Squamous state of the scalp, cheeks, torso

AD + seborrhoeic dermatitis (Head & Neck Dermatitis)

Change in skin microbiota/fungal microbiota

Contributory factors
- Malassezia: pro-inflammatory role (possibility of specific IgE)
- Prolonged use of topical corticosteroids (TCS)?

Treatment
Priority should be given to topical calcineurin inhibitors (TCI)
Topical antifungals are often ineffective or poorly tolerated
Relevance of systemic antifungals

- Itraconazole (initially prescribed by the hospital)
- No consensus on dosage but she suggests using 2 tablets (tb) of 100 mg per day for 1 month and, if the patient responds to treatment, continue with 2 tb 2x/week for 2-6 months as maintenance treatment

2nd patient

48-year-old female
AD since childhood
Topical corticosteroids on the face if flare-up
Frequent pruritus on the face
Ophthalmological follow-up for blepharitis

AD + rosacea (Head & Neck Dermatitis)

Contributory factors

- Prolonged use of TCS
- Demodex?

Appropriate Management (AM)

- TCS elimination
- Relevance of TCI
- Where appropriate, topical metronidazole (not always tolerated), topical or systemic ivermectin or cyclins

3rd patient

AD and perioral dermatitis
Contributory factors

- TCS + greasy emollient + mask

4th patient

27-year-old female
Severe AD since childhood
2009-2019 Treatment of flare-ups with TCS, Desonide on the face
Burning and tingling caused by TCI: use of TCS resumed by the patient
2019 acne induced by corticosteroids, discontinuation of TCS
>> Uncontrollable, painful, pruritic, inflammatory AD, involving cracks and scabs

Topical Corticosteroid Withdrawal / Red Skin Syndrome / Topical Steroid Addiction

Treatment

- TCS resumed and gradually discontinued
- TCI or systemic

5th patient

AD since childhood
Acute worsening in the past year

AD and contact eczema

Patch test
Allergic contact dermatitis / irritant dermatitis / AD flare-ups induced by airborne allergens / UV: photoaggravated, photosensitisation

6th patient
AD predominantly affecting the head and neck
Proactive treatment with TCI 2x/week
Flushing with burning sensation when drinking alcohol

Flushing induced by TCI + OH

7% of patients treated with TCI
Inform patients
Avoid OH 2 hours before and after TCI
Flushing induced by dupilumab + OH

7th patient
72-year-old female
Severe AD since childhood
Treatment failure
Introduction of dupilumab
Severe facial erythema with “patchy” appearance, burning, pain

Facial erythema induced by dupilumab

Average time to onset: 11 weeks
AD with head and neck involvement or de novo
Only assumptions for aetiology
May cause treatment to be discontinued (8%)

Non-codified AM
- TCS
- TCI
- Antifungals: itraconazole?
- Relay with JAKi?

8th patient
Severe AD
Introduction of JAKi
Fluctuating papular rash on the face and sometimes trunk

Papular rash when taking JAKi

Acneiform eruption
No comedones (or rarely)
Particularly with selective JAK1 inhibitors, dose-response
Physiopathology unclear
Non-codified management

9th patient

Severe AD since childhood
Prescribed local treatment (TCS and emollients)
Unusual, painful rash for the past 72 hours
No improvement after taking TCS

Eczema herpeticum

Appropriate Management

- Introduction of valaciclovir without delay
- Short suspension (discussed) of local anti-inflammatories (a few days)

News item 1: News about neurofibromatosis - Pierre Wolkenstein

Neurofibromatosis 1 (NF1)

- Incidence 1/3,000 i.e. 20,000 patients in France
- Life expectancy 67 years (-10 years)
- 2005 emergence of phenotypic analysis

o Patients with **cutaneous neurofibromas (NF)**
o Patients with **subcutaneous NF** and huge tumour masses with, in **20%** of cases, transformation into **malignant nerve sheath tumours** ->

- **Cutaneous NF** most often occurs **after puberty**, while **subcutaneous NF occurs in childhood**

o Identification by clinical examination of a **high-risk phenotype** which should be closely monitored

- 2007 SPRED1 mutation **Legius Syndrome**: families with subjects with café-au-lait spots but without cutaneous NF, macrocephaly, delayed development
- 2008-2010 **Face transplants** in patients with a huge plexiform facial tumour

o Discontinued due to chronic rejection

- 2011 **modifier genes**

o An SNP located in non-coding **RNA ANRIL**, very close to melanoma susceptibility genes CDKN2A and CDKN2B, correlated with a large number of **plexiform tumours** in NF1 patients (J Natl Cancer Inst 2011)

- 2011 identification of the **premature mortality** of NF1 patients aged 20 to 40 due to cancer, mostly malignant nerve sheath tumours (Orphanet Journal of Rare Diseases 2011)
- 2013 prognosis of **malignant nerve sheath tumours** confirmed, **95% death after 3-4 years** (Orphanet Journal of Rare Diseases 2013)
- PET scan identification of tumours in the process of transformation (PLOS 2013)

o All patients with **subcutaneous and internal neurofibromas** undergo a **PET-MRI or PET scan**

- All patients with an **SUV** (standardized uptake value), i.e. hypermetabolism, are **biopsied**

o **Diagnosis of dysplastic neurofibromas prior to malignant transformation**
o Hope of reducing the incidence of malignant nerve sheath tumours and **better prognosis**

- 2013 classification of RASopathies molecular pathway diseases
- 2016 publication of the first selumetinib **MEK inhibitor therapeutic trial** for children with inoperable plexiform NF (NEJM 2016)

o In 60% of cases included in the trial, 40% reduction in the volume of tumours with many side effects

- 2017 identification of an **increased risk of frequent and aggressive breast cancer** which is quite often bilateral (British Journal of Cancer 2017)

o Risk increased by 20-30%
o Recommendation of a **breast MRI at the age of 30** and debate on systematic mammography, risk increased by 20-30%

- Emergence of ovarian cancer
- 2019 first consistent **animal model** by mutating the stem cells of boundary caps in mice (Nf1 Knock-Out; Cancer Discov 2019)

o A mouse with cutaneous, internal neurofibromas and developing nerve sheath tumours

- 2019 **swine model**

o **Pharmacological** characteristics identical to humans, making it possible to test drugs

- 2020 2nd selumetinib trial for children with inoperable plexiform NF, 30% reduction in the tumour mass

o Slow effect: becomes apparent after 8 months, better effect after 16 months – will **not change the indication for surgery**

- 2021 **selumetinib marketing** authorisation in Europe and the USA
- 2020 Change of **diagnosis criteria** including (Genetics in Medicine 2021)

o Introduction of the Nf1 mutation

o Choroidal hamartomas: can replace molecular biology in young children (under 8 years of age) to differentiate from the Legius Syndrome

- *Annales de dermatologie et de vénéréologie* 2022 **French Version of criteria**
- Criteria for which no consensus was reached: nevus anemicus, juvenile xanthogranuloma, unidentified bright objects detected by brain MRI, often correlated with learning disabilities
- Cutaneous NF destruction techniques (European Journal of Medical Genetics 2022)

o CO2 laser BUT

o 30% stop laser: insufficient results

o Scarring problems

- Therapeutic trials of **anti-MEK gels** moderately effective in phase 1
- **Minimal pERK** activity when the NF is fully formed (Wolkenstein 2023)
- **Preventive treatment** when **NF under development** still have a pERK activity (Wolkenstein 2023)

o Anti-MEK topical effect ++

- In the future **preventive strategy**

o Treatment from the age of one to 18?

- Genome scan in 1,333 cases of NF1: 40 genes of interest (Wolkenstein 2023)

o GAS1 growth arrest protein, SPRED2 etc. new treatment possibilities: **target genes**

What's new in interventional dermatology?

Dr Jean-Michel Mazer

Dr Jean-Michel Mazer presented articles published in 2022 on the treatment of skin cancer, dermatological surgery, surgical oncology and laser.

Hutchinson sign biopsy in the diagnosis of subungual melanoma (SUM)
Hutchinson Sign: Biopsy May Assist in Diagnosis of Subungual Melanoma in Situ. Oh et al. Dermatol Surg 2022; 48: 28-31.

- Retrospective study of 12 patients who had subungual melanoma in situ, confirmed by histology + punch biopsy of the Hutchinson Sign (HS)
- All cases showed histological abnormalities indicative of the diagnosis of SUM in situ
- A HS biopsy is a simple way to confirm a subungual melanoma diagnosis
- It **should not however replace a nail matrix biopsy** for fear of underestimation (Adam I Rubin, Bertrand Richert , Eckart Haneke. Commentary on Hutchinson Sign: Biopsy May Assist in Diagnosis of Subungual Melanoma in Situ. Dermatol Surg 2022; 48: 32-33.)

Effectiveness of tacking sutures and triamcinolone (TAC) injection in preventing pincushioning in bilobed transposition flaps
Evaluation of Intraoperative Triamcinolone Injection or Primary Lobe Tacking (Pexing) Sutures for Preventing Pincushioning in Bilobed Transposition Flaps. Boylan et al. Dermatol Surg 2022; 48:191-194.

- Subcutaneous tacking suture involving the muscles
- Retrospective study of 342 patients
o 37 tacking sutures
o 42 perop injections of TAC
o 263 no other procedure
- Incidence of pincushioning on D35
o Group with no other procedure 30.8%
o Triamcinolone group 23.8% (p=0.358)
o Tacking suture group 5.5% significant (p=0.001) +++

Adjuvant radiotherapy in the event of completely excised squamous cell carcinoma (SCC): no significant difference in the number of deaths
Adjuvant radiotherapy may not significantly change outcomes in high-risk cutaneous squamous cell carcinomas with clear surgical margins: A systematic review and meta-analysis. J Am Acad Dermatol 2022; 86: 1246-1257.

- Relevance of adjuvant radiotherapy in the event of completely excised squamous cell carcinoma (SCC)
- Meta-analysis of 33 studies with 3,867 high-risk SCC
- No significant difference in the unfavourable outcome between the surgery group VS surgery + radiotherapy
- Deaths were more frequent in the radiotherapy group BUT these were retrospective studies with no sufficient data on patients and the difference was not significant

Silver-containing dressings may set off airport metal detectors
Use Caution When Travelling and Wearing Medical Dressings: Foam Dressings Containing Silver May Set Off Metal Detectors. Kesaria et al. Dermatol Surg 2022 Feb 1;48(2):256-257

- A patient having undergone MOHS surgery whose skin graft donor site was covered with a 30 cm2 hydrocellular foam dressing containing silver set off the alarm at the airport's security control
- Mepilex Ag 10x10 cm contains 1.2 g/cm2 Ag sulphate, i.e. 12g of Ag sulphate
- The airport’s metal detector can detect silver volumes as low as 0.6 g
- Inform patients J

Lasers

Treatment of neoangiogenesis induced by pulsed dye laser treatment
- **Pulsed dye laser** is the reference laser for **port-wine stains**, in particular in children and infants
- Despite a good initial response, **secondary neoangiogenesis** may cause subsequent relapse

Study to assess the depth and diameter of vasculature for better treatment results
Analysis of port-wine birthmark vascular characteristics by location: Utility of optical coherence tomography mapping. Wang et al. Lasers Surg Med 2022; 54: 98-104.

Neoangiogenesis inhibitors (ANTI-VEGF) to prevent relapse

- Topical rapamycin
- Topical metformin (Topical Metformin suppresses angiogenesis pathways induced by pulsed dye laser irradiation in animal models. Wong et al. Exp Dermatol 2022; 31: 393-397)
- Tivozanib (vascular endothelial growth factor receptor tyrosine kinase inhibitor (VEGFR-TKI)) (Initial Research on the Effect and Mechanism of Tivozanib on Pulsed Dye Laser Induced Angiogenesis. Wand et al. Lasers Surg Med 2022; 54: 1157-1166).

Videos reports by

Dr. Joël CLAVEAU

Dermatologist, Quebec

Interactive skin cancer session

How to optimise and manage healing in dermatological surgery

Session on the dermatology of solid organ transplant recipients

Dermatological emergencies

Reports written by

Dr. Nicolas KLUGER

Dermatologist, Finland

Burnout in French dermatologists

According to PO12-259. Burnout in French dermatologists: a prospective nationwide study.
N. Jouan, C. Taieb, B. Halioua

A study conducted under the guidance of FFFCEDV assessed the prevalence of burnout in French dermatologists, regardless of the type of practice, in order to evaluate the risk and protective factors. An online questionnaire was administered that included the Maslach Burnout Inventory (MBI), professional characteristics (thesis year, methods of practice, daily working hours), and personal data. A total of 577 responses were analysed. Ninety-one percent of the respondents were women, 78% had a private practice, 72% reported working more than 35 hours per week, and 63% said they had met with aggressive patients at least once. Sixty-seven percent reported over 20 years of practice. Almost 48% of French dermatologists were experiencing burnout: 29% mild burnout, 15% moderate burnout, and 3% severe burnout. Burnout affected both genders with no significant difference (40.3% of men and 49.1% of women). The type of practice (self-employment or employment) and the number of days worked had no impact; however, the factors that seemed associated with burnout included the

number of hours worked (>35 hours), the lack of a secretary, dissatisfaction with compensation, dealing with aggressive patients, and the absence of physical exercise.

The aggression of dermatology patients in France

PO12-263. The aggression of dermatology patients: a national study
B. Halioua, C. Taieb, N. Jouan*

In parallel with the previous study on burnout (PO12-259. Burnout in French dermatologists: a prospective nationwide study. N. Jouan, C. Taieb, B. Halioua), the same dermatologists shared the results of a survey on physical and verbal aggression towards French dermatologists. Of the 650 responding dermatologists (84% women), over 81% reported having dealt with demanding, aggressive or rude patients. The majority were women as well as dermatologists in business for less than 30 years. There was no connection to the number of days of consultations, weekly working time, the region, or the way of working. Less than 3% filed a complaint or police report, while another 3% hesitated to do so. Sixteen percent benefited from support (colleagues, psychiatrist, medical council). Such events have effects such as prompting dermatologists to consider retiring early; above all, they have consequences for families. There is also physical and verbal violence against French dermatologists. Its prevalence is underestimated despite it having major consequences for professional and family life.

The cutaneous manifestations of VEXAS syndrome

According to CO06-052. Clinical and histological characteristics of the cutaneous manifestations of VEXAS syndrome: a centralised, retrospective study of 59 cases
Clinical and histological characteristics of the cutaneous manifestations of VEXAS syndrome: a centralised, retrospective study of 59 cases
E. Zakine, F. Rodrigues, L. Papageorgiou, S. Georgin-Lavialle, A. Mékinian, B. Terrier, O. Kosmider, P. Hirsch, M. Jachiet, S. Audia, S. Ardois, L. Adélaïde, A. Bigot, P. Duriez, J.-F. Emile, E. Lazaro, D. Fayard, J. Galland, M. Hié, S. Humbert, A. Jean, M. Kostine, V. Lacombe, G. Le Guenno, H. Lobbes, N. Magy-Bertrand, P. Marianetti-Guingel, A. Mathian, R. Outh, C. Saillard, M. Samson, G. Vial, J.-D. Bouaziz, P. Moguelet, F. Chasset*

VEXAS syndrome (vacuoles, E1 enzyme, X-linked, autoinflammatory, somatic) is a late-onset auto-inflammatory disease caused by myeloid-restricted somatic mutations in the ubiquitin-like modifier activating enzyme 1 (UBA1) gene located on the X chromosome. It is associated with cutaneous and extracutaneous manifestations. The patients, who are often men, present with an altered general condition, fever, systemic manifestations such as chondritis, uveitis and thrombotic events, as well as biological findings such as cytopenia, myelodysplasia and less often, monoclonal gammopathy.
The skin is the most affected organ in 85-90% of cases and cutaneous involvement is often inaugural with features of neutrophilic dermatosis, livedo or cutaneous vasculitis.
This retrospective study aimed to review the clinical and histological aspects of VEXAS syndrome. It manifested mainly as often numerous and sometimes arcuate maculopapules and nodules, affecting the torso and face, that varied in size and colour; less often, it took the form of pustules, bullae or livedo. Histological analysis of the papulo-nodules and maculopapular exanthema showed a

perivascular and interstitial dermal infiltrate of lymphocytes, myeloid precursors and dysplastic neutrophils, sometimes with capillary thrombosis and leukocytoclasia but without true vasculitis. Overall, the spectrum of cutaneous lesions in VEXAS is characterised by great heterogeneity. VEXAS syndrome should be considered in patients ≥ 60 years presenting with general symptoms, systemic inflammation and cutaneous infiltrates showing neutrophilic dermatosis with immature myeloid cells.

Perceived stigma among hidradenitis suppurativa patients

PO13-267: Prevalence of perceived stigma and coping strategies in patients with Verneuil's disease in France
Bruno Halioua (Paris)

FO26 - Stigma and coping: understanding to take action.
Pierre Vabres (Dijon)

The prevalence of perceived stigma and coping strategies in adult patients with hidradenitis suppurativa (HS) is high. In a study focusing on 544 patients (78% women, average age 37), 89.3% reported feelings of embarrassment or self-consciousness associated with HS. Factors predicting perceived stigma were a younger age; moderate or severe disease; and the presence of itching, pain or burning. Patients perceiving stigma were less satisfied with their medical care. 96.5% of patients reported an aesthetic inconvenience and >94% expressed dissatisfaction with their appearance. Patients with VD often report coping strategies: they do not take part in family or work events, do not go on holiday or do not participate in leisure activities; they avoid photographs and selfies and choose clothes suited to their disease.

Moreover, during the FO26 - Stigma and coping: understanding to take action forum, Bruno Halioua explained the major impact of discharge and odour on the patient's sexuality. The consequences of these secretions include shame and isolation due to fear of a negative reaction, concealment of lesions, and the use of fragrances and dressings. HS has a stronger impact on sexuality than other chronic dermatoses (prurigo, bullous diseases, psoriasis, etc.).
Dermatologists should consider these psychological disturbances that are often difficult to take into account because patients do not always dare to express them. The development of appropriate patient education tools would help shed light on their psychological distress.

Sequential terbinafine – itraconazole therapy for microsporic tinea

PO09-169: Managing microsporic tinea after the age of griseofulvin: a true challenge
Nour el Imene Ouni (Monastir)

The disappearance of griseofulvin from the market is problematic in terms of the management of tinea infections in children. The only current alternatives available are terbinafine and itraconazole.
A Tunisian prospective study assessed the efficacy of oral terbinafine in the treatment of microsporic tinea (*Microsporum canis*) at "high" doses (> 6 mg/kg/day) with monitoring after four weeks. If improvement was observed, treatment was continued for another two weeks. If there was no response, sequential therapy with itraconazole was administered with doses based on body weight (> 40 kg: 200 mg/day; 20-40 kg: 100 mg/day; 10-19 kg: 50 mg/day). The sequential regimen (a seven-day cycle and then stoppage for three weeks, with a maximum of five courses) was proposed due to

issues involving the cost of itraconazole for families. A liver panel test was performed before terbinafine and before each course of itraconazole. Children weighing under 10 kg were excluded. Thirteen children were treated (average age 5.7 years, average dose of terbinafine 7.6 mg/kg). Only two children were cured with terbinafine, while four showed partial improvement. With itraconazole, the number of courses ranged from one to five. Tolerance was excellent. Sequential therapy with terbinafine – itraconazole could be relevant for the management of microscopic tinea.

Extensive dermatophytosis resistant to topical treatments: think of *Trichophyton indotineae*!

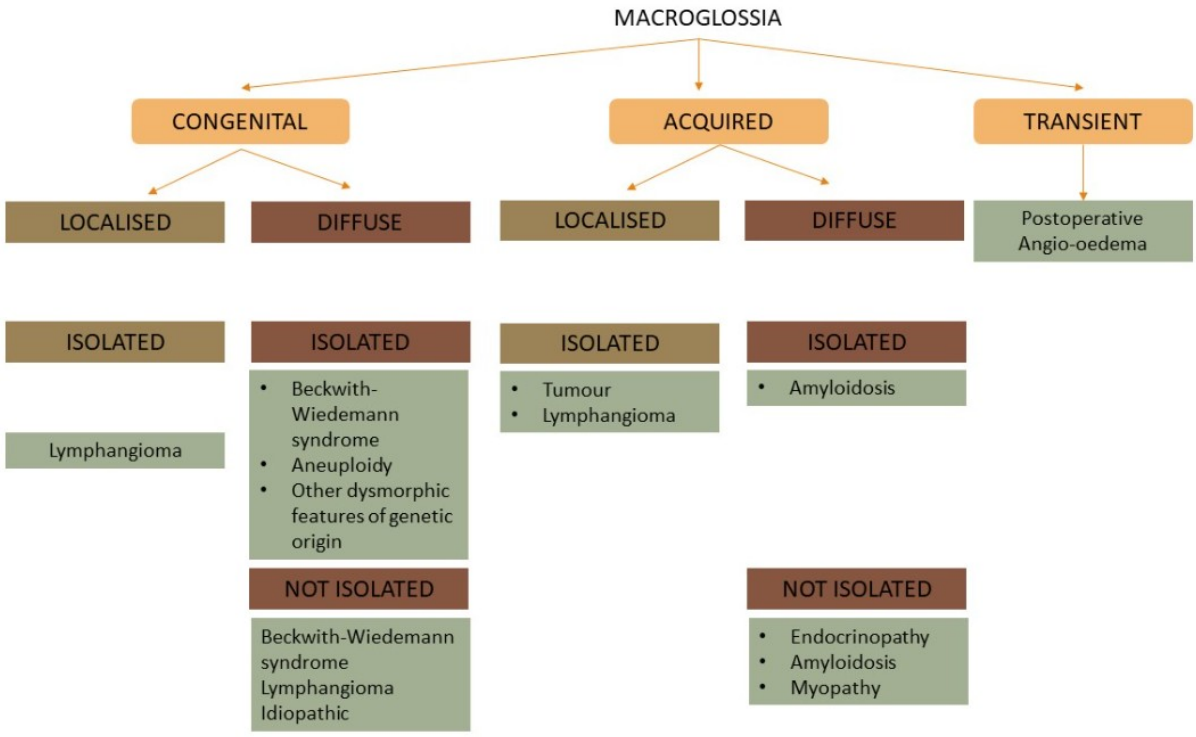
According to PO06-131: Extensive cutaneous dermatophytosis caused by *Trichophyton indotineae*: also a possibility in children.
Sabine Karaa (Paris)

Trichophyton indotineae is an emerging dermatophyte in developing countries whose spread is now global and whose unique feature is terbinafine resistance due to mutations in the squalene epoxidase enzyme. It should be considered in adults and children having extensive isolated cutaneous infections resistant to topical treatments (topical azoles) and also in contexts of travel to at-risk areas (Central/Southern Asia). *Trichophyton indotineae* is a species of the Trichophyton mentagrophytes complex that can only be identified via molecular biology. Treatment with itraconazole lasts six weeks; family members should be tested and long-term follow-up should also be proposed.

Diagnosing macroglossia

PO16-293: Aetiological diagnosis of macroglossia: literature review and proposal of a diagnostic algorithm
E Dietrich (Angers)

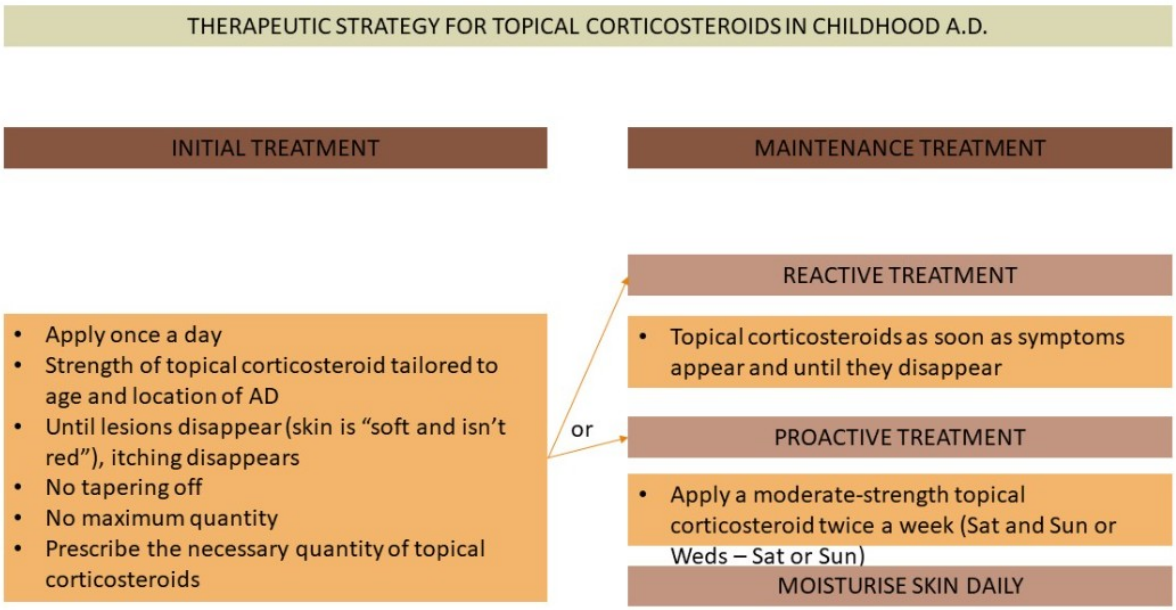
Teams from Angers and Tours proposed a diagnostic algorithm for macroglossia based on an extensive literature review (1096 abstracts reviewed). The algorithm is reproduced in Figure 1. Amyloidosis, endocrinopathies, myopathies and tumours are the most frequent acquired causes, whereas for congenital forms, the causes include lymphatic anomalies, aneuploidy (Down syndrome, etc.) and Beckwith-Wiedemann syndrome (or exomphalos-macroglossia-gigantism syndrome, OMIM 130650).



General principles for the topical treatment of atopic dermatitis

According to the communication of Hélène Aubert FMC 63. Atopic dermatitis in childhood: 10 clinical situations

Some reminders on topical treatments for atopic dermatitis (AD) in childhood, especially topical corticosteroids, are always welcome. As for formulations, it should be noted that ointments are suitable for lichenified, hyperkeratotic and dry areas. They should be avoided in skin folds. Creams are suitable for oozing areas, skin folds and large skin surfaces, lotions for areas with hair and for skin folds, and gels for the scalp. Preparations can be proposed for very large surfaces. Age, the location of the lesions, the size of the area to be treated and the oozing nature of the lesions are criteria that affect the chosen class of topical corticosteroids. The strategy for using topical corticosteroids is reviewed in Figure 2.
Tacrolimus (0.03% in children over the age of two and 0.1% from the age of 16) is mainly suitable for atopic dermatitis of the face in adolescents and young adults. It is poorly tolerated in children and should be proposed as maintenance therapy following treatment with a topical corticosteroid. It is not recommended for severe flare-ups of AD, nummular forms, or forms affecting the extremities. However, it is now established that tacrolimus does not increase the long-term risk of cancer in children with AD.
The new treatments that may emerge on the market in the near future include crisaborole (a phosphodiesterase-4 inhibitor), which already has MA in North America; delgocitinib (a pan-JAK and TYK2 inhibitor), available in Japan; ruxolitinib (a JAK 1 and 2 inhibitor), which has MA for vitiligo; and lastly, roflumilast 0.3% (a phosphodiesterase-4 inhibitor), which has an affinity 25 to 300 times higher than crisaborole).



Traditional tattoos are disappearing in Morocco

PO11-251: Traditional tattoos in Morocco
S Bouabdella (Oujda)

Traditional tattoos in Morocco can still be found among elderly women; however, this cultural practice is disappearing, as younger generations are turning to "modern" tattoos. This prospective single-centre study carried out in Oujda aimed to better characterise this practice. In total, 119 patients (92% women, average age 70) were included. In order of frequency, the tattoos were located primarily on the forehead (74%), wrist (67%), and chin (56%). Age at the time of the 1st tattoo was 15 years, with around two tattoos per woman. None were done by a professional tattoo artist. Most of the time, they were done by a friend or family member. In 96% of cases, they were drawn with a needle and kohl, sometimes with the addition of green wheat juice (34%). In order of frequency, the reasons included beauty (29%), belonging (19%), protection (14%), therapy (12%) and fertility (7.5%). The few men with tattoos had gotten them for therapeutic reasons (headaches, osteoarthritis). Note that 83% regretted their tattoos, mainly due to religion.

Reports written by

Dr. Joséfina MARCO BONNET

Dermatologist, France

How to optimise and manage scars in dermatological surgery?

By Dr J.Labarthe, Dr J. Lulin, Pr N. Litaiem, Dr JM Amici, Dr O. Cogrel

1- During surgery

After excising the lesion, it is important to analyse tissue movement by asking 3 questions:
- What should not be deformed?
- Where does the laxity come from?
- Where are sutures needed?

The following is important:
- Never complicate matters when a simple option is available
- Place the scar along the lines of least skin tension.
- Respect the sub-aesthetic units of the face
- Respect facial symmetry: principle of horizontal suture placement. Forget the approximation of edges at the centre of the face.
- Retain alignment with eyebrows and lips
- Do not cross the edges of the bones
- Repeat in the same sub-aesthetic unit
- Do not deform the orifices
- Do not make the flaps too small
- Importance of everting deep stitches. Most of the stitches should be deep with only a few superficially.
- Know excellent directed healing zones
- Immobilise the scar with strips such as silicone bandages.
- Apply compression bandages.
- Know the scarring risks of staples and warn the patient (dyschromia, retraction, necrosis).

2- Manage immediate complications that may impact the final quality of the healing

Very early < 5 days:
- Bleeding: exercise caution with patients who have already had episodes of bleeding after skin surgery. Bleeding is normally due to insufficient haemostasis, the rise in adrenalin vasoconstriction, a hypertensive flare-up, clotting disorders, haematological disorders, taking platelet antiaggregant drugs, VKA and DOAs.

Medium size arterioles should be sutured with an X stitch and not electrocoagulated.
Apply compression bandages and fasten them to avoid tampering.
- Bruising of the eyelids.

This is linked to the spread of bleeding on around D3 to a lower, more lax area in patients on VKA.
There is no impact on healing nor tissue suffering. Patients should just be warned as it is very obvious and therefore concerning.
- Haematoma.

If liquid, they should be punctured.
If there are blisters, they should be emptied, haemostasis resumed and a suitable bandage reapplied.
There are risks of necrosis and superinfection.
- Loosening of suture (unsuitable thread, skin fragility, loosening of knots, too much tension in the suture). The suture will need to be redone.
- Pain. This may reveal haematoma or infection.

Secondary > 5 days.
- Inflammation, start of infection.

To avoid these: * shave hair to allow for better cleaning
* at orifices, particularly if skin is seborrhoeic, apply bactroban ointment to the nostrils and mupiderm

ointment to the ears. Consider prescribing zelitrex if there is a history of herpes on the lip or lip surgery.

- Disunity: review the scar if it impacts healing.
- Necrosis if the pedicle is insufficient, if the transposition is an open angle, if there is mechanical tension, staple.

3- Focus on damaged skin

- Post-inflammatory hyperpigmentation (PIH)

By way of prevention, early photoprotection with mineral screen should be considered. During surgery, the skin should be handled delicately (Gillis crochet) with a deep plane, avoiding tight sutures and superficial stitches with non-absorbable thread. Then address the inflammation, applying bandages, hydrocolloids, treating infection and using dermocorticoid drugs.
Tranexamic acid injections (100mg/ml twice, one month apart) have been tested on scars with encouraging results and few side effects.

- Keloid scars.

It is important to enquire as to a history of keloid scars and recommend against any unnecessary surgery if it is a risk.
By way of prevention, it is important to control tension around the wound.

4- Treatment of keloid scars

- First line

Late injection of corticosteroids into the lesion (40mg/1ml on D15 with xylocain adrenalin solution, retro-tracing and in array until blanching. Then apply a wound bandage (silicone or hydrocolloid). Repeat every 6 weeks.
This relieves itching and pain. Follow-up from 18 to 24 months.
Always add a compression: compressive clothes, face mask.

- Second line

Triple therapy should be applied: excision of the keloid scar retaining the wound cover + IL corticosteroids + compression. We consider scars to be stable after 18 months.

- The rest is on a case-by-case basis.

- Imiquimod with fractionated laser
- Bleomycin: no MA
- 5% FU
- Radiotherapy and brachytherapy
- Phenolisation

5- Early hypertrophic scarring (EHS) or make into a ball, pincushioning

EHS above all involves the nose (tip, wing, canthi) and then the flaps. They onset early at D15. They are asymptomatic but sensitive to pressure. Spontaneous regression is possible but still partial. To avoid them, it is important to apply a fixing stitch deep down. To treat them, IL corticosteroids can be injected from D15 or D45.

6- Botulin toxin and scarring

The toxin has been found to improve scars in multiple studies. It acts on the fibroblasts and reduces muscle mobilisation. It therefore needs to be injected deep into the muscle and superficially into the dermis.

7- Addressing scars

Scar management is an integral part of surgery. They should be reviewed at 1, 3 and 6 months.

- Managing inflammation. It is important to oppose the forces of tension with wound effect dressings, strips or botulin toxin.
- Treat inflammation early with dermocorticoid drugs (over a short period of time), late IL corticosteroid injections and vascular laser.
- Massage: drainage, action on fibrosis. Repeat twice a day after 15 days, for 3 months, with a non-irritating topical treatment.
- Superficial atrophy/hypertrophy: fractionated lasers are better than ablative lasers or dermabrasion. A combination with hyaluronic acid can also be offered.
- Hypertrophic scars: pulsed dye laser 585nm/595nm or IPL improve erythema, texture and reduce itching and pain. Stitch ablation can be performed. After lasers, dermocorticoid drugs can be applied twice a day for 7 days.

Myths and facts about tattoos

By Dr Nicolas

Between 18 and 20% of adults have at least one tattoo. More women have tattoos than men.
The reasons vary depending on the continent. In Europe and the USA, it is most often to commemorate an event, whilst in Russia it is seen as beautifying the body and in Asian countries, it is done for love.

In France, tattoo artists must declare their business to their ARS (regional health authority). They must complete mandatory training on health and hygiene rules and provide the ARS with certification of this.
They undertake to inform their customers of the risks involved and precautions to be taken, to respect general rules and good practices of health and hygiene and to sign an agreement for waste disposal (DASRI).

In tattoo consults, 75% of patients seen by Dr Kluger describe problems with tattoos, 7% have problems with permanent make-up and 20% simply want information before getting a tattoo.

In the event of a reaction to a tattoo, it is important to:

- Question and examine the patient
- Take a 3-4mm punch biopsy
- Avoid over-diagnosis of allergy to ink
- Contact that tattoo artist
- Make a declaration to the ANSM (French Agency for the Safety of Medicines and Health Products)

The complications seen are:

- Allergies to a colour.

It is very difficult to find out the exact composition of inks and labels are not reliable. An ink contains pigments, binders, solvents and additives. Attempts have been made at implementing regulations but they are difficult to standardise across the whole of the EU.
Most of the time, it is the colour red that is the culprit (70% of cases). This gives rise to eczema-type reactions that appear in 43% of cases quickly (in less than a month) but reactions can be seen for up to a year after the tattoo. Allergological tests are pointless because the allergen is not in the ink,

rather the problem lies in the haptenisation of the tattooed skin, which is responsible for the reaction. Small test tattoos serve no purpose.
The colour and its derivatives should therefore be contraindicated and cold colours recommended.

- Early post-inflammatory oedema.

It onsets rapidly. It is temporary and benign.

- Contact eczema

The erythema overruns the tattoo. It is important to look for topical products and disinfectants to apply to patients.

- Tattoo blowout.

This corresponds to the spread of ink in fat, it is how tattoos end up blurring deep down. This complication is permanent. It affects women most often in the inner arms and thighs. Laser therapy can be offered to patients.

- Superficial thrombophlebitis.

This is a rare complication whose risk factors are spending a long time lying down and the presence of varicose veins.

- Traumas linked to the needle.

This is an open wound or traumatic excoriations over the whole of the tattoo, which onsets immediately after performance, with bleeding or unusual secretions followed by extensive scabbing and prolonged scarring (D1 to D31).
Recommend oily healing creams and antibiotics if there is any suspicion of superinfection.

- Infections.
- Atypical mycobacteria.

This complication occurs quickly or more slowly depending on the quantity of inoculum. The most frequent are on grey, as tattoo artists mix black with distilled or tap water to make the colour. Consider calling the tattoo artist to look for other cases.

- Nodules on old tattoos.

Always assess sarcoidosis.
There are 3 possible scenarios: it is an isolated granuloma without sarcoidosis, it is a sarcoidosis on the tattoo or it is a reaction of a known sarcoidosis.
Granulomas and sarcoidosis on tattoos may also be caused by targeted cancer treatments.
Another entity requires understanding: the association of uveitis and sarcoidosis granulomas with or without systemic sarcoidosis. These forms of uveitis are often resistant to treatment and recovery has been described after tattoo removal.
Nodules can be treated with dermocorticoids, tacrolimus, hydroquinidine, cyclines or methotrexate or alternatively we can simply wait as some disappear spontaneously.

- Eruptive keratoacanthomas do not appear to be coincidental.

They appear on average-aged tattoos, which are red, exposed to the sun, inflammatory or traumatised.

Dermatological disease and tattoos.
Some of our patients with dermatosis want to have a tattoo. We can offer them some advice:

- The disease should be stable, not evolving and quiescent.
- Warn patients that diseases can occur with skin damage, giving rise to lifelong lesions on the tattoo (lupus, sarcoidosis)
- Consider immune suppressant drugs. There are more complications with this type of treatment.
- Avoid tattoos on certain areas: skin lesions, sclerotic skin and paralysed limbs.
- No particular precautions are needed with colchicine, disulone or hydroxychloroquine.

If patients are on immune suppressant drugs:

- No tattooing during the initial treatment phase
- Wait for treatment to be at minimum or maintenance dose

- Oral corticotherapy impacts healing (>10mg/day)
- Tattoos are considered as surgery with a low risk of infection
- The delay in resuming biotherapy after getting a tattoo depends on the biotherapy and that tattoo must have healed with no sign of infection or inflammation.

Myths:
- Cancer.

To date, any association of tattoos with skin cancer is purely coincidental. There is no link to myeloma and lymphoma. For the rest, no data is available. Notably on nanoparticles and bowel cancer.
- Quid on nickel allergies.

There is nickel in the ink but there is no CI to date. Discuss the risks of an eczema flare-up with the patient.
- I need to perform an MRI. There is no CI.

Black and brown pigments contain iron oxide, particularly old tattoos. During an MRI, tingling, pain or burning sensations may occur. This can disturb the images if regarding the area of interest.
- Glands may turn black: true
- Tattoos and transfusions.

No increased risk of transmittable infection after tattooing, piercing or acupuncture if carried out in a country where conditions are regulated.
- Vaccinations and pricks can be applied at a tattoo site: YES
- Tattoos and pregnancy: No. No data available to date
- Tattoos and breastfeeding: No

Mothers are recommended to wait at least 9 to 12 months after birth, so that the baby is no longer dependent on breast milk alone.

Congenital heart disease and tattoos
- Ask for a cardiologist's opinion
- Discuss antibiotic prophylaxis even if the risk of endocarditis is small.
- Exercise caution in respect of general symptoms during the weeks that follow a tattoo, even if it appears unharmed.

Clotting disorders:
- No case recorded of cataclysmic haemorrhage in literature. Eccymosis is rare.
- For Von Willebrand, haemophilia and thrombocytopoenia, seek specialist advice.
- For VKA, antiaggregants, NACO: first light session and maintain treatment.

Organising the ecological transition in dermatology the the SFD (French Society of Dermatology) envi'derm group

Dr M. Boileau, Dr C. Laumaillé Cadiou, Dr C. Derancourt, Dr C. Pannequin and Dr M. Bataille

The healthcare industry releases 46 million tonnes of CO2, accounting for 8% of the national total. The first question to be asked is how the healthcare industry emissions break down, so as to know what levers to use.
Electricity consumption accounts for 3% of emissions, heating/climate control and medical gas account for 10% of emissions. 87% are represented by the purchase of medicines, medical devices (MDs), the transport of employees and patients.
How to decarbonise the healthcare system with reducing access to care and quality of care. The following is important:

- Prerequisites: train carers and patients, assess the carbon footprint of our practices and analyse the life cycles of medicines and MDs.
- Action plans: propose concrete measures for doctors and patients, put pressure on the industry and institutions and measure the impact of measures taken on the carbon footprint.

We can act on our prescriptions, our digital consumption and waste, to start.

Endocrine disruptors (EDs) and chemical contaminants in cosmetology

By Dr C. Laumaillé Cadiou

EDs have been known for more than 60 years now. In 2021, there are 906 substances classified as EDs. They may be natural or synthetic chemical substances, foreign to the body and which may interfere with the function of the endocrine system and thus have negative effects on this organism or its descendants. We find them in cosmetics, pesticides, medicines, synthesis hormones, phytohormones (soy), heavy metals, plastics, flame-retardants and anti-adhesives. Certain pathologies are associated with EDs: congenital malformations, reproductive disorders, hormone-dependent cancers (breast, prostate, testicular, thyroid, ovarian?), metabolic disorders (obesity and diabetes), neurodevelopmental disorders (reduced IQ, autistic spectrum disorder, attention disorder, hyperactivity) and neurodegenerative disorders (Parkinson's, Alzheimer's and SLA).
The problem is that we do not know the toxic dose, if the effect is proportional to dose, if there is a cocktail effect, a deferred effect, epigenetic or transgenerational effect like Diethylstilbestrol. It varies according to the different periods of life. In actual fact, periods of vulnerability are pre-conception, pregnancy, the first 1000 days and adolescence.

In cosmetics, EDs are:
- Preservatives (parabens, glycol ether, including phenoxyethanol and triclosan)
- Silicones
- Plastics (phthalates, bisphenol A and others)
- Sunscreens
- Not to mention the container that interacts with the contents.

Nanoparticles enable a larger exchange surface and are more reactive, but there is no specific MA for a nanoparticle and the legal obligation for labelling is not very well respected. The risk to health and the environment is not very well-known.
Toxicity is carried out via deep penetration in the organism, the production of cytotoxic free radicals, interaction with DNA and the increased passage of other molecules (the "trojan horse" effect).

The titanium dioxide used in cosmetics like sunscreens, white dye and texture. It is classified as carcinogenic by inhalation in the form of powder.

Plastics.
These are essentially disposable packaging (around 1500 tonnes/year of cosmetic containers end up in microplastics in the water environment. There is an ever-increasing carbon footprint during their production. In addition, recyclable plastic does not necessarily mean recycled plastic.
Micro and nanoplastics are due to the deterioration of macroplastics. There are vectors for chemical pollutants and vector-borne diseases.

Sunscreens are OTC cosmetics, without MA, without any clear evidence of efficacy in terms of their prevention of skin cancer, with nano risks, the risks of ED, contained in plastic, whose consumption is growing and that have environmental toxicity.

The dermatologist's prescription and EDs

by Dr C. Derancourt

There are 3 vulnerability periods for which we need to be more attentive: pre-conception - pregnancy, the first 1000 days and adolescence. We have a duty to general information in respect of EDs.
It is therefore important to avoid prescribing cosmetics for healthy skin during these periods.
It is important to assess the risks and benefits, when prescribing them. For example, do not use emollients to prevent ED as studies have shown that this serves no purpose. There is no evidence of emollients in premature babies in preventing infection. Emollients have no impact in preventing food allergies.
In these situations of prevention with no evidence of efficacy in people without symptoms, the risks outweigh the benefits.
For inflammatory pathologies, conclusions will be drawn by the enviderm working group.

In terms of sunscreens, there as as yet no precise recommendations given by the enviderm group. It is important to privilege protective clothing and non-exposure during periods of vulnerability. Prefer products with labels including words such as “nature” and “progress”.

It is important to sensitise patients to the risk, explaining to them that they should:
- avoid over-use of cosmetic products
- avoid those containing parabens, triclosan, toluene and nanoparticles.
- prefer products with ecolabels
- avoid essential oils (risk of sensitisation)
- limit the simple prescription of emollients if there is no skin pathology.

A guide to the use of private practice doctors has been published by the PACA (Provence-Alpes-Côte d'Azur) URPS (Regional Union of Health Professionals). It contains practical cases. It can be requested or downloaded from **http://www.urps-ml-paca.com**

The chemical risk
by Dr A. C. Davaine

Dangerous chemical agents are labelled with pictograms explaining the physical dangers, the risks to health, the environment and aquatic organisms.

For us, there are chemical risks
- Seen in pre-disinfection, the cleaning of instruments, the cleaning of surfaces, formal and phenol.
- Emerging risks with nanoparticles (aerosolisation of disinfectants) and EDs

These molecules penetrate the skin and inside air. It is therefore important to ventilate our surgeries even outside COVID.

Plastics also have a pictogram; it resembles a green triangle with a figure inside. Classes 1-2-5 are recyclable. Classes !-3-7 are dangerous to health.

What should we do?
- Investigate: product sheets, pictograms, QR codes
- Eliminate what is unnecessary: perfume, deodorants, dyes
- Reduce the number of products and components; use correctly and at the right doses
- Protection using suitable protective equipment and appropriate ventilation.
- Replace with ecolabelled products (phago-soft) and biobased detergents.
- Thermal or mechanical cleaning of floors: professional steam cleaning or use of reusable polyester polyamide or acrylic microfibre wipes with bactericide standard NF EN 13697, applying the

maintenance procedure (cleaning 60 degrees and quick, complete drying).

Waste management

by Dr A. C. Davaine

There is treatment waste that is
- hazardous:

- DASRI: elimination traceability
- Chemical waste: recovery of containers by the manufacturer or specialised waste disposal operators
- Medicines: cyclamed
-The light tubes of phototherapy machines

- not hazardous: selective sorting

There are classic waste types:
- Hazardous: batteries, vials, electronic waste
- Not hazardous: selective sorting for recyclable, selective collection (surgical masks, ink cartridges), re-use, compost (examination sheet).

To reduce our waste, we an apply the rule of the Rs:
- REFUSE: boxes, advertising, samples
- REDUCE: share equipment, avoid duplicates, reduce disposal materials
- REUSE
- RECYCLE
- RETURN: compost
- REFLECT: change our habits

Digital quid

by Dr C. Pannequin

In 2020, digital accounted for 10% of world electricity consumption and 4% of greenhouse gas emissions, of which 25% came from data centres, 28% from infrastructures (networks, aerials) and 47% from equipment.

Equipment requires rare metals for its manufacture, which consumes water, transport and reduces our reserves. They are difficult to recycle (20% recycled, 4% buried or incinerated and &^% not managed).
It is important to keep material for as long as possible and to have polyvalent machines (printers, scanners, photocopiers), which reduces the number of items of equipment. By extending the life of the material from 2 to 4 years, the environmental impact can be reduced by 50%.
Switch off your equipment, a printer consumes 80% while on stand-by. Unplug chargers that are not being used by telephones, multi-sockets and boxes. Avoid stand-by mode (= 2 nuclear plants a year)
Avoid switching to 5G, your material will be obsolete more quickly. Purchase reconditioned material2

IT use.
Every little thing we do, has a cost. Internet pollutes 1.5 times more than air transport.
An internet search = 6g CO2
An e-mail saved = 10g CO2
An e-mail sent = 10g CO2
An e-mail sent with an attachment = 35g CO2
Information travels underground and under the sea via optic fibre.

To save, we need to empty mailboxes, dropbox, googledrive, photos, not “reply all”, add websites you use frequently to favourites and reduce the volume of attachments. By setting a smartphone to wifi, it consumes 4 times less energy that in 4G. Avoid the cloud, saving documents to external hard drives instead.
Use less energy-intensive browsers than Google Chrome, like Ecosia or Lilo.
Display webinars in low definition. Choose SMS messages over WhatsApp and Mail.

It is important to think things through in this all-digital era, considering paperless orders, given that a paper order weighing 20g sent by post consumes 18g CO2!

The impact of digital on our health.
Digital is responsible:

- for acne (2018 study by Prof. Dreno)
- for an increase in sedentary lifestyles and the range of comorbidities
- screen addictions

There is no data available on 4G and 5G frequency bandwidths nor on low frequency electromagnetic waves emitted by our portable telephones (carcinogenic role for humans?).

In conclusion: it is worth reflecting on the role of digital in a world with less and less energy, defining priorities for a societal reflection and envisage a conversion fro high-tech to low-tech.

Hot topics

Vitiligo by Prof. T. Passeron

- The risk of skin cancers: there is a lower risk of skin cancer than vitiligo. There is 3 times less risk of melanoma.
- Without natural or booth UV, it is extremely difficult to restore pigmentation.
- 40 to 50% of vitiligo lesions recur during the first year after repigmentation. This recurrence must be prevented.

- if the damage is limited, prescribe 0.1% tacrolimus twice a week, with no need for solar exposure. The risk reduces from 40% to 9.7%. Dermocorticoid drugs are probably just as effective but this has not been proven.
- if the damage is widespread, UVB can be suggested from 2 to 4 times a month by way of maintenance treatment. This is the opinion of an expert, not of a study.

- **The association of gastro-protected SOD and UVB**. gg

A prospective, randomised, blind placebo study has shown that the association of gastro-protected SOD and UVB allows for twice as much repigmentation at 6 months than placebo and UVB. This food supplement was given at a dose of 500mg 2x/day for 3 months, followed by 500mg/day for 3 months and associated with UVB twice a week. Tolerance was excellent and there were no side effects.

- Ruxolitinib cream (anti-JAK cream).

A study carried out on patients aged over 12 years old shows greater efficacy on the face than on the body. Tolerance is good, without any severe reaction at the application sites. Side effects are essentially folliculitis and minor pruritus. In July 2022, the FDA gave its authorisation. MA is expected for Europe for early 2023.

- Oral JAK inhibitors (ritlecitinib and tofacitinib).

Probable interest in oral JAK inhibitors but needs to be confirmed, notably for non-facial damage. Some zones are still very difficult to re-pigment with these approaches (hands and feet). Tolerance is good. Studies are needed in association with UVB.

What's new on JAK inhibitors in dermatology by Prof. J. Séneschal

There are 4 study models for JAK inhibitors in skin inflammatory diseases:
- Model 1: vitiligo and alopecia. Targets are IFNa, IFNg, IL 2, IL 15.
- Model 2: atopic dermatitis (AD). Targets are IL4, IL 13, Il 22, TLSP
- Model 3: psoriasis and Verneuil's disease. Targets are IFNa, IL 12, IL 23.
- Model 4: granulomatosis. Targets are IFNg, IL 10.

In alopecia, the JAK inhibitors tested are baricitinib and ritlecitinib. They are effective in moderate to severe alopecia. To obtain results, treatment is lengthy (24 to 36 months). The response is best if the alopecia is recent and not universal or completely balding.
In AD, we test upadacitinib, abrocitinib and baricitinib. The topical route is currently being assessed.
In psoriasis, we test deucravacitinib.
In severe granuloma annulare and sarcoidosis, tofacitinib is tested. It reduces inflammatory markers.
Numerous indications still need to be considered: chronic eczema in the hands, cutaneous lupus, GVH, prurigo, Verneuil's disease, systemic sclerosis, morphea and dermatomyositis.

The topical route allows for local skin immune regulation without skin atrophy, which allows for maintenance treatment of the face or genital area with only minor to moderate, local side effects. This route is not possible for all indications and is not suitable for a large area of skin.

The oral route is an alternative to SC injections of biotherapies. They have a broader effect that is of interest for more complex pathologies with more heterogeneous phenotypes and patients with multiple inflammatory pathologies. The clinical efficacy is rapid but the half-life is short, meaning that recurrence occurs after stopping. It is important to monitor for infection, cancer risk, cardiovascular accidents and the risk of deep vein thrombosis.

Focus on monkeypox infection by Dr Gentiane Monsel

Monkeypox or MPOX belongs to the Poxviridae family, like smallpox, vaccinia and cowpox. The current epidemic peaked in July-August. The outbreak has been limited thanks to modified behaviour of MSMs (reduced number of partners and reduced use of sexual meet-up applications), the efficacy of vaccination, the speed with which cases were handled and, perhaps, a reduced pathogenicity of the virus.
Transmission is by direct skin-mucosa contact with an infected patient, most frequently during sexual relations or respiratory secretions. Indirect transmission is possible by contaminated objects or laundry. Transmission to animals is possible.
The population at risk are MSMs, HIV positive patients, PrEP and ChemSex users, patients with a history of STDs and patients who have had more than 5 partners in the last 3 months.

Clinical expressions consist of:
- Primary lesions.

These appear 3 to 14 days after infection, there are umbilical pustules, ulcerations, black crusts associated with inflammatory, satellite and painful adenopathies. We see them on the genitalia, anus, oropharynx and skin.
- Secondary lesions, 2 to 4 days later. Pustules are generalised. They are associated with influenza-like illness (fever 50%, asthe2nir, myalgia, headache).

There are also complications: impetigo, dermo-hypodermitis, abscesses and scarring.
Diagnosis is clinical first and foremost but a PCR should be carried out if symptoms are non-typical or the contamination context is not characterised. It is important to swab a mucosal lesion for preference

and send it triple packed to a suitable laboratory, group 3 viral agent.
It is important to search for the other STDs, starting with blood tests for HIV, HBV, HCV, syphilis and a PCR for gonococcus infection and chlamydia trachomatis. For isolation purposes, the report must be completed by an anal, pharyngeal and vaginal PCR for gonococcus infection and chlamydia trachomatis. A sexual health consult after isolation is strongly recommended.

Probablistic syndromic treatment of other STDs should be applied depending on the forms:
- Urethral forms: ceftriaxone + doxycycline
- Anal-rectal forms without visible skin lesions: ceftriaxone + doxycycline
- Anal-rectal forms with perianal pustular-vesicular lesions: valaciclovir?
- Ambiguous forms of anal-genital ulceration; BPG 2.4 mIU in intramuscular injection +/- valaciclovir +/- doxycycline?

It is a disease that must be declared on an updated CERFA (administrative form review and registration centre) form.
It calls for 21 days of isolation from when symptoms start and until complete healing (disappearance of crusts). If going out, a mask is mandatory, along with covering clothing, gloves or bandages.
A condom must be used during sexual relations for up to 8 weeks after complete hearing, as the virus remains in sperm.
Treatment is symptomatic. Corticosteroids and NSAIDs are contraindicated. An antiviral treatment, Tecovirimat, may be prescribed in immunosuppressed individuals.

Vaccination using 3rd generation vaccines (Imvanex, Jynneos) is important:
- In pre-exposure in MSMs and/or transgender individuals with multiple partners, sex workers, professionals working in places of sexual consumption.
- Post-exposure in contact persons at risk, ideally from 4 to 14 days after first contact.

Prevention of infection risk in immune-modulating therapies by Prof. Valérie Pourcher

Assessment recommended with the start of biotherapy.

- At interview

Lifestyle, profession, travel, infection
Comorbidity, history (notably of infectious diseases)
Presence of foreign material
Smoking

- Paraclinical examination

HBV, HCV, HIV, CMV, toxoplasmosis, chicken pox (if no known history)
Depending on context: dental consult, gynaecology consult
Screening for latent tuberculosis: radiography of the lung and quantiFERON®

- Verification of personal and family vaccination status.

Vaccination is important, if possible before starting treatment and better during a period when the disease is quiescent.
The impact of immune-modulating treatments is variable. The immunogenicity of vaccines persists in patients treated with anti IL/TNF/JAK but reduces on anti-CD20 antibodies and corticotherapy.
Live vaccines are contraindicated in subjects treated with immune suppressant drugs, with biotherapy or oral corticotherapy at immune suppressant doses (>10mg).
DPT should be repeated every 10 years
Vaccine those around the patient

COVID vaccination (2 to 3 doses at 1 month intervals followed by a booster dose 3 months later)
Anti-pneumococcal vaccination is recommended as well as influenza and HPV.
There is no recommendation in respect of vaccinating against chicken pox. Consider this above all if thinking of using a TNF inhibitor.

- Run serology for hepatitis B (above all for anti-CD 20)

If HBsAg + and anti HBs Ac and anti HBc Ac are negative: assign curative treatment
If anti HBc Ac + anti HBsAg and Ac are negative (possible concealed hepatitis B infection): pre-emptive treatment.
If anti-HBs Ac and HBc + and HBsAg are negative: monitoring or pre-emptive treatment

---

Contact eczema (CE) in the child

By Dr A. Barbaud, Dr Brigitte Milpied and Dr N. Bellon

CE is children is frequent. Contact sensitisation is found in 40-50% of children tested. CE is responsible for 20% of dermatitis seen in children. It is possible at any age and frequency increases with age. The pertinence of positive tests in children is between 56.4 and 93.3%. Co-sensitisation is found in 3.2 to 54.4%.

When should it be mentioned?

- Palmoplantar involvement
- Treatment-resistant peribuccal involvement
- Periumbilical involvement or below the waistband
- Seat involvement
- Unilateral involvement
- Nummular eczema
- Eczema resistant to proper treatment
- Erythroderma
- Eczema and oedema of the face and eyelids.
- Worsening of a previously stable dermatosis

How can we explore it?
Importance of targeted interview.
We need to look for the 6 exposure methods:

- Direct
- Airborne (perfume, essential oils, paint, flowers)
- Manually taken (things they put in their mouths)
- Procured
- Photosensitivity (rare)
- What the patient has used to treat him/herself

The action needs to be reproduced as it may be relevant when looking for allergens

Always:

- Carry out the standard set of tests, this identifies 50% of causes.
- Test the products brought in. Do not hesitate to take cuttings from clothes, shoes or shin pads. Make 5x5cm squares, moisten them and carry out a semi-open test.
- Test medicines: dermocorticoid drugs, emollients, local antibiotics, preservatives, disinfectants.
- If the patch tests are negative, consider contact with proteins and perform prick-to-pricks.

- Do not trust food allergens in foods and cosmetic products (vegan or natural products). Risk of contact eczema or urticaria and anaphylaxis in the event of ingestion after cutaneous sensitisation.

The most frequent allergens are:
- Nickel: metals and electronic materials. Nickel can be found in an object by means of a dimethylglyoxime spot test. The applicator turns pink if nickel is present. There is also a spot test available for cobalt with Nitroso-R.
- Thiuram, MBT: rubber
- PPD/IPPD: black rubber, black henna tattoos
- Sesquiterpene lactones: plants
- Fragrance mix: perfumed products
- MIT or MCIT: paints, household products
- Fair few ingredients of household slime products (shaving foam, glue, washing up liquid, toothpaste, contact lens solution, etc.)

If diving or water sports clothes are suspected, thioureas are most often the culprit.
If shin pads are suspected, the allergen involved is acetophenone azine. Allergologists have no reason to test them.

Aluminium is the allergen of the year in 2022.
It is responsible for post-vaccination granulomas on the thighs. Children are sensitised through vaccines. This does not, under any circumstances, make vaccination contraindicated. This allergy reduces over time.
The risk factors are premature birth, a family history of granuloma, female gender and a young maternal age.

CE in atopic children.
One third of atopic dermatitis (AD) explored by patch tests have a contact allergy. The search for a contact allergy must be an integral part of exploration of AD.
The allergens found are the same as in the general population but topical medicines (antibiotics, dermocorticoids (DCs), emollients) are more frequent.
The risk factors of sensitisation to topical products are the severity of the AD, the early onset of the AD and IgE-mediated sensitisation.

When to test AD?
- Unusual topography or topography that suggests CE
- Change in location of the AD
- AD not responding to a properly applied treatment or worsening with treatment
- Before application of systemic treatment.

How to test?
It is important to test
- When flare-ups are not present and after the CD has ceased (2-3 days before).
- Some time after systemic immune suppressant drug treatment, oral corticotherapy and UV.
- **Testing while on DUPILUMAB is possible**

A standard set of “child” tests is run as well as specialised sets. Topical treatments of AD must always be tested. The reading is often difficult to interpret. Irritation is a frequent reaction seen (pustular or follicular reaction with metals). Late readings are important through to D7 for CD.
Complications are possible during testing: flare-up of the AD and “angry back”.

Isotretinoin: the main molecule of dermatology
Conclusion of the 2022 seminar, organised SFD (French Society of Dermatology) test centre and SFD themed group on facial dermatoses ("DEFI").

1- Isotretinoin and the psychiatric risk

Pharmacological epidemiological studies do not show any risk of suicide associated with isotretinoin on a population scale, nor any trigger effect of suicidal behaviour. By contrast, pharmacovigilance data shows a chronological and biological plausibility as to why isotretinoin plays a role in reported suicides. This calls for close monitoring of the psychiatric risk.

The board proposes:

- The inclusion in the information brochure of the ADRS (a 10-question scale assessing the psychological state of adolescents). It is available from the SFD website in the "score" tab.
- Prospective monitoring of patients with a high ADRS, outside the initial consultation and follow-up consultations.
- Check that prevention measures are in place in this population at risk.

2- Pregnancy and isotretinoin

The number of pregnancies while on isotretinoin has been stable since 2012 (175/year) despite the measures in place.

The board proposes:

- Strengthening information about mandatory double contraception in the case of oral contraception and on maintaining contraception during the month after stopping treatment (54% of pregnancies while on isotretinoin are discovered during this period).
- Check the HCG dosage 5 weeks after stopping.
- Co-prescription of condoms and emergency contraception: prefer levonorgestrel (NorLevo) over uliprstal (ellaOne).
- A pharmacological-epidemiological study on isotretinoin and IUDs should be considered
- Do not offer reassurance in the event of a normal antenatal ultrasound examination in the event of exposure to isotretinoin during pregnancy: recommend termination of pregnancy. Malformation syndrome is well known, but mental retardation has also been reported along with limited IQ, even at low doses.

3- Relevance of biological follow-up

- Hypertriglyceridaemia.

TGs increase by 20 to 40% before stabilising. High hyperTG (>5g/l) is rare (0.7%) and onsets during the first 2 to 3 months. Only 3 cases of pancreatitis have been described in women aged over 35 years old with pre-existing hyperTG and TG above 20g/l.

- Hypercholesterolaemia has no cardiovascular consequences, considering the duration of treatment.
- Asymptomatic hepatic cytolysis is frequent and resolves spontaneously with continuation of treatment. High cytolysis (ASAT or ALAT >5 times normal) onsets in 0.5% of cases during the first 2 months, before stabilising. No cases of symptomatic hepatitis secondary to isotretinoin have been reported in pharmacovigilance.

The board proposes a de-escalation of biological monitoring: TG, ASAT and ALAT to be dosed only at M0 and M3 unless there is an identified risk.

4- Should prescription of cyclines be required in the year before administration of isotretinoin?

No. There is no clinical pertinence in respect of this time frame of a year before starting treatment. Acne is a chronic pathology and patients have often had cyclines at some point before. Some clinical pictures are particularly at risk of permanent scarring and it should be possible to prescribe isotretinoin from the outset (follow the French SFD/HAS [French National Authority for Health]) guidelines to the correct use of isotretinoin and the absence of loss of opportunity).

Prescription outside the MA

1- Moderate acne in pigmented skin (1st line)

Considering the scarring risk of hyperpigmentation, this should become an indication. The board has implemented the ETHNIC trial. This is a therapeutic trial during acne in dark phototype patients (4 to 6), comparing early introduction oral isotretinoin vs classic strategy (hospital clinical research programme filed). It will allow for the strengthening of the recommendation made by the HAS: prescribing isotretinoin in first line in patients at risk of scarring.

2- Low facial acne in adult females (AFA)

Is there any interest in early treatment of AFA with isotretinoin, even if moderate, to obtain long-term benefits in respect of the evolution of the acne? Is there an optimal dose and duration of isotretinoin for AFA? Should a link or association with aldactone be considered to counterbalance the increased serum rates of S-DHEA observed on isotretinoin in some studies?
The board proposes studying and assessing these different propositions.

3- Pre-adolescent acne

The board proposes

- Studying/validating the use of isotretinoin in pre-adolescents in cases of severe acne, refractory to other treatments.
- The potential effect of isotretinoin on puberty should be studied
- Carry out a multidisciplinary reflection on the potential fitness and management of oral contraception in young, prepubescent girls.

4- Use of prolonged low dose isotretinoin for chronic, recurrent, dependent, highly-inflammatory acne

The board proposes both treatment through repeated administration and prolonged treatment. The choice must ultimately take into account the patient’s opinion.

5- Failing rosacea, recurring after properly-administered antibiotic therapy

Board proposals:

- Initial treatment of 4 months for rosacea with isotretinoin can covered by a compassionate prescription request made to the ANSM (French Agency for the Safety of Medicines and Health Products) with the laboratories marketing this drug.
- The interest in a maintenance treatment (low dose isotretinoin or doxycycline) to avoid recurrence may be recommended by academia once the indication of isotretinoin as initial treatment is made official.

6- Periorificial and perioral seborrhoeic dermatitis, mixed facial dermatitis

- Work needs to be carried out by the DEFI group on mixed facial dermatitis, so as to better characterise it (project subject to the 2022 call for tenders of SFD).
- At a later stage, low dose isotretinoin needs to be assessed during mixed facial dermatitis, by means of a randomised controlled trial versus placebo, using the same design as for rosacea.
- We need to study the interest of low dose isotretinoin in SD resistant to topical treatments, notably in the case of skin intolerance.

7- Verneuil's disease, follicular forms of Verneuil's disease

- The clinical characteristics of Verneuil's disease affecting the neck and face, should be clarified.
- It is important to identify which hidradenitis suppurativa patients are at risk of worsening on isotretinoin.

Reports written by

Dr. Christian MUTEBA BASEKE

Dermatologist, Democratic Republic of the Congo

Treatment of pruritus

By Laurent Misery

- A real problem
* Main complaint of patients
* They suffer as much as with pain
* Frequency: 30% of the population!
* Major impact on sleep, appearance (scratching gestures and lesions), mental well-being, emotional and sex life, quality of life
* Poorly understood up to now
* Effects of treatments are disappointing
* Primarily etiological treatments: dermatological pruritus, renal pruritus, hepatic pruritus, endocrine or metabolic pruritus, haematological pruritus, neuropathic pruritus, psychogenic pruritus, Iatrogenic pruritus

Treatment is symptomatic: general guidelines, local and general treatments.
* General guidelines: learn to replace scratching with the application of antipruritics; keep nails cut short; wash with cold or warm lukewarm water; use lipid enriched soap bars, syndets, emollients and moisturisers; avoid irritants (antiseptics) and anti-inflammatory drugs (both steroidal and non-steroidal); avoid nylon, wool, and tight-fitting clothes; avoid excessive heat and heating
* Placebo effect: placebo effects of 30%-70% on pruritus; topical treatments > systemic treatments; no relationship with underlying psychological disorders; relationship with verbal and non-verbal suggestions
* Cosmetics: mentholated (Pruritol, Atopicontrol Intensive), polidocanol: (Sensinol), algae extract (Xeracalm), combos (SOS Atopy Control, Sedacalm), calamine (calamine gel), Lipikar AP+, etc.
* Topical medications: dermocorticoids (solely in cases of inflammatory dermatoses), calcineurin inhibitors (tacrolimus: Protopic, pimecrolimus: Elidel, local anaesthetics (Versatis for shingles), capsaicin
* General treatment of pruritus: UVA, UVB, antihistamines, naltrexone, gabapentinoids, ciclosporin, psychotropics, psychotherapies, acupuncture

- Antihistamines: only effective in cases where histamine plays a role, MA: solely for uticaria and rhinitis
* First generation:
Alimemazine: Theralene
Cyproheptadine: Periactin
Dexchlorpheniramine: Polaramine
Hydroxyzine: Hydroxyzine

Mequitazine: Primalan
Promethazine: Phenergan

* Second generation:
Cetirizine: Zyrtec, Virlix
Levocetirizine: Xyzal
Loratadine: Clarityne
Desloratadine: Aerius
Ebastine: Kestin
Fexofenadine: Telfast
Mizolastine: Mizollen

- New treatments: naltrexone (MA for alcoholism), gabapentin and pregabalin (epilepsy, anxiety, neuropathic pain)

- Psychological approach: psychotropic drugs, psychoanalysis, analytical psychotherapies, relaxation, biofeedback, yoga, sophrology, CBT: cognitive behavioural therapies, brief therapies, hypnosis, meditation, therapeutic education, supportive psychotherapy, role of society and social circle

- Duloxetine (MA for depression, anxiety, neuropathic pain, of interest in neuropathic pruritus)

- Chronic prurigo
Distinct disease defined by the presence of chronic pruritus for at least 6 weeks, a history and/or signs of repeated scratching, and multiple localised or generalised pruritic skin lesions (whitish or pinkish papules, nodules and/or plaques) due to neural sensitisation to pruritus and the development of a vicious pruritus-scratching cycle (initial pruritus may be of any origin)

- Dupilumab (MA for AD, asthma, nasosinus polyposis)

- Renal pruritus in 2022
1st line: emollient creams, general measures
2nd line: capsaicin or tacrolimus if localised, UVB if generalised
3rd line: gabapentin, pregabalin, antidepressants, antihistamines
4th line: nalfurafine (MA in Japan: Remitch), naltrexone, setrons
5th line: acupuncture

- Neuropathic pruritus
Treatment of the underlying cause
Topical lidocaine (Versatis)
Compounded capsaicin
Capsaicin 8% (Qutenza)
Pregabaline (Lyrica), gabapentine (Neurontin) Cyclosporine (Neoral)
Thalidomide
Aprepitant (Emend)
Botulinum toxin
Acupuncture
Electrical stimulation

Pruritus: diagnosis

By Dr Jiachim WF

Classification of pruritus
- Clinical classification (IFSI)
* Step 1 - based on clinical appearance: pruritus and lesional skin, inflammatory pruritus, pruritus and non-lesional skin, non-inflammatory pruritus, pruritus with excoriations (prurigo)
* Step 2 - based on diseases: dermatologic diseases, systemic diseases, neurologic diseases, psychiatric/psychogenic diseases, mixed, other (no known cause)

- Neuroanatomical classification: based on the possible origins of pruritus
* Pruritoceptive: pruritus originating in the skin
* Neuropathic: pruritus resulting from a peripheral nerve lesion
* Neurogenic: mediators cause pruritus in the central nervous system with no nerve damage
* Psychogenic

- Diagnosis of chronic pruritus, general clinical diagnosis
* Location: generalised/localised pruritus?
* Chronological approach: Since when? Continuous/intermittent? Daily/seasonal?
* Pruritus characteristics: pure pruritus, burning, tingling, irritation, etc.
* Triggering factors: stress, sweat, dry skin, food
Other: Manner of scratching? Influence on sleep?

- Diagnosis of chronic pruritus
* Pruritus characteristics: pure pruritus, burning, stinging, prickling, piercing pain
* Pruritus evolution: point of onset, continuous or intermittent, daily or seasonal, increase or decrease in symptoms

- Diagnosis of pruritus
* Documentation of pruritus intensity
Verbal Rating Scale (VRS): 0-10
Numeric classification: 0-3
Visual Analogue Scale: 0-10
VAS = 5.6
Pruritus:
- Pruritoceptive pruritus: originating in the skin
- Dermatoses with pruritus: urticaria, mastocytosis, AD, psoriasis
- How does it develop in other organs: liver or kidney: nephrotic xerosis, accumulation of pruritogenic substances, production of pruritogenic substances, opioids, autaxin-LPA
- Systemic diseases associated with pruritus: renal (10-75%), hepatic (25-70%), Hodgkin's disease (30%)
- Diagnosis of chronic pruritus:
* primary workup: FBC/platelets, liver workup, renal function, TSH, total IgE, blood glucose, abdominal ultrasound, urinalysis, LDH
* Complementary workup according to the clinic and etiological orientation: microbiological samples, allergological diagnosis, skin biopsy, bone biopsy, BP180/230-antibodies, SPEP, AANS, tryptase, HIV, HBV, HCV serologies, other analyses
* Neuropathic pruritus: resulting from peripheral lesions
* Neurologic disorders
* Neurogenic-psychogenic pruritus: production of pruritogenic mediators in the CNS with no nerve lesions

- Diagnosis of chronic pruritus: psychiatric diseases: tactile hallucinations, delusional parasitosis, somatoform and dissociative diseases, schizophrenia, hypochondria, emotional factors (anxiety, fatigue, burn-out, depression, etc.), adjustment disorders
Eliminate other potential causes through psychiatric consultation!
- Medications often associated with pruritus: ACE inhibitors, statins, paclitaxel, GM-CSF, IL-2, hydroxyethyl starch (HES), opioids

- Practical conclusions
In cases of chronic pruritus on initially non-lesional skin, one should consider internal-nephrologic, neurologic and psychiatric diseases as well as drug side effects.
* If the palms of the hands or soles of the feet are itchy without primary lesions, the cause is usually a hepatobiliary disease.
* Aquagenic pruritus is common in myeloproliferative neoplasia.
* Basic laboratory diagnosis is recommended for general practice.
* Rifampicin and bezafibrate are of the most benefit in hepatic pruritus.
* As off-label treatments, gabapentin and pregabalin in kidney-friendly doses relieve chronic kidney disease associated pruritus (CKD-aP) in many cases.
* Difefalin has shown very promising study results in haemodialysis patients and was recently approved by the Food and Drug Administration (FDA) and the European Medicines Agency (EMA) specifically for the pharmaceutical treatment of CKD-aP.
* In myeloproliferative diseases, JAK-STAT inhibitors are particularly indicated for the treatment of underlying pruritus.

Acne and hormones

By Fabienne BD, Valerie B, Olivier C

- Preteens:
* 7-12 years of age.
* Increased frequency of preteen acne (4.8-6%), earlier onset of puberty.
* Probable role of lifestyle, diet, PE, screens.
* Affects girls more than boys.
* Appears before other signs of pubertal development.
* Most often, no underlying endocrinopathy.
* Increased SDHEAT
* Minimal to moderate acne, T-zone, retentional+++.
If severe acne + bone age advance + early puberty → hormonal assessment
* Difference between 7- 9 and 10–12-year-olds: severity, distribution of acne
* Risk factors: being overweight; consumption of candy, chocolate, & pizza; < 9 hours of sleep/night
* Average age of onset: 10.8 years, family history+++
* Acne type and severity: 95% inflammatory, 79% retentional
* Response to treatment: 12% relapse, 35% no response to treatment
* Preteen acne = risk factor for more severe and prolonged acne in adolescence.
* In acne-prone children: earlier onset of sebum production; greater bacterial diversity on facial skin: streptococcus+++, staphylococcus and cutibacterium, earlier proliferation of propionibacteria.
→ Therapeutic approach adapted to acne-prone preteens
→ EARLY treatment.
Appropriate hygiene care, topical retinoids; if mixed acne: combined treatment; no prolonged topical antibiotics; if topical treatment is insufficient and troublesome: per os antibiotics: short course of

erythromycin or azithromycin.
CI to cyclins if < 8 years of age.

- Hormonal assessment to be performed:
After 3 months of stopping hormonal contraception
Between D2 and D5 of a spontaneous or duphaston®-initiated cycle, in the morning
✓ Hcg, ✓ E2, LH, FSH, ✓ Total testosterone (+ SHBG in overweight patients), ✓ PRL, ✓ TSH, ✓ 17-OH progesterone.

- Pelvic ultrasound
Not recommended before 8 years post-menarche.

- Tumour-related causes: To be investigated.

- How should female patients be treated?
Recommendation #1
* In cases of moderate hirsutism and/or acne in non-menopausal women:
Combined oestrogen-progestin contraceptives are the first-line treatment.
* In the absence of sufficient efficacy, oestrogen-progestin contraceptives combined with spironolactone = the second-line treatment. This use is currently off-label (i.e., outside the MA).
* Spironolactone alone, with effective contraception, is the third-line treatment in the event of side effects, contraindications, and/or lack of efficacy of combined oestrogen-progestin contraceptives; this use is currently off-label.

- Adult female acne:
* Genetic predisposition in 50% of patients; CAG polymorphism of the androgen receptor is associated with severe acne in women; major role of androgens → peripheral hormonal pathology, normal hormonal balance, risks of antibiotic resistance

- Therapeutic choice: acne severity, presence or absence of hyperandrogenism, hormonal workup results, response to previous treatments, psychological and social impact, possibility of pregnancy, risk of scarring

- Influence of the exposome: nutrition, medication, pollutants, psychological & occupational factors

- Premenstrual flare-ups: no link between premenstrual flare-ups and acne severity

- Spironolactoma and acne: off-label use; used to treat acne since 1980; potassium-sparing diuretic; anti-androgenic action (sebogenesis inhibitor, 5α-reductase inhibitor, increases SHBG, reduces free testosterone); no antigonadotropic effect
Doses of between 50 and 200mg/day
Contraception recommended +++
Peak efficacy: 3-5 months
Average duration of treatment: 13 months
Results: 75% improvement
Same efficacy as cyclins
Efficacy on body acne

- Spironolactone and side effects: 5% frequency: gynecomastia, dizziness, etc.
- Interest of spironolactone: decreased use of systemic antibiotics; decreased risk of selection of resistant C. acnes; few side effects, non-phototoxic, no systematic biological monitoring; potential

combination with antibiotics, as a follow-up treatment to isotretinoin

- When to recommend spironolactone: in cases of moderate inflammatory acne, acne that is associated with hyperseborrhea and premenstrual flare-ups, acne that is resistant to conventional treatments, as an alternative to isotretinoin, in cases of moderate to severe acne, as a follow-up to AB or isotretinoin treatment, if acne is the result of Verneuil's disease

- In the case of moderate adult female acne:
* EARLY and EFFECTIVE treatment
* Spironolactone in cases involving hyperseborrhea, premenstrual flare-ups
* Isotretinoin in cases where there is a risk of scarring
* Systematic follow-up with topical maintenance treatment or spironolactone

- Anti-acne treatment and pregnancy:
* contraindicated: systemic (isotretinoin, tetracyclines, aldactone)
topical (tretinoin, adapalene)
* indicated: systemic (zinc gluconate, erythromicin)
topical (erythromicin, benzoyl peroxide, azelaic acid, fruit acids)

- Acne and menopause:
* Hormonal factors: decreased Oe, decreased HBG
→ Postmenopausal hyperandrogenism
* Obesity/hyperinsulinemia/hyperandrogenism link
* Eliminate other endocrinological factors
* Comparison of adult female acne and post-menopausal acne

Clinique	Acné femme adulte	Acné ménopausique
Site	Face+++ mandibules, joues	Face : périorale tronc
Sévérité	++	+
Type de lésions	Papulo-pustules	Papules Nodules profonds
Comédons	possible	macrocomédons
Papules inflammatoires	fréquentes	rares
Excoriations	fréquentes	Plus rares
Séborrhée	↗↗	↗
Réponse au TTT	+/-	+/-
Photovieillissement	+/-	+++
Stress / Dépression	+	+++
Signes hyperandrogénie	0	A rechercher +++

- If acne appears after menopause and is associated with signs of hyperandrogenism, look for an ovarian or adrenal tumour

- Treatment:
* Depends on the presence or absence of other signs of hyperandrogenism and severity

* Sensitive and reactive skin: appropriate hygiene care
* In cases where acne is minimal: topical treatment with anti-photoaging effect: topical retinoids+++
* In cases of moderate to severe acne: spironolactone with biological monitoring
* In cases of resistant, rapidly reoccurring acne: isotretinoin
* Physical treatments: NP, peels

The place of cosmetics in acne

By Fabienne Ballanger

- Pharmaceutical preparations:
* solutions, shampoos, lotions, tonics, micellar solutions
* emulsions: lotions, creams
* suspensions: toothpastes, foundations, BB creams, lipsticks, nail polish

- Composition of cosmetic products:
* active ingredients: single or multiple (product efficacy)
* excipients: neutral ingredients (vehicle for active ingredients)
* additives: preservatives

- Cosmetics and dermocosmetics:
* Dermocosmetics: products applied to the skin, scalp, and hair.
* They combine both cosmetic and dermatological action.
* They are formulated to preserve the health and beauty of the skin or hair
* They fall into the category of products sold under pharmaceutical advice
* They are recommended by dermatologists and general practitioners.
* “Cosmeceuticals”: cosmetic+ pharmaceutical
* Presence of pharmacologically active ingredients
* Complementary products, or even alternatives to medicines

- Active ingredients in cosmetics: antioxidants, niacinamide (nicotinamide), alpha-hydroxy acids, salicylic acid, lipo-hydroxy acid, glycolic acid, linoleic acid, lauric acid, retinaldehyde, zinc salts, fatty acids, oat plantlet extract

- Cosmetics and topical drugs in acne: where is the line?
* Targets of dermocosmetics: stratum corneum, maintenance of skin microbiota, control of innate immunity, action on sebaceous glands
* Repair of skin barrier alterations
* Pharmacological action < drug

- Interest of dermocosmetics in acne: the combination of topical and cosmetic treatments improves tolerance and treatment compliance

- Special case of isotretinoin:
* Isotretinoin impacts the skin barrier: modifies lipid composition
* Impacts the skin microbiome: increased colonisation by S. aureus.
* Advice and prescription of dermocosmetics +++
* From the beginning of treatment +++

* To reduce side effects, promote adherence to treatment, obtain better therapeutic efficacy, and improve patients' quality of life.
Water-based products containing active ingredients to repair the skin barrier.

- Cases of adult acne:
Moderate but chronic, recurrent acne
* Particularities: reactive skin, slow response to treatment, high rate of recurrence→ maintenance treatment
* Impacts quality of life
* Tendency to manipulation, excoriations and risk of scars
* Self-medication. Application of unsuitable products, i.e., "home-made products", etc.
* Choice of dermocosmetics +++

- Interest of cosmetics in acne: management of minimal acne, synergistic effect with topical anti-acne treatment, management of side effects, maintenance treatment, prevention of new lesions, reduction of inflammation, strengthening of skin barrier

- Photoprotection:
Sun exposure can aggravate or even trigger acne
Choice of suncare products: non-comedogenic, light emulsions, 30-50 index, UVA coverage, easy to remove

- Make-up: increases self-confidence

Physiopathology of acne: from research to treatment

By Prof. Brigitte Dreno

- Seborrhoeic imbalances: free fatty acids are increased in acne, and their mechanism of action has now been identified

- Palmitic acid activates the NLRP3 inflammasome in human sebocytes.
In terms of therapeutics: polyunsaturated fatty acids inhibit the NLRP3 inflammasome and could thus control acne inflammation

- The high number of sebum production-inducing receptors makes the control of hyperseborrhoea complex: hyaluronic acid, leptin, sugar, free fatty acids, cholesterol, histamine, androgens, stress, cannabidiol

- Hyaluronic acid receptors: reduction of sebum production individuals with high seborrhoea

- Androgen receptors: clascoterone 1%, which binds to the androgen receptor with a higher affinity than DHT with the effect of decreasing the transcription of genes involved in sebum production and inflammatory cytokines

- Early use of anti-acne treatments to prevent atrophic scars

- Inhibition of the inflammatory infiltrate:
topical tacrolimus is a macrolide calcineurin A inhibitor approved for atopic dermatitis.

It inhibits: T cell maturation and activation - VEGF with anti-angiogenic potential
Tacrolimus 0.1% ointment once nightly in 8 patients with macular erythema sequelae induced a visible reduction in erythema in 5-7 weeks. Follow-up at 14 weeks: no signs of relapse. No local or systemic adverse effects

- Sebaceous gland and follicle junction zone:
Persistent activation of Wnt promotes the formation of junctional zone (JZ) cysts that strongly express stem cell markers
Wnt activates the Hedgehog signalling cascade
Retinoid acid can reduce comedonal cysts through dual inhibition of Hedgehog and Wnt signalling.

- Microbiome and innate immunity: C. acnes, S. spidermidis, corynebacterium, other commensal bacteria, S. aureus, M. furfur

- C. acnes is a commensal bacterium of the follicle: maintains acidic PH of the skin, secretes a lipase, produces short chain free fatty acids (SCFA) and glycerol * Mainly acetate (C2), propionate (C3) and butyrate (C4), autophagic activity

- Acne is not related to C. acnes overgrowth

- Acne is due to a loss of C. acnes phylotype diversity due to the predominance of one phylotype: IA1, CC18, A1

- Loss of C. acnes diversity activates innate immunity with secretion of inflammatory cytokines, so restoration of C. acnes phylotype diversity suppresses inflammation

- C. acnes constitutively releases extracellular vesicles that increase keratinocyte proliferation, modulate keratinocyte differentiation with a decrease in keratin 10 and desmocollin 1 and an increase in filaggrin

- 2 bacterial actors in acne: S. epidermidis and C. acnes interact together, one inhibiting the development of the other and vice versa

- An imbalance between C. acnes and S. epidermidis induces the activation of inflammatory molecules including IL-1ra, IL-6, IL-8, Il17, etc.

- In therapeutic approach: s. epidermidis is the new target for acne treatments

- Benzoyl peroxide does not induce dysbiosis

- Washing can alter the microbiome: intensive skin washing, use of detergents, etc.
The therapeutic future:
* Vaccination: targeting virulence-related genes of C. acnes
* Bacteriophage: acne is found in the pilosebaceous unit, with potential phage therapy targeting C. acnes
* Bacteriotherapy (topical and oral): variety of isolated bacterial products or various bacterial species (bacterial transplants, probiotics (live bacteria)/prebiotics (bacterial extracts), pro/prebiotics combination, postbiotics: AMP

- The future of acne drugs!

1. Targeting the skin microbiome
- Probiotics, prebiotics
- Antimicrobial peptides (postbiotics)
- Bacteriophages: potential phagotherapy targeting C. acnes 1A1
- Bacterial transplantation

2. Targeting innate immunity
- TLR inhibitors
- Anti-IL1, anti-IL17 biologics

3. Targeting C. acnes
- Vaccination (anti-CAMP)
- Biofilm inhibitors (Myrtacine)

Acne and alternative medicine

By JP Claudel

The most popular “alternative” or “complementary” medicines:
osteopathy, hypnosis, meditation, relaxation, mesotherapy, aromatherapy, homoeopathy, traditional Chinese medicine/acupuncture, Ayurvedic medicine, “alternative” or “complementary” medicines, etc.

- Aromatherapy: “essential” oil is not an oil!
* EO: product obtained from a natural raw material of vegetable origin containing 99% volatile compounds
By steam distillation: hydrolysate (or floral water) and essential oil
Hydrodistillation (oleoresins, flowers, sawdust): immersed plant
By mechanical processes: citrus
By dry distillation (juniper and birch)

- The same plant can produce several EOs: bitter orange (bigarade orange) -> neroli, bitter orange, & small grain bigarade essential oils

Aromatherapy glossary
* Deterpenated essences = without limonene: favours oxidation and allergenicity
* Rectified essences = bergapten-free: photosensitising
* Extracts: plant exposed to 1 or more solvents, contains non-volatile compounds
* Absolutes: (perfumery and cosmetics) obtained by solvent (hexane), then ethanol, then evaporation...
* Chemotype: dominant chemical molecule of an EO

- But where does it come from?
* Parts of an aromatic plant: whole plant, flower, leaf, fruit, zest, resin, wood/bark, seed
* Different yields depending on the plant
* Different composition depending on the origin: example of Cinnamomum camphora

- Essential oil: biochemical cocktail
* 10 molecules for wintergreen oil and 450 for rosemary oil, etc.
* Terpenes, phenols, aldehydes, esters, ketones, ethers, coumarins, lactones, phthalides, etc.

* Concept of terpenes

- Not all that gentle ...
* To be avoided by pregnant women?
* Use with care in children less than 3 years of age
* Risks if it gets in the eyes or ears/pure EO on nasal mucosa
Phototoxicity/photoallergy

- To be avoided during pregnancy
* All essential oils containing ketones should be avoided during pregnancy (first trimester +++)
* Potentially neurotoxic ketones (abortive risk in cases of ketone intoxication)

- CRAT opinion on EO
* Do not alarm female patients inadvertently exposed to essential oils.
* In light of available evidence, the general recommendations during pregnancy are as follows: in terms of therapeutic use, it is preferable to abstain in the absence of data on both benefits and risks.
* Breastfeeding: in the vast majority of cases, there is no data on the passage into milk, nor on the condition of children breastfed by mothers using EO
General recommendations during breastfeeding = those of pregnancy

- Risk of EO endocrine disruption?
* Tea tree and lavender EO: three boys, respectively 4, 7 and 10 years of age, with gynecomastia
Derek V Henley et al. Prepubertal gynecomastia linked to lavender and tea tree oils. N Engl J Med. 2007 Feb 1; 356 (5): 479-85.
* Lavender EO (eau de cologne, air freshener)
Anti-androgenic action of several compounds in vitro
Niaouli EO (Melaleuca viridiflora)

- Many in vitro and in vivo studies and published reviews

- What about EO and acne?
* Anti-inflammatory action: Melaleuca alternifolia EO, Santalum album EO (sandalwood) (0.16%) with inhibition of COX-1, COX-2 and 16-LOX
* Antibacterial action on C. acnes & S. epidermidis (and aureus): Melaleuca alternifolia EO similar to other EOs
Synergistic action? Tea tree EO and Cymbopogon martinii EO (palmarosa)

- Homoeopathy’s 3 principles: the law of similars [principle of like cures like], the law of infinitesimal doses [principle of dilution] in terms of the active substance • DH= dilution of 1/10th, CH = dilution of 1/100th, and the law of individualisation and globality

- Homeopathic medicines are not all the same!

- In conclusion
* Aromatherapy:
true pharmacopoeia with indications, contraindications, and side effects • Efficacy shown in animals in vitro, but few studies in humans
* Homoeopathy:
no published studies with a high level of evidence
but frequently prescribed and used as self-medication in France

Is a systemic vitiligo treatment possible?

By Thierry Passeron

- How to recognise an active form of vitiligo: Koebnerss phenomenon, blurred edges, confetti depigmentation

- Active vitiligo should be treated as an emergency

- Corticosteroids:
* low dose prednisone, 0.3mg per kg for 2 months
*IV methylprednisolone bolus, 8mg per kg for 3 days
Stops progression in over 85% of cases
* betamethasone or dexamethasone minipulse twice a week on 2 consecutive days for 3-6 months
Stops progression in over 85% of cases

- Side effects: weight gain, insomnia, acne, agitation, etc.

- Other approaches: methotrexate, ciclosporin, minocycline (100mg per day), simvastine: 40 mg/day
- Treatment of active vitiligo with atorvastin + UVB. Note: UVB effective in stopping progression, atorvastin does not add to UVB treatment

How to block a flare-up:
* cortisone minipulse twice a week for 3-6 months
E.g., medrol 16 mg twice a week in an adult, 8 mg in a child, max. of 3 months in puberty
* UVB (TL-01) phototherapy 2-3 TIMES A WEEK also helps to induce repigmentation

- Interest in associating UVB and OMP in very active forms, blocks relapses in over 90% of cases

- Gastro-protected SOD and vitiligo
Rational
* Robust scientific data showing the role of oxidative stress in vitiligo
* But little or no clinical antioxidant efficacy
* Not all antioxidants are equally effective and are degraded during digestive passage

- SOD (superoxide dismutase) = key antioxidant enzyme

- Protection of its digestive degradation by gliadin

- Robust data showing its efficacy in animals and humans

Interest in the treatment of vitiligo?

- Gastro-protected SOD and vitiligo
Methods
*Prospective double-blind, randomised versus placebo study at Nice University Hospital: 50 patients with vitiligo affecting more than 5% of total body surface area
2 V-SOD gel applications in the morning and at noon (1/2 before meals) for 3 months and 2 gel applications in the morning for 3 months or placebo + UVB twice a week

* Blinded evaluation of treatment received on VES score by 2 evaluators
* Conclusions:
GP-SOD increases the percentage of repigmentation at 6 months under UVB phototherapy, 20% versus 9% for phototherapy alone
Population of patients with very old vitiligo resistant to one or more lines of treatment
Excellent tolerance
Interest of GP-SOD as a complement to phototherapy in vitiligo

---

How to evaluate the clinical severity of vitiligo and patients' quality of life?

By Khaled Ezzedine

- Importance of initial assessment: Wood's lamp examination

- Initial assessment: may influence the screening of autoimmune and auto-inflammatory pathologies.

- Importance of knowing and evaluating the VES (Vitiligo Extent Score), a disease extension score. But no evaluation of inflammation or potential for evolution.
www.vitiligo-calculator.com

- No obvious clinical signs of skin inflammation in vitiligo

- Signs of vitiligo's rapid progression include trichromatic vitiligo, confetti vitiligo, and Koebner phenomenon

- Assess signs of vitiligo activity with the VSAS score:
*Confetti-like depigmentation: C1-C2-C3, grade 1 < 10, grade 2 = 10-50, grade 3 > 50
* Koebner type: K1-K2-K3, grade 1 = 1, grade 2 = 2-5, grade 3 > 5
* Hypochromic areas/borders: H1-H2-H3, grade 2 = 2-5, grade 3 > 5

- Lesions that respond best to treatment:
* classification of vitiligo lesions according to the follicular reservoir: lesions close to follicular lesions have good depigmentation potential

- Quality of life and vitiligo: vitiligo has a major impact on patients' quality of life
* Public perception of the disease: depends on the cultural context, confusion with leprosy in some regions (India, Africa) and a major cause of social stigma in some countries.
* Many people are frightened and embarrassed by vitiligo
* Signs of discrimination, shame, depression, and anxiety are common and result in a social isolation and a frequent drop in self-esteem.
* Vitiligo has a major impact on sexuality

- Important quality of life factors:
*age of onset of the disease
* phenotype
* distribution and extension of lesions

- Factors affecting quality of life:

* vitiligo beginning in childhood and adolescence = lower self-esteem persisting into adulthood
* children with vitiligo limit their participation in sports and are more often absent from school
* greater stigma when lesions are visible
* photoexposed lesions are associated with higher DLQI scores
* the degree of stigma depends on the cultural context
* impact on quality of life is greater with phenotypes IV, V AND VI, patients report that their life would be different without vitiligo
* anxiety, depression, low self-esteem, and other psychiatric comorbidities.

How to optimise local vitiligo treatment: dermocorticoids and tacrolimus?

By Julien Seneschal

- Motivation and disease impact: treatment is long (more than 6 months) and daily

- Define the areas with the best chances of repigmentation with the patient:
* face/upper limbs and lower trunk
* folds, elbows and knees
* acral areas

- Updated international expert recommendation being finalised:
* geared more towards stabilisation than repigmentation
* use of strong and very strong dermocorticoids
* avoid application to the face due to the risk of side effects, applications: 15 days per month (once a day) over 6 months
* use on limited surfaces: risk of systemic passage
* potential use in children and adolescents

- Patients' opinion out of a sample of 325: 49% felt that treatment was not effective, 50% were not satisfied with their current treatment

- Evaluation of the efficacy of tacrolimus 0.1% for facial involvement in 42 patients: tracolimus 0.1% twice daily for 6 months:
* correct tolerance
* burning sensation on the face
* sensation of facial redness when drinking alcohol

- Tracolimus 0.1%: of interest in maintaining facial repigmentation:
* 40 to 50% of vitiligo lesions recur during the first year after repigmentation
* no preventive treatment for recurrence
* prospective two-year double-blind, bicentric, randomised vs. placebo study (Nice-Bordeaux) - facial vitiligo with more than 75% repigmentation

- Tracolimus 0.1% twice weekly vs. placebo twice weekly

- Assessment by 2 independent evaluators via standardised photographs: 40% of lesions depigmented in the placebo group and 9.7% in the tracolimus group, no significant difference in terms of vitiligo activity between the 2 groups, 1st effective treatment to prevent recurrence.

* Findings: difficult to use tracolimus outside MA for vitiligo
* Tracolimus: a good safety profile, children and adolescents included.

Reports written by

Dr. Marie-Eve PINET

Dermatologist, Quebec

10 systematic reviews that are an indispensable part of my practice

Speakers: L. Le Cleach, P. Senet, E. Sbidian

This presentation allowed us to address various pathologies seen frequently in the clinic and to compare our practices to those suggested by the most recent systematic reviews.

Treatment of chronic spontaneous urticaria

- Patients aged 12 and up:

- 2nd generation antihistamines at one dose per day
_ None of these antihistamines have been shown to be superior to any other
_ Note that some may prolong the QT interval

- In case of failure at a single dose:
_ Increase dosage to 4 doses per day
o Cetirizine and levocetirzine have the most data available
o Gradual increase vs. maximum dosage then decrease based on symptoms
o Dose can be divided into two administrations

- In case of failure at quadruple dose:
_ Add omalizumab 300 mg / 4 weeks long-term OR cyclosporin 3-5 mg/kg/day x 6 months
o More data and higher-level evidence for omalizumab; this should be the preferred option

- Recent treatments:
_ Anti-IgE antibodies: omalizumab, ligelizumab
_ Anti-IL4 antibodies: Dupilumab
_ Bruton's Tyrosine Kinase (BTK): remibrutinib, rilzabrutinib

- Among systemic treatments, note that cyclosporin, dapsone, hydroxychloroquine, zafirlukast, **ligelizumab and omalizumab** have shown better results than placebo

Moderate to severe plaque psoriasis

- Multiple systemic treatments are available:
_ Non-biological: fumaric acid esters, acitretin, cyclosporine, methotrexate
_ Biological: anti-TNF, anti-IL 12/23, anti-IL 17, anti-IL 23
_ Small molecules: apremilast

- Anti-IL 17 and anti-IL 23 treatments are the most effective according to current data

- Psoriatic arthritis*
_ Biological: CTLA-4, anti-TNF, anti-IL 12/23, anti-IL 17, anti-IL 23
_ Small molecules: apremilast, tofacitinib
_ Anti-TNF, anti-IL 17 and anti-IL 23 treatments are reported to be the most effective for achieving ACR 50
_ Note that ACR evaluates only peripheral involvement

- In patients with psoriasis, anti-TNFs may be associated with weight gain, but not with anti-IL 12/23 and anti-IL 17 treatments

Actinic keratosis (treatments for immunocompetent patients)

- Recurrence rate is lowest with:
_ Cryotherapy
_ 5-ALA-PDT
_ MAL-PCT
_ Imiquimod

- Recurrence rate is highest with:
_ Diclofenac
_ Ablative laser
_ 5-FU

Increase in relative risk of actinic keratosis, basal cell carcinoma and squamous cell carcinoma with thiazides

- Photosensitising effect
- Variable effect in different populations
- Variable effect depending on the lesion involved
- Highly heterogeneous results with regard to doses and treatment duration
- For melanoma, reported to be only slightly increased in non-Asians taking hydrochlorothiazide (HCTZ)
- Increased risk with HCTZ for squamous cell carcinoma (low)

Adjuvant radiation therapy in squamous cell carcinomas with free surgical margin

- Current data do not allow for any further optimisation of current guidelines
- Studies needed to clarify what circumstances should lead us to choose adjuvant radiation therapy

Risk of developing vulvar cancer in patients with lichen sclerosus or lichen planus

- No meta-analysis

- Lichen sclerosus:
_ Slightly increased risk (de novo risk 2.2%)
_ No association with duration of involvement
_ Not much data on risk factors: over 70 years old, 1-3 years after onset of lichen sclerosus?
_ Treating the condition appears to reduce the risk

- Lichen planus:

_ Risk may be slightly increased?

Classic cutaneous signs in internal medicine: Finding our bearings in practice.

Speakers: E. Laffitte, R. André, M. Jachiet

Supported by clinical cases, our speakers presented an overview of the underlying causes and treatment of cutaneous signs associated with systemic involvement.

Raynaud's syndrome

- Request an evaluation in atypical forms

- Capillaroscopy + AAN
- Imaging if unilateral presentation
- In case of necrosis or other systemic signs: ANCA, cryoglobulin, cryofibrinogen, cold agglutinins, complement, EPP

_ Cardiac ultrasound for embolisms, doppler and/or CT angio for Buerger's

- Verify medication history
- Treatment:

- Non-pharmacological

_ Keep extremities warm
_ Protect against micro-traumas
_ Stop tobacco use
_ Avoid vasoconstrictor drugs

- Pharmacological

_ Calcium channel blockers
_ ACEIs or sartans
_ Phosphodiesterase type 5 inhibitor
o Sildenafil shortens ulcer healing time, but limited activity with Raynaud
_ SSRIs
_ Iloprost
o Also shortens ulcer healing time
_ Bosentan
o Active on digital ulcerations, but only to prevent recurrence
_ Botulinum toxin?
o Possible option in case of ulcers

Chilblains

- Benign vascular acrosyndrome, linked to hypersensitivity to cold

- Prolonged abnormal vasoconstriction → cutaneous hypoxia → local inflammatory response (if predisposition is present)

- Recurrent macular/oedematous acral lesions
- If atypical: pseudo-chilblain (lupus or haemopathy) or differential diagnosis

- Typical conditions:

- Under age 30
- Predominantly female patients
- Family history
- Pre-existing acrosyndrome (Raynaud or acrocyanosis)
- Recent weight loss/underweight
- Clinical presentation

_ Pruritus, pain, burning
_ Erythematous to dark purple papules and macules, bilateral and symmetrical

o Sometimes oedematous or bullous

_ Toes, back of fingers

- Recurring flare-ups that last 1-3 weeks

o Diagnosis is usually clinical
o Evaluation if atypical: FSC, EPP, AAN

- Possible biopsy and/or capillaroscopy (in case of acrocyanosis/Raynaud)

o Treatment:

- Keep extremities warm and dry
- Stop tobacco use
- Stop use of vasoconstrictor medications
- Topical corticosteroids under occlusion
- Calcium-channel inhibitors
- Local treatments on ulcers
- Pain control

Sjögren's syndrome

- Suspect when

- Mentioned by patient
- Associated disease (lupus, scleroderma, dermatomyositis)
- Regularly recurring inflammatory joint pain
- Rash compatible with Gougerot-Sjögren
- Anti-SSA/SSB antibodies on an evaluation for another cause

- Questions for screening

- Dry eyes and dry mouth?
- Feels like you have grains of sand in your eye in the morning?
- Use of artificial tears?
- Regularly drink water during the night?
- Ability to eat rusks or a hard-boiled egg without drinking water?

- Possible objective tests

- Saliva flow
- Schirmer strip

- Evaluation in case of Sjögren's syndrome, even isolated

- AAN and anti-ENA
- Salivary gland biopsy if no auto-antibodies detected
- MRI of parotid gland?

- Treatment

- Little or no improvement with systemic treatments
- Sour candies
- Artificial saliva
- Artificial tears
- Muscarinic agonists

Systemic urticaria

- Urticarial vasculitis

- Fixed lesions (over 24 hours)
- Subsequent pigmentation
- Pruritus and pain
- Possible angioedema
- Normo- vs. hypocomplementenic

- Schnitzler syndrome

- Chronic urticarial rash
- Monoclonal IgG or IgM
- Recurrent fever
- Bone remodelling
- Neutrophilic infiltration on skin biopsy
- Leukocytosis and/or increased SR

- Adult-onset Still’s disease

- Flagellate urticarial rash
- Arthritis
- Fever
- Decline in overall health
- Weight loss
- Adenopathy
- Increased CRP
- Increased ferritin

- Neutrophilic urticarial dermatosis

- Non-pruritic pinkish macules

_ Regression in 24-48 hours without pignmentation

- Fever
- Arthralgia

- Perform a skin biopsy in case of:

- Atypical lesions (fixed, no pruritus, residual pigmentation)
- Combined with purpura, necrosis, blisters, livedo, Raynaud
- Extra-cutaneous signs: fever, decline in overall health, arthralgia, adenopathy

- Evaluation if appropriate for clinical orientation:

- Thyroid evaluation
- FCS, CRP, EPP
- AAN, anti-ENA, complement, ANCA

Livedo

- Caused by slowed blood flow in dermal venules

- Vasoactive: homogeneous blood stasis (livedo reticularis)

- Physiological:
_ Vasoactive
_ Limbs affected
_ Small, completely enclosed segments
_ Disappears with warmth or decubitus
_ No infiltration or necrosis
_ Isolated

- Pathological: heterogeneous blood stasis (livedo racemosa)
_ Presence on the trunk
_ Large open segments
_ Permanent
_ Possible associated necrosis or infiltration
_ Other systemic signs

- Causes of venous stasis:
_ External factor
_ Vessel wall (vasculitis, vasculopathy)
_ Intervascular (thrombosis, embolism, occlusion, viscosity)

- Broad differential diagnosis

- Evaluation in case of pathological livedo:
_ FSC, antiphospholipid antibodies, FAN, ANCA
_ If associated with cold: cryoglobulins, cryofibrinogen, cold agglutinins
_ Fusiform skin biopsy (net segment + centre of the segment)
_ Brain MRI, cardiac ultrasound
_ Evaluation of cardiovascular risk factors

- Treatment
_ Control of cardiovascular risk factors
_ Aspirin?
_ Monitoring

Atopic dermatitis in children: 10 clinical situations

Speakers: H. Aubert, S. Barbarot, C. Droitcourt, A.-C. Bursztejn

In this presentation, our speakers reviewed the various treatment modalities used with atopic dermatitis in children, based on the most recent recommendations.

Local treatment

- Rapid attack phase (1-2 weeks)
- Seeks to achieve rapid clinical remission and improved quality of life

- Daily corticosteroids adapted to age and lesion location
- No tapering off needed to stop
- No maximum recommended quantity

- Maintenance phase

- Seeks to maintain remission
- Immediate treatment of lesions from the first symptoms vs. application of topical corticosteroid or Protopic twice weekly
- Daily emollient

- Extremities are more difficult to treat and therefore require more powerful corticosteroids

- Important to explain treatment modalities to parents/patients and the chronic nature of atopic dermatitis

- Adapt the treatment plan to each patient

- Find the right treatment that meets the treatment objectives and patient's expectations

- Use of a magistral preparation may be useful, but the mixture can be done by the parent or the patient (in their hand)

- Potential interest for large affected areas and/or proactive treatment to control subclinical symptoms

- Emollient often poorly tolerated in acute phase

- Protopic is probably more appropriate for use in maintenance than with flare-ups

- Mainly on the face in adults
- No increase in cancer risk has been shown in children with atopic dermatitis

- In case of failure:

- Evaluate treatment adherence
_ Tendency to treat oneself less consistently when the condition is worse
_ Evaluate the patient's daily routine
_ Show how to perform the treatments
_ Address any potential corticophobia

- Look for aggravating factors (irritants or allergens)
_ Food allergy is rare
_ Contact allergy is the most frequent, but still rare

- Optimise local treatment
_ Order of topical treatments does not appear to be important
_ Corticosteroid diluted in an emollient
_ Moist bandages

- Reconsider the diagnosis

- Add antiseptics/antibiotics in case of secondary infection

- New treatments forthcoming:

- Crisaborole
- Delgocitinib
- Ruxolitinib
- Roflumilast
- Tapinarof

Systemic treatment

- Before age 6

- Methotrexate
- Cyclosporin
- Dupilumab
- Others?

- 6-11 years

- Dupilumab as 1st line
- Cyclosporin or methotrexate as 2nd line

- 12-17 years

- Dupilumab or upadacitinib as 1st line
- Cyclosporin or methotrexate as 2nd line

Immune deficiencies

- IKBKG mutation

- Women: mild signs of incontinentia pigmenti
- Men:
  _ Ectodermal dysplasia with immune deficiency
  _ Osteoporosis and lymphoedema
  _ Susceptibility to atypical mycobacteria

- Hyper-IgE syndrome

- SAM syndrome

- Severe dermatitis, multiples allergies, metabolic wasting syndrome

Primary prevention of atopic dermatitis and food allergies

- Foods are less allergenic than skin allergens
- Little evidence for the possibility of changing the atopic process

Severe early forms resistant to local treatments

- Strategies adapted to the disease’s changing profile?
- Anti-JAK?
- Bacteriotherapy?

- Selection of beneficial bacteria could control the skin microbiota

---

Challenging situations in paediatric dermatology

Speakers: J. Mazereeuw-Hautier, C. Chiaverini, E. Bourrat

The speakers presented various cases encountered in their practices which proved to be learning experiences in the context of paediatric dermatology. They discussed the following conditions:

Acute traction alopecia
- Case of acute alopecic plaques on the scalp of a girl with braids after wearing a bicycle helmet
- Reports of cases of scalp ulceration after trauma
- It is important to thoroughly question the patient/parents
- Speaker's recommendation: opt for large braids rather than small ones and take them out in case of pain

Langerhans cell histiocytosis
- Intermittent multidactylic erythronychia
- Erythronychia is a rare manifestation of Langerhans cell histiocytosis

Ulcerated abortive haemangiomas
- Painful ulcerations of the buttocks in an infant
- Increased risk of ulceration in the buttock region

Wong-type dermatomyositis
- Case of facial erythema in a 13-year-old patient manifesting as small confluent erythematous papules of recent onset.
- Appearance similar to pityriasis rubra pilaris
- Remember to look at the rest of the skin, in this case the hands

Epidermolysis bullosa
- Extensive bullous eruption of the skin and mucous membranes
- Appearance of linear IgA bullous dermatosis (LABD) – (rosettes)
- May be exacerbated by systemic corticosteroids
- So it's important to remember that rosettes are not exclusive to LABD

Atrophic pityriasis versicolor
- Round atrophic squamous papules on the back
- Form described with pseudo-atrophic appearance
- Yellow fluorescence under Wood's lamp
- Atrophy resolves with treatment
- Responds to usual topical or oral treatment

Panniculitis-like T-cell lymphoma + lupus of the scalp
- Progressive alopecic plaques on the scalp, non-scarring and with an inflamed appearance
- Possible appearance of comedones
- Indurated plaques, may be more or less diffuse
- Decline in general state of health, fever, polyadenopathy

Linear scleroderma
- Case of atrophic sub-palpebral plaque and ipsilateral oral lesion
- Requires an evaluation of extent which may include cutaneous, subcutaneous, facial, muscle and bone involvement
- Treatment must be aggressive to avoid sequelae as much as possible

- Examine mucous membranes when lesions suggestive of this condition are observed

Ketorash (prurigo pigmentosa)
- Pruriginous papulovesicular skin rash, particularly on the trunk.
- Subsequent reticulate hyperpigmentation
- Caused by a ketogenic diet that includes less carbohydrates and more fat and protein
- Treatment includes administration of sugar and check for ketonuria
- Stay informed of trends and behaviours among young people

Pathomimia
- Self-induced skin lesions
- Affects accessible areas of the body
- Most common in girls

Scurvy
- Follicular hyperkeratosis, perifollicular purpura, corkscrew hairs
- Haemorrhagic gingivitis
- Frequent lower limb pain
- Check for dietary problems

- Diet lacking in fruits and vegetables
- Vitamin C deficiency → reduced collagen synthesis → haemorrhagic syndrome due to capillary fragility
- Ensure correct handling of blood sample because vitamin C degrades easily
- Treatment via administration of vitamin C
- Be sure to check for other nutrient deficiencies

Secondary syphilis
- Painless ulcerated lesions of the palate + history of persistent anal fissures
- Men who have sexual relations with other men

- 3% of cases in patients under age 20
- Increasing prevalence in young patients
- Screening for other sexually transmitted infections

Hutchinson-Gilford progeria
- Case of a child presenting:

- Early appearance of sclerodermiform lesions on the back
- Bumpy appearance of the buttocks
- Low capillary density
- Increased visibility of veins of the scalp
- Associated delay in growth
- Associated bone involvement
- Autosomal dominant transmission, but often de novo mutation

Pseudoxanthoma elasticum
- Autosomal recessive transmission
- ABCC6 gene
- Clinical signs appear in the twenties or earlier
- On the skin, yellowish papules and sagging skin in adulthood
- Retinal and arterial wall involvement
- No treatment per se

Congenital mastocytoma
- Rare form
- Treatment:
- Serum tryptase test
_ Over 100 µg/ml suggests systemic involvement
- Perform investigations if:
_ Sharp break in weight curve
_ Signs of mast cell degranulation
- Possible treatment:
_ Antihistamines in case of pruritus or digestive symptoms
_ Topical corticosteroids on skin lesions
_ Avoid factors that exacerbate the lesion or systemic symptoms (friction, heat, cold, histamine-rich foods, certain medications)

Leprosy
- Caused by Mycobacterium leprae
- Endemic in South/Southeast Asia and sub-Saharan Africa
- Contamination by a patient infected with a multibacillary form
- Bacilli are eliminated via the nasal mucosa and the skin (wounds)
- Confirmation via skin or nerve biopsy with Ziehl-Neelsen stain + PCR
- Absence of pathogen in tests does not exclude diagnosis, so clinical and epidemiological signs are sufficient to make this diagnosis

Tuberous sclerosis complex
- Autosomal dominant transmission (TSC1 or TSC2 gene)
- Hamartomas that may appear in various organs
- On the skin, the most common sign is hypopigmented spots
- Other cutaneous signs: Koenen’s tumours, shagreen patches, angiofibromas, fibrous cephalic plaques
- Associated epilepsy
- Other affected organs: heart, kidney, brain, lung

Epidermolytic ichthyosis
- Autosomal dominant transmission (KRT1 or KRT10 gene)
- At birth: skin detachment/erosions
- Later, decrease in detachment in favour of hyperkeratosis
- Sometimes palmoplantar keratoderma
- Symptomatic treatment

Anhidrotic ectodermal dysplasia
- Only affects boys
- Girls are carriers with attenuated signs in the form of hyperpigmented linear lesions
- Xerosis, ezcema, pigmentation, periocular wrinkles

LEOPARD syndrome
- Acronym that covers the primary signs:
- Lentigines (multiple café-au-lait spots)
- ECG abnormalities (hypertrophic cardiomyopathy)
- Ocular hypertelorism
- Pulmonary stenosis
- Abnormalities of the genitals

- Retarded growth
- Deafness or hearing loss
- Caused by PTPN11 mutation (autosomal dominant)

Neonatal lupus
- Skin lesions within the first month of life
- Papular, often ring-shaped lesions, typically on the face (“raccoon”)
- Lesions resolve spontaneously within a few months
- Possible persistence of subsequent permanent changes
- Hepatic or haematologic involvement in some cases, generally also resolves spontaneously
- Hepatic evaluation, ECG and photoprotection for the newborn
- Don’t forget to do an autoimmune evaluation of the mother and monitoring of subsequent pregnancies
- May recur in adolescence

Fashion Week at JDP: when clothes and clothing accessories cause allergies!

Speakers: E. Collet, N. Raison Peyron, C. Leleu, F. Tétart

Many elements other than dyes may be involved in a case of contact dermatitis associated with clothing or fashion accessories
- Various materials
- Sizing agents and finishes
- Preservatives and fungicides
- New materials in use or in development…

Topography can often be a big help in diagnosis

Clinical appearance:
- Dry, symmetrical eczematous dermatitis
- Affects areas in close contact with the causal agent
- Promoted by friction and perspiration
- Sometimes atypical presentations

Tendency to resist topical corticosteroids

Careful examination of the suspected clothing or accessory is important
For patch tests, a good-sized sample is preferable
- The fabric may be moistened
- Use semi-open method
- A negative test does not completely eliminate the diagnosis

Dyes:
- One of the primary causes of textile allergies
- Large number of dyes reported
- The most commonly used:
- Disperse dyes
_ Used for synthetic fibres, never natural ones (linen, cotton, wool, silk)

_ Inexpensive
_ The most allergenic
_ Note that patients who react to Disperse Orange 3 react to PPD
- Reactive dyes
- Expect delayed detection (22% delayed reactions with disperse dyes)
- Advice for patients:
- Read labels to familiarise yourself with fibre composition
- Wash new clothes before wearing them
- Choose natural fibres and white underwear in case of allergy to disperse dyes

Metals: in textiles, shoes and fashion items:
- Cross-sensitisation is common among metals
- Frequent sensitisation, but relevance is not always clear
- Dimethylglioxime spot test detection kit to detect presence of nickel (an equivalent also exists for cobalt)
- Only possible on metallic objects
- Can be used by the patient
- Regulations exist for the composition of items containing metal, especially nickel

But unfortunately they are not always complied with
- Chrome is also used in leather tanning
- Are “chrome-free” claims on shoes reliable?
- Allergic contact dermatitis is not uncommon in children
_ Affects soles of feet
_ Spares areas not in contact with the shoe
- In case of a proven allergy:
- Opt for purchases in regulated areas
- Purchase spot tests for nickel and/or cobalt
- Lined shoes, avoid moisture, barrier cream (uncertain value)
- Remember that effects may be asymmetrical, even with shoes

Rubber
- Vulcanisation agents
- Thiurams
- Benzothiazoles
- IPPD
_ Black rubber (e.g. tyres)
- Dithiocarbamates
- Guanidines
- Thioureas
_ Used in neoprene production
>> Clothes and sporting accessories
>> Waterproofing and thermal insulation
- Other

Textile finishes and sizing agents
- Stabilises the clothing’s shape
- Wrinkle-proofing and resistance to repeated washing
- Improves dyeing and bleaching
- Mite resistance
- Anti-static properties
- More pleasant texture

Rosin
- May be found in shoes
- Adhesive used in heel and toe supports
- Latex adhesives
- Shoe polish
- Plastic
- Leather tanning or finishing agent

Other allergens
- Acetophenone azine:
- Allergen in individual sporting equipment and footwear
- Mainly affects children
- Eczema, sometimes severe (generalised or severe dyshidrosis of the feet)
- Formaldehyde releasers
- Most commonly found in cosmetics
- Methylisothiazoline
- Silk – most commonly with urticaria-type reaction

Are you a myco master? Test your knowledge of mycological and mycobacterial cutaneomucosal dermatoses

Speakers: D. Kottler, A. Dompmartin, M. Lehericey, A. Leymarie

1. Mycoses

a. Dermatophytes

i. Reproduction via keratinising spores or arthroconidia

ii. Development in the stratum corneum and adnexa

iii. Anthropophilic, zoophilic, telluric or geophilic
1. More inflammatory clinical signs with zoophilic mycoses (may even appear to have a secondary infection due to the pustular and crusty appearance, but this is not the case)
2. M. canis can heal on its own in animals… making investigation more complicated

iv. Genera: Microsporum, Tricophyton, Epiermophyton

v. Clinically, round centrifugal plaques
1. May be altered by the use of topical corticosteroids

vi. Examine the skin thoroughly, including hands and feet

vii. Important to use physical treatments and manage local factors during treatment
1. For pustules, abscesses and necrosis: mechanical and surgical treatment

b. Mould

i. Haematogenous diffusion or invasive mycoses
1. Presence of necrotic lesions on the skin

ii. Sometimes involved in onychomycosis
1. Confirm with direct examination + two analyses via culture or PCR
2. Grinding
3. Topical treatment (amphotericin B solution, can be combined with a urea-based keratolytic cream)
4. Systemic treatment?
5. Mechanical avulsion of the nail

iii. May be associated with a dermatophyte

iv. Mainly affects immunosuppressed patients (systemic corticosteroid therapy, leukaemia, lymphoma, transplant patients, etc.).

c. Yeasts

i Includes candida, malassezia and cryptococcus

ii. Reproduction by budding

iii. Candida:
1. Normally present in the digestive tract and female genital mucosa
2. Majority of cases caused by C.albicans, C. glabrata, C. tropicalis, C. parapsilosis and C. krusei
3. Response to antifungals is variable, depending on species
4. Non-albicans: 20% of vulvovaginitis cases
a. Resistant to azoles
5. Emergence of resistances
6. Candidosis risk factors:
a. Extended exposure to acidity
b. Age, physiological condition
c. Pathological states (diabetes, Hodgkin's, AIDS, immune deficiencies)
d. Therapeutic factors (antibiotics, corticosteroids, immunosuppressors, psychotropics, radiation therapy)
i. Associated with use of anti-IL-17 drugs or immune deficiency affecting the production of IL-17
e. Drug addiction, malnutrition…
7. Hair is not affected (but skin, mucosa and nails are)
8. Clinical presentation:
a. Affects bottom of folds, oozing, small satellite pustules
b. Periorificial involvement with desquamative collarette on the buttocks
9. In case of doubt, take a sample
10. Treat all sites simultaneously
a. Topical treatment +/- fluconazole (right away in case of oesophagus involvement)

d. Samples

i. Avoid during antifungal treatment (at least a 15-day interval)

ii. Provide details of clinical context to the lab

iii. Sample where the fungus is located

iv. Direct examination: fast and simple
1. Essential before a treatment
2. Low sensitivity (negative in 50% of cases)

v. Culture
1. Variable time
a. Ordinary yeasts in less than a week
b. Up to 3 weeks for common filamentous fungi
c. 6 weeks or more for exotic mycoses
2. PCR
a. Specific vs. panfungal
i. Aspergillus and mucorales can be detected by specific PCR tests
ii. Panfungal PCRs have low sensitivity, low specificity and are costly

e. Treatments

i. Local
1. Amphotericin B: yeasts and moulds
2. Terbinafine: dermatophytes, Candida
3. Imidazoles: dermatophytes, yeasts
4. Amorolfine: dermatophytes, yeasts
5. Cyclopyroxolamine: dermatophytes, yeasts, bacteria

2. Mycobacteria

a. Tubercular

i. Subsp. tuberculosis: tuberculosis
ii. Subsp. bovis: contact with livestock or transmitted through milk
iii. Subsp. africanum: tuberculosis, especially in tropical Africa
iv. Bacillus Calmette–Guérin

b. M. leprae

c. Non-tubercular

i. True pathogens (e.g. M. ulcerans or M. marinum)

ii. Opportunistic pathogens (e.g. M. avium, M. abscessus, M. chelonae)
1. M. chelonae
a. Rare infection
b. Indolent
c. Does not always heal

iii. Saprophytic or commensal

d. To make the diagnosis

i. Remember
ii. Skin biopsy

iii. Direct examination
iv. Culture
v. Tuberculosis PCR
vi. Histology
1. Caseous necrosis

e. Treatment

i. According to the resistance profile of the species
ii. Clarothromycin is the most commonly used antibiotic
iii. Longer treatment in case of immunosuppression

Important messages from our speakers:

- Interpret lesions according to clinical context (immunosuppression, comorbidities)
- Perform a complete clinical examination including mucosa, hair and nails
- Lesions often atypical via prior therapeutic trials
- Possible association of mycoses with inflammatory dermatitis
- Sample the largest possible quantity of hyponychial keratin/tissue
- Include a mechanical component in the treatment
- Beware of risk of tuberculosis reactivation risk with anti-TNFs (currently not reported with interleukins)