BIODERMA
CONGRESS **REPORTS**

JDP 2024 Bioderma Congress Reports

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Child abuse: recognising the cutaneous signs and appropriate management

Coordinator: Hélène Dufresne (socio-educational manager in the Paediatric Dermatology Department at Necker)

Epidemiological data and legal framework - Hélène Dufresne

An article in Le *Monde* published in 2024 shows that all the indicators for child protection are red. Nearly 3,000 children live on the streets and 5,000 are awaiting placement.

Units for the collection of worrying information (CRIPs) were set up in 2016 and the Taquet Act voted in February 2022 strengthened the protection of young adults leaving the child welfare (ASE) system.

According to the law, a child is in danger if they are facing difficulties or a risk that endangers their health. Abuse goes beyond the notion of physical or sexual violence; it can involve acts or the **absence of acts (neglect)**.

The COVID pandemic has changed lifestyles, with a steady increase in violence, particularly **defenestration**.

The various types of abuse:

- Physical
- Sexual abuse or solicitations
- Psychological
- Neglect, or failure to meet a child's basic needs.

Most of the perpetrators are adults, but some are teenagers and children. The child's siblings must **systematically** be looked at. In 95% of cases, the perpetrator is in the immediate family (it is the mother in 50% of cases and the father in 36%).

Abuse has an impact on a child's overall health and development, which is why it is so important to act at an early stage.

There is a legal obligation to report child abuse; anyone can report it.

- As a citizen
- As a doctor – they must protect any minor who has been the victim of abuse or deprivation by authorising the lifting of medical confidentiality.
- Any local social assistance office can help us.
- CRIPs, UAPEDs (hospital child protection units that can help **private practitioners**)

What are the warning signs?

- A sudden increase in head circumference
- Retarded growth
- Enuresis
- Encopresis
- Traces of blows, burns or bruises
- Drug poisoning
- Various behavioural problems (eating disorders, disinvestment)
- A child seemingly subject to secrecy

Always undress the child completely, look at them and fill in their health record.

Many people who have been victims of maltreatment or sexual abuse do not show any specific signs.

The report can be written anonymously and without the involvement of a social service.

We can report abuse even if we only suspect it; the French Health Authority (HAS) and Medical Council (CNOM) provide several reporting templates.

With regard to the written text, **it is important to remain factual and objective, use inverted commas to report conditional statements, not accuse anyone, ask for a**

doctor's agreement to quote them, and inform the parents of the written text (with no obligation to provide its content) UNLESS it endangers the child.

For further information and templates, visit the CNOM website under "le médecin face à la maltraitance" (Doctors and abuse).

Evocative dermatological signs - Dr. Julie Bonigen

Dermatological signs are present in **90% of child victims of physical abuse, which is a type of abuse that is more common before the age of three years.** Half of these children go home with a misdiagnosis; it is important to be wary of dermatological lesions at different ages.

- Bruising

It corresponds to a traumatic contusion with no skin breach vs. haematoma, which is palpable.

Before four months, it is a case of abuse until there is proof to the contrary, or even up to nine months, before the acquisition of the beginnings of autonomous mobility.

It then becomes commonplace after the age of walking (affecting around 50% of children). **The areas that should raise the suspicion of abuse are: unexposed areas, the buttocks, genitals, thighs, arms, head, back and abdomen (violent shock)**, and the shape of the lesions.

- Burns

This is the presentation in 8-20% of cases of abuse.

A delay of > 2 hours between the burn and the consultation is the **1st warning sign.**

Accidental burns are more likely to be superficial, with incomplete burn marks, than deeper, multiple, **well-defined** inflicted burns.

In the case of forced immersion, we can observe a zebra-like appearance with respect for the skin folds or a "donut" shape (buttocks touching the bottom of the bathtub are spared).

- Abrasions/erosions should be interpreted according to location and age.
- Nail detachment, traumatic alopecia, bites.
- The oral mucosa: tearing of the lingual frenulum, dental fractures, erosions, burns.
- Genital warts:

Transmission is not only sexual.

Hypotheses: transmission during pregnancy, childbirth, by direct contact or via bathroom linen.

When should you suspect sexual abuse? There is a great deal of variability in the literature – but an increase is noted with age (this is less common before the age of four months) with genital involvement being more frequently associated with abuse.

Phthiriasis is not synonymous with abuse.

Pathomimia, which refers to self-inflicted lesions that the patient does not admit to. They should raise two questions: are we sure of the diagnosis (abuse?) and is this a cry for help?

Clinical cases – Prof. Smail Hadj-Rabia

Professor Hadj-Rabia stressed the red flags that must not be repeated when a report is made:

- No direct accusations
- No value judgements
- No recommendations
- No unnecessary information, breach of medical confidentiality if no report
- Do not say which body should investigate.

Key messages:

- Presentations of abuse are polymorphous and non-specific.
- We have a legal obligation to report.
- The CRIP, UAPED and CNOM can help us draw up a report.
- Be particularly wary before the age of three years and even more so before the age of nine months.

Alopecia in children will no longer be a mystery: step-by-step diagnosis and treatment

Coordinator: Dr. Sophie Leducq (Dermatology Department, Tours University Hospital)

Speakers : Dr. Stéphanie Mallet (Dermatology Department, Marseille University Hospital), Dr. Fanny Morice-Picard (Dermatology Department, Bordeaux University Hospital), Prof. Juliette Mazereeuw-Hautier (Dermatology Department, Toulouse University Hospital), Dr. Sophie Leducq (Dermatology Department, Tours University Hospital).

Localized congenital alopecia

Case 1 involved **congenital alopecia of the scalp**. Brain MRI is indicated if:

- Large lesion
- Median location with bone defect
- **Hair collar or hair tuft sign.**

In the case of an isolated small lesion, transfontanellar ultrasound is sufficient.

Case 2 was a child with congenital neonatal occipital alopecia. This is very common, affecting 20% of newborns, and corresponds to the **physiological shedding** of telogen hairs initiated in utero. There is a misconception about the occiput rubbing against the mattress: **do not turn babies over in their cots**. The differential diagnosis is halo scalp ring during prolonged local ischaemia during childbirth, which can be scarring.

Case 3 was a case of congenital triangular temporal alopecia, which is bilateral in 20% of cases.

Diffuse congenital alopecia

Case 1 was a five-year-old child with short, thin, sparse hair and tooth agenesis. The question to ask is: **does your child sweat? Look for hypohidrosis** as part of ectodermal dysplasia, which can also affect the nails and mammary gland. The risk is **malignant hyperthermia.**

Case 2 was a three-year-old child whose hair was growing little and falling out easily. This corresponded to loose anagen hair syndrome with > 50% of hairs in the anagen phase on trichogram with ruffled cuticles. The pull test is a good diagnostic tool, and the condition improves spontaneously.

There are many genes involved in isolated hypotrichosis.

Acquired inflammatory alopecia – Alopecia areata

Alopecia areata is **not so rare**: 0.29 cases per 1,000 patients per year, girls > boys in paediatrics and Asians > blacks. It is **disabling**, causing embarrassment and a poor self-image, according to eight paediatric studies looking at the quality of life of these children.

The French alopecia areata association is called **La Tresse.**

Diagnosis is clinical and often straightforward, but it can sometimes be more complex, particularly in cases of diffuse hair loss. Take a close look at the **eyebrows and nails (thimble-like pits, trachyonychia and leuconychia).**

<u>On dermoscopy, the suggestive signs are:</u>

- Yellow dots **(active alopecia areata)**
- Black dots **(active alopecia areata)**
- Exclamation mark hair **(active alopecia areata)**
- Shrinkage at the base of the hair
- Broken hair
- Vertical regrowth
- Downy hair
- Corkscrew hair

There is no consensus on the biological work-up, but Prof. Mazereeuw recommends a CBC, ferritin, vitamins B9/B12/D, zinc and TSH.

<u>Concerning management:</u>

- *In localised* forms: local corticosteroids: kenacort is painful and minoxidil not useful. Tacrolimus, laser and phototherapy have little data and do not appear to be effective.
- *In extensive forms*: it is important to provide information about this **chronic disease, characterised by flare-ups and remissions with only suspensive treatments.**

Ritlecitinib has European MA from the age of 12 in the 1st-line treatment of severe alopecia areata (> 50%) at 50 mg per day on a standard prescription.

Acquired inflammatory alopecia – Ringworm

The classic presentation involves purulent and/or scaly lesions.

There may be anthropophilic, zoophilic or telluric species.

Trichophytic ringworm produces **small patches of dry alopecia vs. the microsporic form, characterised by large alopecia patches.** Wood's lamp is a diagnostic aid and can sometimes point to the causative germ. Dermoscopy can rule out differential diagnoses in cases of ringworm **with little or no scaling**.

The four most specific dermoscopic signs are:

- **Comma hairs (endothrix - trichophyton)**
- **Corkscrew hairs (endothrix - trichophyton)**
- **Zig-zag hairs (ectothrix - microsporum)**
- **Bar-code hairs (alternating white and black stripes) (ectothrix - microsporum)**

At week 12, despite a negative culture, non-specific signs such as scales, black dots and erythema can still be observed, but the **four specific signs have disappeared**.

!!! Itraconazole syrup has not been marketed since the end of November 2024!!!!

The thematic group has published new recommendations on the SFD website.

For children under 20 kg with microsporic ringworm, we should try ¼ tablet of terbinafine for six weeks +/- two weeks. If over 20 kg, ITRACONAZOLE 100 mg tablets.

Mechanical alopecia

The first case involved poorly delimited non-inflammatory alopecia of the vertex that had been going on for a few weeks, with a negative pull test and normal appendages => **trichotillomania:** single, irregular, sudden alopecia (a single patch), beware of eyebrow +/- eyelash involvement.

Dermoscopy:

- **Broken hairs of different sizes**
- **Black dots**
- **Tulip hairs (damaged distal end)**
- **V sign.**

The patient usually does not admit to pulling their hair, and management is psychological (cognitive-behavioural therapy).

The second case was a little girl being treated with topical corticosteroids for alopecia areata; she presented with clear worsening of the retro-auricular region => **traction**

alopecia with alopecia areata, where the mother was pulling the remaining hairs to mask the alopecia. Stop all traumatic factors; administer topical corticosteroids if papules.

Acquired diffuse thinning alopecia

A 13-year-old girl who had had alopecia for two years => **androgenetic** alopecia. There is symmetrical recession of the temporal gulfs, vertex involvement, **anisotrichosis = different hair sizes**, hormonal assessment **often normal**, mixoxidil is proposed off-label. VS.

Telogen effluvium is often sudden and caused by physical or psychological stress. It is important to reassure patients that the episode lasts two to four months with constant regrowth. It can sometimes become chronic, so deficiency testing should be carried out, as well as looking for hormonal disorders and investigating any use of medication, and be wary of diffuse alopecia.

Tumour lesion

Brain MRI is only requested in cases of segmental infantile haemangioma of the cephalic or pelvic extremity.

A sebaceous hamartoma can progress into a benign tumour and can be removed for cosmetic purposes at a fairly early stage in life.

Congenital naevi of the scalp do not require brain imaging unless there are **several congenital naevi and/or a neurological starting point**. Spontaneous lightening is sometimes observed, and surgery is a major procedure. **The earlier proliferation nodules appear, the less serious they are.**

Hair shaft abnormalities

A two-year-old child with sparse short dry hair with a beaded appearance => **monilethrix;** micronychia and keratosis pilaris can be observed. The onset is early, with variable expression, transmission is autosomal dominant and the genes involved are **KRT 81/83/86**.

A newborn with no family history who, at around two months, presented with hypotonia, convulsions and very fine, dry, fair hair. The hair was twisted into pili torti, and **copper levels had collapsed => Menkes disease.**

A four- to five-year old child with a history of severe eczema at birth, stunted growth, sparse and brittle **bamboo** hairs = **which were folding into each other = trichorrhexis invaginata** => Netherton syndrome, which combines neonatal erythroderma, atopy and polysensitisation, sometimes with **Comel's** linear circumflex ichthyosis, SPINK5/LEKTI mutated gene.

A three-year-old patient with short, dry, very brittle hair + scaly legs. He was born a collodion baby with moderate improvement on the skin but persistence on the scalp.

Dermoscopy: **trichoschisis = fracture**, tiger tail = trichothiodystrophy, photosensitivity in 50% of cases.

An 18-month-old infant with very blond, dishevelled hair with an unusual appearance, with pili trianguli and canaliculii on microscopy = **uncombable hair syndrome**, where patients are in good health and the condition tends to improve.

Dr. Stéphanie Mallet (Dermatology Department, Marseille University Hospital).

An eight-year-old child with years of hypotrichosis, woolly/curly hair, angular cheilitis, recurrent cheilitis and, more recently, fissured striated palmoplantar hyperkeratosis => **Carvajal Naxos syndrome**, a desmosomal disease; cardiac monitoring is required; results in dilated cardiomyopathy, rhythm disorders and sudden death.

Changing hair colour

The first case involved a 13-year-old child whose hair had been greying since the age of eight => **Premature greying =** starts before the age of 20 in Caucasians, 30 in Africans. The causes are hypothyroidism, Plaquenil, chemotherapy, B9/B12/zinc/copper deficiency or constitutional.

The second case was a child with lighter then light brown proximal lines distally with a **flag sign;** various causes: iatrogenic, toxic, deficiencies (tyrosine kinase inhibitor, methotrexate, Plaquenil, metals, alcohol, malnutrition).

The last case was a girl with green hair **at the distal ends** = **green hair** linked to copper sulphate crystals found in swimming pool water or propofol in anaesthetic products.

Teledermatology: what new modes of organisation? What innovations? What prospects?

Coordinator: Dr. Matthieu Bataille (Saint Vincent de Paul Hospital, Lille)

Introduction

Teledermatology has exploded since the COVID pandemic and the growing shortage of doctors led to it being reimbursed under the French health insurance scheme.

We are currently in the process of familiarising everyone with these tools.

The role of advanced practice nurses (APNs) in dermatology-oncology and stabilised chronic disease seems promising.

Dr. Fabrice Ribeaudeau (private dermatologist in Le Mans) - Practical example

Dr. Ribeaudeau works in an under-resourced area and has to manage the shortage on a daily basis in his practice.

1. Medical assistants: He has two State-certified nurses in his practice, who are responsible for PDT, phototherapy, undressing, interviewing, pre-examining, assisting with surgery, patient education with biotherapies (qualified), initial injections, taking photos, cryotherapy and Curacné monitoring.

He also has physical secretaries who handle reception, billing, consent forms, correspondence, complementary examinations and **sorting appointments.**

2. Tele-expertise: He is the only one to do this in the Sarthe *département* of France, including 90% via Omnidoc. The aim is to sort out complaints, but also out of pleasure, for the surprise, will lead to face-to-face consultations in 30% of cases, **in addition to his usual work**. TE does not reduce the number of consultations. Over time, there are more and more correspondents, questions are more and more relevant, and photos are of better quality.

3. Delegations of tasks, new modes of organisation: it is vital to increase the skills of GPs, **with TE playing an indirect role.** It is also important to broaden the skills of medical assistants and look into the possibility of APNs in dermatology.

Questions :

Prof. Dompmartin: "If you don't mind my asking, how do you finance your staff? Do you receive financial assistance from the ARS?" This raises the question of responsibility towards nurses.

Answer: Dr. Ribeaudeau does not receive assistance; he works in sector 2 and supervises all the procedures carried out by his State-certified nurses.

Dr. Marie-Sophie Gautier (private dermatologist in Joinville-le-Pont) - Specialist care team (ESS) and TE

The ESS was introduced in 2019 = a group of healthcare professionals with specialist doctors, in a given area, with a healthcare project that must focus on a medical discipline, based on private medical practice, enabling a social security structure package to be fulfilled.

It is an exercise **without walls, based on voluntary participation, working together, with shared resources, which distinguishes it from specialist groups.**

Purpose: dedicated funding from January 2025, which will structure the provision of secondary care in geographical and financial terms.

In pratice: This is an association, with the need to draft a letter of intent for the ARS and CPAM (it is possible to get help in drafting the letter and the project). Funding is still being negotiated, on average €80,000 per year, with the need to **have at least 10% of the region's specialists.**

Brittany ESS: DermatoBreizh, 39 dermatologists out of 114, more than 800 TE services/month with 40% face-to-face consultations. Training for general practitioners in tumour screening and dermoscopy.

Corsica ESS: 10 out of 13 dermatologists, four pharmacies equipped with dermatoscopy, remote MDT meetings in oncodermatology with Nice University Hospital.

In the Paris region: ESSDV-IDF, for dermatologists and general practitioners. TE for skin cancer.

Fewer TE services than in Brittany, 1/3 skin cancer, median response within two hours, avoids 48% of consultations.

Then chronic inflammatory diseases.

Has a remote MDT meeting system run by private dermatologists, providing geographical independence for both oncology and biotherapies, with hospital and private practitioners.

Dr. Jiliana Monnier (Dermatology Department, Marseille University Hospital) - AI for skin cancer detection

Increasingly, AI applications are using a labelled database that produces very good results. For example, there is an AI application that studied over 12,000 melanoma/naevus images in 2019 in three centres in Barcelona, Vienna and London that were checked against new images and tested.

Dr Monnier reiterated the importance of "clean" data (no hair, equivalent light, black dermoscope edges).

For new indications, it is important to give the AI full body maps (atypical naevus syndrome).

In Marseille: VECTRA system: automated melanoma screening centre with full-body images, which saves a considerable amount of time.

Australia is in the process of setting up a national VECTRA system.

Prospects: Pseudo dermoscopy booth in Spain for the whole body, DERMAP avatar on smartphones, you take photos and transfer to a computer, free.

Discussion:

Many FMC participants have raised the question of liability in the event of an error, and it is clear that the manufacturer who sells the AI-enabled solution, whether VECTRA or other, disclaims all responsibility, which will fall to the dermatologist, which is why there are still no driverless cars, even though they can drive on their own. One FMC participant stressed the importance of joining forces in the coming years to combat this shortage and the growing responsibility and pressure on dermatologists.

Dr. Nathaline Lalanne (private dermatologist in Bordeaux)

Dr. Lalanne raised the issue of systematic annual screening for our patients.

On the one hand, it would appear that screening does **not reduce** melanoma-specific mortality, but patients who detect themselves do so at a late stage and are therefore more likely to be metastatic. Practices are saturated, and so the role of TE is emerging.

In her opinion, it is important to develop **teledermoscopy (TD)** and ask ourselves whether AI can help us to sort things out? We are tempted to say yes; example of an AI application that gives seven diagnoses; it is effective for screening but not for management. It is very sensitive, so it does not miss melanomas, but it does remove 50% of dysplastic naevi and it is quite time-consuming.

According to one study, one in three melanomas are missed without a full clinical examination.

Prospects:

This brings us back to ESSs centred on the dermatologist, TE and TD, with the need for a **protocol**.

A recent study showed that post-consultation self-education by an APN prevented 50% of subsequent consultations.

Dr. Emmanuel Mahé (Dermotology Department, Argenteuil Hospital).

According to Dr Mahé, we need to re-invent medicine using TE.

Things have been speeding up over the past year. Initially, Argenteuil Hospital was using ORTIF (a TE network created by the IDF ARS for the prison system); now it mainly uses OMNIDOC.

Argenteuil Hospital offers three TE modules: paediatric dermatology, general dermatology and wounds & healing (the 3rd of which is available to State-certified nurses).

It has started a TC for elite swimmers in the Paris region, which allows it to see things it was not familiar with before (no examples), as well as TE with French-speaking African paediatricians via Omnidoc which **requires adaptation, sees leprosy, monkeypox – the same care is not available there.**

Prospects:

Publications on feedback and innovative practical work (psoriasis and Curacné monitoring), particularly for patients who are visiting from far away, need to be **accepted** by doctors and patients. How is acne monitored? Psoriasis in children? It would be interesting to come to an agreement, with monitoring templates and photos.

Responding to surveys via working groups and patient associations.

Role in national infectious disease surveillance: in 2022, a large number of streptococcal impetigo cases were reported, and a few months later, the health authorities declared an epidemic of streptococcus. The same happened in 2023 with parvovirus B19.

JDP 2024 announcement: **National cohort for monitoring skin-tropic infections through the Omnidoc TE network** (200,000 TE services/year, excluding cancer), with a focus on viral, tropical, scabies and fungal (ringworm) infections.

Practical paediatric dermatology

Coordinator: Prof. Frédéric Cambazard (Dermatology Department, Saint-Etienne University Hospital)

Dr. Alice Phan (Dermatology Department, Lyon University Hosital)

Case 1 involved a four-day-old girl, with no problems during pregnancy or childbirth, who went to A&E on the 4th day for rapidly spreading erythema of the legs with skin detachment. The mother had applied shea butter following onset of the rash.

Pruritus could not be assessed at D4 – **from three months onwards**. Dressings were **painful.**

There was no fever, the haemodynamic status was good, and the bullae and underlying erythema were extensive.

Another eight-month-old child, bullous detachment on the back, extremely itchy => **bullous mastocytosis** (often from birth, diffuse involvement, **infiltrated skin, yellowish colour, pruritus)**, best seen when cold, infiltrated skin folds, high tryptasemia, easy flushing.

Returning to the first case, there was no mastocytosis because there was no infiltrate.

In another case, a newborn was presenting with bullae on healthy skin => **hereditary epidermolysis bullosa**, to be considered from birth with pathological scarring and milia. **Caution: sometimes misleading, nothing at birth**.

Another case of very superficial detachment on the buttocks and face with skin fragility = **autosomal dominant epidermolytic ichthyosis**, fairly rapid scarring, giving way to yellow palmarplantar hyperkeratosis.

Another case: an 11-day-old baby, erythema and localised superficial detachment, not just on fragile areas with a **hypopyon => bullous impetigo**.

Another case: an 18-day-old newborn, first lesion on the **inguinal fold**, then very rapid spread, very slight erythema, very superficial detachment => **Staphylococcal scalded skin syndrome (SSSS)**, haematogenous diffusion of exfoliative toxins, **peri-orificial streaks and crusts and involvement of the folds**. Generally painful, non-conventional fever, **no mucosal involvement.**

Another case: a four-year-old girl, rapidly confluent bullous papular lesions with **mucosal involvement => toxic epidermal necrolysis**

So they went back to questioning the mother and ended up with a diagnosis of burns, t**he mother had given a bath that was too hot, clues: knees and skin folds respected.**

Dr. Eve Puzenat (Dermatology Department, Besançon University Hospital)

The first case was a two-month-old infant with a rash on the **buttocks** consisting of erythematous **pustular** lesions.

Think of irritant nappy dermatitis, sometimes in the form of vesiculo-pustular dermatitis, due to the skin coming into contact with faeces and urine (article in Les Annales de Dermatologie). In this condition, the deep area of the fold is respected; there are erythematous forms and papulo-erosive forms of Sevestre and Jacquet dermatitis, which take weeks to heal.

Granuloma gluteale infantum is another form of irritant nappy dermatitis, historically linked to fluoride-containing topical corticosteroids associated with secondary Candida infection or diarrhoea and, more recently, to washable nappies. This is a **multifactorial** dermatitis that will take time to regress.

Genital herpes, a differential diagnosis, is more localised and not pustular (vesicular); **the data from the literature are reassuring; in fact, transmission is usually indirect.**

Nappy psoriasis persists for as long as the baby is wearing nappies, so warn parents that the rash will recur.

Case 2 was a newborn child with a rash on the left lower limb, involving the scrotum, reticulated lesions with anaemic borders, with an ante-natal diagnosis of a single kidney, and sexual ambiguity with hypospadias and a micropenis.

Precursor lesion of segmental systematised infantile haemangioma, warning sign to evoke SACRAL syndrome, consider it in the presence of systematised IH with genito-renal anomalies.

Need for spinal cord MRI and abdominal and pelvic ultrasound.

Klippel Trenaunay syndrome combines systematised flat angiomas + limb asymmetry + venous malformation; the triad is not always present at birth.

We now have a genetic classification linked to PIK3R1, PIK3CA and KRAS mosaicisms.

Sometimes, isolated monomelic flat angiomas are observed; after puberty these children are no longer at risk of hypergrowth.

In the case of localised cutis marmorata telangiectatica congenita (CMTC), there is no need for further examination, but **attention must sometimes** be paid to what is said to the parents.

Case 3 involved a small asymptomatic congenital millimetre pubic opening in a two-year-old girl. Once you have seen one, you can diagnose it at a glance.

This was a **congenital prepubic sinus**, diagnosed clinically, with a small opening between the umbilicus and the lower pubis, **always on the midline**. Treatment is always surgical, as the fistula extends to the bone, and there is therefore a **risk of secondary infection**. This sinus is always blind, never with associated anomalies, and no additional examination is necessary.

Case 4 was a two-month-old girl with an erosive lesion covered by a thin translucent membrane with an epidermal collar between the anus and the vulval fourchette.

This was a **congenital perineal groove**. At birth, the skin is aplastic, although over time it is covered with epidermis, but the collar is still present. More likely to affect girls, the pathophysiology is unknown, most often isolated but sometimes anogenital and exceptionally renal anomalies, the outcome is favourable within 12 months.

Another case: an **acquired soft, raised fungated anal** lesion in a four-month-old girl = **pyramidal protrusion, always at 12 o'clock**, affects girls more than boys under one year of age, the pathophysiology is uncertain, pushing effort? Abstention is the rule.

A **constipated** four-year-old girl with a soft lesion **not** in the area of the protrusion **=> skin tag.** Skin tags occur in older children and not in the 12 o'clock position; more common in **patients with Crohn's disease; look for diarrhoea and abdominal pain.**

Think about BUYING A TOILET STOOL to treat constipation.

Prof. Frederic Cambazard (University Hospital - Saint Etienne)

There was an eight- to nine-year-old child with *"spots everywhere",* a history of AD, with pruritus and pain => profusion of molluscum, the eczema had to go, so use of DIPROSONE then MOLUSDERM, treated three at a time, healing but lots of scars. According to Prof Cambazard, the occasional application of MOLUSDERM stimulates immunity.

Case 2: a six-year-old girl, parents were first cousins, **sudden damage to all 20 nails, with thickening, inflammation around the nails, etc.** Two months later, patches of hair loss suggesting alopecia areata, usually AA nails show **trachyonychia, streaks and pits** (also seen in twenty-nail disease), general corticosteroid therapy and methotrexate can be tried.

Case 3: a 13-year-old patient, nodular acne, had been on cyclins then Curacné with flare-up, so had corticosteroid therapy with recurrence when cortisone doses were reduced.

Prof Cambazard suggested 200 mg of TOLEXINE for 15 days, then 100 mg, then reassessment after two months. The face was better, but the back was a mess, BUT an axillary nodule had appeared.

Put on a TNF inhibitor and checked after one month, no more active lesions.

Key messages from this case: inflammatory acne can be the gateway to Verneuil's disease, general corticosteroid therapy is of no use because it recurs on discontinuation, always start isotretinoin at a low dose and give cyclins beforehand to calm the inflammation.

Case 4: a three-year-old boy, diagnosed with hand, foot and mouth disease with dystrophy of eight out of 10 toenails... Nail samples showed **T. Rubrum**. He first suggested Amycor cream and filing the damaged area as children's nails grow back quickly unlike those of the elderly (easier extension in the elderly, childhood onychomycosis very often only distal) but compliance problem over one year of treatment, so ended up with oral terbinafine.

Case 5: a 16-year-old girl, eczema since the age of two, allergic background, highly lichenified lesions on both ankles and wrists. BETESIL plaster or corticosteroid therapy twice a day can be offered. Transient improvement and relapse, IgE 31,000 and Hyper Eo 1 G/L. Put on DUPILUMAB.

Café au lait spots: when should a genodermatosis be suspected?

Coordinator: Prof. Smail Hadj-Rabia (Paediatric Dermatology Department, Necker, Paris)

Dr. Laura Fertitta (Dermatology Department, APHP - Henri Mondor)

In children

- Café au lait spots (CLS): rounded oval pigmented macule with a linear or segmental arrangement, **uniformly pigmented**, varying in colour from light to dark brown, ubiquitous.

- CLSs are common in children, affecting 2 to 3% of newborns and 1/3 of young children.

In practice:

- Typical or atypical CLS (poorly demarcated, irregular borders, inhomogeneous pigmentation not suggestive of NF1)

- Personal and family history

- If there are one or two segmental lesions, this most often corresponds to mosaics.

> Three CLSs: monitoring

> Six CLSs **> 5 mm**: consider NF1 but also McCune-Albright syndrome

Differential diagnoses: freckles, lentigines, congenital naevus, mastocytosis, Becker's hamartoma.

It is **important to count them**, look for associated dermatological signs, extra-dermatological manifestations (RCSP, dysmorphia, cardiovascular, OPH) and family history.

Mccune-Albright syndrome results in large, irregular segmental CLSs delimited by the midline and features the **clinical triad of CLSs**, **fibrous bone dysplasia and precocious puberty**. The post-zygotic mutation affects the GNAS gene.

1. **Clinical case n°1**

Case 1 was an eight-year old child with four CLSs, lentigines and a strong family history of cancer => **constitutional mismatch repair deficiency (CMMRD) syndrome, here a caricature, sometimes there is no history of cancer.** MMR gene mutation, CLSs are not systematic, between two and 10, atypical with irregular borders, and sometimes hypopigmented lesions. The median age at first cancer is 7.5 years, requiring digestive, haematological and neurological surveillance, and then in women, gynaecological surveillance from the age of 20.

Genodermatoses linked to the RAS activation pathway include cardiofaciocutaneous syndrome (CFCS), LEOPARD/NOONAN syndrome and neurofibromatoses. CLSs are the common feature.

Cardiofaciocutaneous syndrome: autosomal dominant, BRAF > MAP2K1 > MAP2K2 > KRAS giving characteristic facial dysmorphia, short stature, pigmentation anomalies, and more rarely pulmonary artery stenosis, septal defects, and hypertrophic cardiomyopathy.

LEOPARD syndrome: autosomal dominant, **PTPN11** mutation, more common than CFCS. Produces **L**entigines, conduction abnormalities (**E**cg), **O**cular hypertelorism, **P**ulmonary artery stenosis, genital **A**nomalies, growth **R**etardation and **D**eafness.

NF1: **20,000 patients in France, prevalence 1/4,000,** autosomal dominant, 50% sporadic forms (no family history).

2. **Clinical case n°2**

Case 2 involved a six-year-old child referred for diffuse cutaneous mastocytosis, who in reality also had NF1 – beware of the train that can hide another.

Penetrance is 97% at eight years, so there must be more than six CLSs.

Presentation of an article showing the probability of NF1 as a function of the number of CLSs:

After two and a half years and more than six CLSs in the last year => more than 80% chance of NF1.

3. **Clinical case n°3**

Case 3 was a 10-month-old child with more than 10 CLSs, unidentified bright objects (UBOs) on MRI and a plexiform neurofibroma of the hemiface, which led to the diagnosis of NF1.

Yasunari nodules = choroidal hamartomas, an essential diagnostic sign recognised since 2021.

It is important to refer to an OPH who is used to recognising the signs of NF1.

Anaemic nevus predominates on the sternum and appears on rubbing. Juvenile xanthogranuloma (JXG) is also more frequent in NF1 children.

Also look for long-bone dysplasia, scoliosis and growth retardation.

Optic pathway gliomas affect 15% of NF1 children, **but only a small proportion need to be treated. We suggest OPH monitoring every six months until the age of six, then annually until the age of 18.**

4. **Clinical case N°4**

Case 4: a 10-year-old boy, more than 20 CLSs, language delay, brain MRI and normal OPH examination. The mother had the same spots. If the mother **had NF1, she would NOT ONLY have CLSs**. Was actually **LEGIUS** syndrome.

Legius syndrome: SPRED 1 mutation, NF1-like syndrome with only CLSs, **no tumour development.**

Segmental NF is a mosaic form based on a post-zygotic mutation, sometimes affecting several segments.

Fanconi anaemia: to be suspected, CLSs + hypopigmented macules, **gradual bone marrow failure, thumb abnormalities in 50% of cases.**

In conclusion, in children, importance of the clinical features and examination of the PARENTS; often the diagnostic criteria are not obvious with the presence of CLSs alone.

In adults

Case 1: An adult patient with CLSs and neurofibromas, **can be diagnosed with NF1**.

Case 2: CLSs + lentigines: NF1 or Legius, molecular analysis, for Legius: **CLSs are always bilateral (as opposed to NF, where they are sometimes segmental).**

CLSs **tend to disappear** in adults.

In NF1 adults, over 95% cutaneous neurofibromas, highly polymorphic, size, shape, colour, sometimes acne-like.

Revision of diagnostic criteria in 2021:

In the absence of affected parents: Two criteria required from among CLSs / bilateral lentigines in folds / at least two NFs / glioma / Yasunari or Lisch nodules / sphenoid dysplasia or anterolateral curvature of the tibia or pseudoarthrosis of the long bones / molecular criterion.

If a parent is affected: only one criterion is needed

In the case of an isolated CLS in an adult, monitoring / molecular analysis / referral to reference centre; the **question of transmission** arises. **NF1 adults who are doing very well can have a child with severe NF1.**

Case 3: Segmental mosaic NF1, **large CLS with neurofibromas on top but next to very healthy areas.** Transmission depends on the mutant allele fraction, but there is no exact figure. Very difficult for genetic counselling < 50% transmission. T**he child will have complete NF1 in the event of transmission**. Skin biopsies are often required for genetic analysis for confirmation and genetic counselling, as only 10% of blood cells carry the variant.

Need for a partnership with the GP. Annual blood pressure and OPH monitoring, **watch for warning signs**: deterioration of general condition, appearance of tumour, pain, neurological deficit.

Proposal of a phenotype at risk of cancer:

More than 10 palpable subcutaneous (those that cannot be seen) NFs, Mondor suggests a PET MRI or whole-body MRI scan.

Increased risk of breast cancer, start screening from the age of 30 and involve the gynaecologist.

Prof. Smail Hadj-Rabia (Paediatric Dermatology Department, APHP Necker) - genetic testing

Genetic counselling **does not require molecular analysis**.

Molecular results take **at least three or four months** to arrive, so parents should be warned of the wait.

He emphasised Yasunari nodules, visible from the age of three, which have helped to unblock uncertain diagnoses.

Molecular analysis is a biological examination, with clinical-biological comparison (parents), governed by the law (does the analysis **change the way the child is managed**) and with the informed consent **of both parents**. Important **warning: a negative result does not rule out the diagnosis**. We start with a panel of NF1 genes, followed by mRNA analysis and whole genome sequencing.

Molecular analysis is necessary before a new pregnancy.

When it is not possible to clinically distinguish between NF1 and Legius, see if there is a parental project; if the parents are doing well, there is a < 1% risk of transmitting the disease to a new child, but they may request a PND, we must look for a molecular accident in the child, which we will try to find in the amniotic fluid or chorionic villi.

Class 5 variant = certain of the causality **(not the severity)** of the mutation, class 4 probably pathogenic, class 3 significance unknown, class 2 probably benign, class 1 benign variant.

In some cases, no anomalies are detected, and mRNA or whole genome sequencing can be requested to confirm or rule out pathogenicity.

In conclusion: NF1 is a genetic susceptibility to systemic diseases. In the majority of cases in children, it is not serious except for developmental delays.

Vascular abnormalities: quiz

Coordinator: Dr Olivia Boccara (Dermatology Department, APHP, Necker Hospital)

Infantile haemangioma

Speaker : Dr. Christine Léauté-Labrèze (Dermatology Department, Bordeaux University Hospital)

This year we are celebrating 10 years of MA for Hemangiol.

Propranolol is **not** a cardioselective beta-blocker. It was synthesised in 1962 and won the Nobel Prize for Sir James W Black; it is on the World Health Organization's list of essential medicines and **crosses the blood-brain barrier**.

It is the only treatment to have MA for infantile haemangiomas (IHs); its efficacy is dose-dependent; regression was total or almost total in 60% of cases in the pivotal study, and it is more effective if started **before the age of three months** (80% total or almost total regression).

A second study following the pivotal study on severe IHs showed a 75.6% success rate between six and 12 months of treatment, with an average treatment duration of 7.6 months.

On the other hand, recolouring was observed in 25% of cases when the treatment was stopped, and half (12.5%) of children were retreated. The risk factors for recurrence are: segmental IH, or with a deep component.

The average length of treatment for S1 and S2 segmental haemangiomas is 12 months; it is 24 months for the S3 "beard" segment.

With regard to safety data: fewer ulcers, no increased risk of unexpected infant death syndrome, risk of hypoglycaemia higher **during/at the end of treatment**, < 0.5% bradycardia, **no evidence of a long-term effect.**

Digital blood glucose tests are useless when introducing solid foods, but patient education is important, may end up forgetting and baby has fewer and fewer meals.

A 2024 study compared 1,000 children treated with propranolol vs 10,000 not treated: **no signal/difference.**

PHACES syndrome and Hemangiol: **treatment should be started as soon as possible,** at the usual therapeutic dose and for **at least 12 months.**

In conclusion:

10% of IHs are treated, i.e. one in 200 infants

Early treatment before the age of three months is recommended

Hypoglycaemia needs to be monitored over time and parents need to be educated

Other haemangiomas

Speaker : Dr. Olivia Boccara (Dermatology Department, APHP, Necker Hospital)

A congenital haemangioma **is not an infantile haemangioma (RICH and NICH**); they are present from birth and do not worsen thereafter, either remaining stable or regressing. This is a different condition.

The clinical picture and chronology enable the condition to be identified, **not imaging**.

Congenital haemangiomas **do not express GLUT-1**, which is a **lymphatic** marker.

Verrucous haemangiomas or verrucous venous malformations are generally present at birth, with a subcutaneous component that may increase over time. They are GLUT-1 positive and do not regress spontaneously or with propranolol. They may be flat at birth and it is difficult to predict thickening, which is sometimes non-keratotic but jagged. However, their colour tends towards dark purple, with dermoscopy showing small red blood cells without telangiectasia.

There is no pathophysiological explanation for the expression of GLUT-1 + / D2-40 - as this is not a lymphatic lesion. It also has a MAP3K3 mutation.

Interventional surgery can be proposed but with frequent recurrences +/- pulsed dye laser but many sessions.

IH is **not an intramuscular (IM) lesion**. So IM haemangioma (IMH) is not an IH.

IMH is a fast-flow vascular lesion of intramuscular topography. It has the same MRI characteristics as IH, but not the same kinetics or consistency. A French study of 66 IMHs showed they had the same mutation as AVMs.

Lastly, cavernous (for dilation) haemangioma is an old term for **venous malformations**. There is pain from stasis and varicose veins. A more infiltrative form known as fibroadipose vascular anomaly (**FAVA**) leads to fibrosis, with slower flow and a somatic mutation in PIK3CA.

Prof. Annabel Maruani (Dermatology Department, Tours University Hospital)

Case 1: a 62-year-old right-handed cleaning lady; the back of her right hand had taken on a bluish appearance a few years earlier but this was now increasing, not very well defined, a little warm to the touch, rather soft, strictly painless, not pulsating, did not swell when she lowered her hand.

Self-induced ecchymosis could be suspected, as she was indifferent to the lesion, or calcified haematoma from work-related trauma, or venous malformation given calcification on imaging.

The lesion grew over the years, but MRI showed **tissue** malformation **(not classic)** but calcification **(venous)**, and arteriography showed arterial flow and venous return.

The Tours team continued to think about VM**, but still not AVM**.

It suggested a biopsy: proliferation of **CD34+ spindle cells (endothelial cells, haematopoietic stem cells, dermatofibrosarcoma protuberans)** with BRAF fusion transcript => only one case in the literature; they are discussing radiotherapy, a MEK inhibitor or abstention (patient's wish).

Key message: if there is atypia on imaging, do not hesitate to perform a **biopsy**.

Case 2: a **17-month-old girl** born full-term, with vacuum delivery, **with appearance at one month** of **firm** swelling of the skull, maternal & child protection services suspected a cephalohaematoma (subperiosteal) or serosanguineous bump (supraperiosteal) => false, **must appear very soon after delivery,** or an IH.

CT scan: homogeneous, slightly vascularised tissue lesion (not an AVM) with no calcification.

They suspected a benign or malignant tumour and therefore performed **excision: CD34- spindle cells, abundant collagen and proliferation of myofibroblasts, CTNNB1 pathogenic variant = benign desmoid tumour**, in rare cases associated with adenomatous polyposis coli (APC gene mutation). Monitoring because **relapse occurs in 70% of cases.**

Differential diagnosis: RICH, which may appear very tumorous but which starts to disappear from D15.

Case 3: Omnidoc opinion (Ouagadougou) of a right temporal lesion which appeared at eight months of age and which was increasing: malignant polylobed angiosarcoma => death.

In the event of vacuum delivery, a cephalohaematoma should be suspected if there is a very bluish appearance.

A congenital lesion that increases in size at six weeks: infantile fibrosarcoma.

In conclusion: it is important to know whether or not the lesion **was present at birth (CH, KHE, benign/malignant tumour, cephalohaematoma, VM).**

Clinical cases of adolescent dermatoses

Coordinator: Dr. Claire Abasq-Thomas (Dermatology Department, Brest University Hospital)

Dr. Claire Abasq-Thomas (Dermatology Department, Brest University Hospital)

Adolescent dermatoses include chronic inflammatory dermatoses, hormonal (acne, androgenetic alopecia), psychological and environmental (infectious diseases, contact dermatitis) disturbances and autoimmune diseases.

Case 1

A 15-year-old girl, tingling of the lower limbs when standing for the past year, with a cyanotic appearance and then anaemic macules => **BASCULE syndrome** = *blue-white-red* vasomotor dermatosis. **Succession of acrocyanosis then anaemic Bier macules followed by a pseudo-urticarial rash.** In a series of 17 adolescents, median age 12, it typically occurred during a hot shower, in a standing position, with **extremities affected in 100% of cases**. The pathophysiology was based on a dysfunction of the autonomic nervous system in 10 patients; 3/3 responded to BBs, and H1 antagonists did not work. There was no major impact on quality of life. Prof BESSIS recommends measuring heart rate lying down and standing up (orthostatic TC if > 30 bpm).

Case 2

A 16-year-old girl referred for bluish nodules that had appeared two years previously, no digestive problems, no anaemia. Sensitive. Normal CBC. The father had similar lesions => **glomuvenous malformations or glomangiomas** = an autosomal dominant subtype of venous malformation. Treated by excision or laser but does not work very well. Appears in childhood or congenital, sometimes hyperkeratotic, multifocal, **painful on palpation**.

Can be confused with Bean or blue rubber bleb naevus syndrome = multiple venous malformations of the skin AND digestive system (bleeding, anaemia).

Case 3

A 10-year-old girl referred for acne lesions on the cheeks, on closer look were **angiofibromas;** An article from 2021 notes the possible histological confusion between acne scars on the nose and chin and angiofibromas in the Hispanic population. No other skin lesions, no epilepsy, normal brain MRI, renal angiomyolipomas, **mosaic mutation which explained the incomplete muted picture.**

TSC is inherited by autosomal dominance and occurs de novo in 70% of cases, with complete penetrance and variable expressivity. The cutaneous signs are achromic macules, angiofibromas, pigmented fibrous patches on the forehead/face, shagreen patches, nail angiofibromas (Koenen's tumours) and mucosal angiofibromas (macroglossia, gingival hypertrophy).

Case 4

A very athletic 12-year-old girl referred for blue nipples in the context of repeated sprains (from sport). Palpation was normal. Type "blue nipple" in PubMed => **retroareolar** cyst in prepubertal/pubertal girls following obstruction of Montgomery glands, spontaneous resolution.

Dr. Audrey Lasek (Dermatology Department, Saint Vincent de Paul Hospital - Lille)

Case 1

A 12-year-old girl with a **rapidly progressing,** slightly erythematous, scaly **infiltrated** plaque, a rabbit, history of atopy. The mother sent photos with daily changes, sometimes more shiny and inflammatory and then cracking. Had already had topical antifungal treatment.

Skin biopsy: CD4+ lymphocytic inflammatory infiltrate and mucinosis => **folliculotropic MF.**

Normal biological work-up, clonality positive on the skin.

Treated with moderate-potency topical corticosteroids with recurrences (3) followed by remission.

Law of series: a second 12-year-old adolescent with a history of atopy and a tumour of the right eyebrow that had been progressing for seven months and was not itchy, same histology, improved with topical corticosteroids.

Case 2

Polymorphous papular lesions that progressed to hypopigmentation of all four limbs; pityriasis lichenoides was suspected but skin biopsy = lymphomatoid papulosis type D, negative extension work-up, three months of methotrexate with complete remission.

A similar case with more necrotic lesions occurring in flare-ups, controlled by topical corticosteroids.

Lymphomatoid papulosis: 100% survival rate at five and 15 years, but must be monitored because lymphomas and leukaemia can develop. The delay in diagnosis is 1.3 years, with 25% misdiagnosis and pruritus in only 20% of cases.

Case 3

Omnidoc opinion for a **12-year-old** child with facial psoriasis treated with Dermoval 4/day, **no Auspitz sign**. Involvement of the face, décolleté and limbs, with erythematous scaly ring lesions. On further questioning, he had two rabbits and three cats, with scalp involvement => **dermatophytosis/ringworm**.

Case 4

Omnidoc second opinion for a thoracic lesion with rapid palpebral extension and necrotic progression; Augmentin did not work. The mother spontaneously mentioned stagnant water following flooding (they lived in Saint Omer). **Spontaneous regression of lesions**, four cats at home => cowpox virus +.

Poxvirus family: causes molluscum, smallpox and monkeypox. Cowpox virus is a zoonosis **(cats and rodents)**, with an incubation period of seven days, direct transmission but no inter-human transmission, notifiable disease, no specific treatment, favourable progression.

Dr. Hélène Aubert (Dermatology Department, Nantes University Hospital)

Case 1

A 14-year-old girl with no particular medical history. Sudden deterioration of general condition with arthralgia, diffuse erythema, vesiculobullous lesions and hyperthermia, for which she was admitted to hospital. **DNA antibodies were + => bullous lupus with renal involvement**. A second case with Evans syndrome: immunological thrombocytopenic purpura and haemolytic anaemia followed a few months later by **lupus**. In 10-20% of cases, lupus begins in childhood, usually between the ages of 11 and 12; it **is rare before the age of five** (think of monogenic lupus). Look for a family history, consanguinity, syndromic disease, short stature in boys. A three-year-old girl with lupus-like lesions, frostbite, anti-SSA autoantibodies + and repeated infections = **monogenic lupus**.

Treatment with Plaquenil 6.5 mg/kg/day reimbursed compound (unscored tablets), **OPH monitoring.**

In paediatric lupus, general signs are frequent.

Case 2

A 16-year-old girl with facial lesions for one year, put on a cyclin in the hypothesis of acne without improvement. **Skin biopsy confirmed the diagnosis of chronic lupus**, ANAs 1/320 with no specificity, tried topical corticosteroids but depigmentation, put on Plaquenil and tacrolimus. For **follow-up: study by Fredeau et al (JAAD 2022) who studied the risk of systematisation of chronic discoid lupus => phototype V-VI, ANA+, onset < 25 years.**

Case 3

Cervical and **abdominal** hyperpigmentation with no previous history, doing very well, -1.3 SD and the whole family was quite tall. Did not go away with an alcohol-soaked compress. Biopsy = **acanthosis nigricans**, normal metabolic work-up, **BW + 3 SD, height < 2 SD around target height,** X-rays: hypochondroplasia => **FGFR 3 gene syndrome** causing hypochondroplasia and acanthosis nigricans; variable phenotype.

Case 4

A 15-year-old boy, psoriasis since age 9, history in mother, itching, psychological impact, Soriatane for one year then local treatments; systemic treatment discussed. **No recommendation for systemic psoriasis in children**.

MTX 10 mg/m^2 can be given, same work-up as for adults **except HIV,** chest X-ray, vaccinations, chickenpox, pneumococcus (PREVENAR 20 single dose), biological monitoring identical to adults and take on Friday evening.

Case 5

Maxime, age 16, rash for 15 days in a stressful context (exam), did not scratch much, history of psoriasis in father. **Early syphilis, which looks like PR/psoriasis/lichen planus but is not itchy.**

Case 6

Melvin, age 10, learning difficulties, array-CGH: RASA 1 deletion, referred by geneticist to find out whether skin lesions were compatible with the deletion.

Clinically: capillary malformation of the lower lip, telangiectasias on the hands and thorax, **centimetric macular lesions with a halo of vasoconstriction**, similar clinical phenotype in the father => **capillary malformation/arteriovenous malformation syndrome (CM/AVM)** of autosomal dominant transmission.

Beware of Osler-Weber-Rendu disease (differential diagnosis). Publication by Olivia Boccara JAAD 2021, 68 CM-AVMs with brain MRI: one AVM, two hamartomas, three arterial aneurysms and three VMs, so not 30% as described in the original study. **No recommendation on systematic MRI or if warning sign on a case-by-case basis in CM-AVM.**

Case 7

14 years old, AD flare-up at Christmas time, his cat had died, school break, did not want to be hospitalised because of Christmas, suggested the introduction of dupilumab, with onset of conjunctivitis at M4 => lost to follow-up. The child was **psychologically worrying** and ran away from the consultation. **Beware of the association between AD and depression**. Hospitalised to **assess suicidal risk and introduce an antidepressant**.

Case 8

Pathomimia in an adolescent boarder with a difficult family background and polymorphous lesions of different ages. Do not go in head-on, reassure the patient that the is no infection or organic cause, joint consultation with a psychiatrist.

Prof. Anne Claire Burnstein (Dermatology Department, Nancy University Hospital)

Case 1

A 12-year-old girl with a BMI of 31 kg/m^2, menarche for one year with typical Verneuil's disease, tried several things until adalimumab, limited results, then **secukinumab, ditto**. Laser hair removal improved it.

Paediatric hidradenitis suppurativa: predominantly female, family history, less smoking, **advised not to start**. No co-morbidities but must be vigilant.

Case 2

Contact eczema caused by epoxy resins (GERDA 2024) with pruritic erythematous oedema of the face, available over the counter.

Case 3

A teenage girl referred for alopecia areata, history in mother, boyfriend also had alopecia areata, proposed bolus of solumedrol because of recent onset, then at age 16 proposed baricitinib (off-label or for associated eczema) with rapid favourable outcome at six months.

LITFULO MA from the age of 12 on a conentional prescription, reimbursed at 30% (caution: mutual insurance).

Case 4

Urticarial pseudo rash + polyuria-polydipsia syndrome treated with general corticosteroid therapy, then a few weeks later weight loss => **keto rash** with T1DM and context of ketoacidosis.

Context of occurrence of prurigo pigmentosa or keto rash: pregnancy, anorexia, prolonged fasting, ketogenic diet, bariatric surgery, diabetes.

Case 5

A case of scabies with nail involvement (6% in children), resulting in onycholysis and pachyonychia. Use of keratolytics for nail damage.

Case 6

An abdominal and genital rash in a 12-year-old boy with scabies with visible furrows. Pruritus affecting the abdomen only. **Topical treatment treats pruritus more rapidly.**

In conclusion : get the parents out of the room to create a climate of trust (syphilis, pathomimia), undress gradually, do not force it, address all the teenager's questions.

Practical flash 3 - Hair

Coordinators: Dr. Bruno Matard, Dr. Philippe Assouly (Sabouraud Centre, Paris), Prof. Henri Montaudié (Dermatology Department, Nice University Hospital)

Folliculitis decalvans

Dr. Bruno Matard (Sabouraud Centre, Paris)

Folliculitis decalvans (FD) is a poorly understood condition for which there is no known cure and for which isotretinoin does not work. This is a sometimes very severe disease, which **almost exclusively** affects **the scalp**, **particularly in men and never before puberty**. In the majority of cases, there is no family history or associated disease.

Histologically, it is destructive folliculitis with TH17+ neutrophils.

Clinically, it is inflammatory scarring alopecia characterised by **pustules that develop into a crust and folliculitis in tufts with more than six hairs per follicular unit**. The differential diagnoses are lupus and lichen planopilaris (LPP).

There is a lichen form with few symptoms, and treated FD cases show a partial reduction in inflammation.

There is a five-stage IGA score to measure the efficacy of TNF inhibitors.

Some authors have assumed there is a spectrum between lichen planus and folliculitis decalvans, but the Bordeaux team disproved this by studying the inflammasomes of the two diseases, which differed.

This is because the infiltrate in FD is neutrophilic (short-lived) and clinical inflammation therefore rapidly regresses. Sometimes the infiltrate is lymphoplasmacytic **but without interface dermatitis (present in lichen).**

A new histological sign: **epidermal thickening** suggesting FD rather than LPP.

FD is highly polymorphic, so beware of ringworm and do not hesitate to take fungal samples.

We find *S. Aureus, GNB,* transient and opportunistic flora. The barrier anomaly is therefore **unknown**.

How Dr. Matard explains FD to patients: "It is a skin barrier disease that leads to invasion of the hair follicles by opportunistic flora, which is not regulated with antibiotic therapy".

Management remains poorly codified, with no curative treatment. The aim is to **maintain the absence of inflammation without general treatment** via monitoring through photos with the patient's smartphone.

First-line treatment: double-dose tetracyclines for three weeks, then single dose for several months. Second-line treatment: rifampicin + clindamycin for 10 weeks, then first-line treatment or Bactrim 400 to 800 mg/day for several months. Locally, topical corticosteroids or Kenacort can be used, but dapsone is not used and zinc does not work. PDT is theoretically interesting => example of textile PDT in Lille. Few data on biotherapies, but one study in Bordeaux on 11 patients treated with TNF inhibitors: 50% of patients stabilised.

The study on the efficacy of isotretinoin did not lead to the conclusion that it is the best treatment, as the title states. Transplants are not recommended because of the theoretical risk of recurrence. Dr Matard presented two personal cases of transplantation with recurrence at 3.5 and 4.5 years, but with satisfied patients. **In conclusion, prolonged remissions do exist.**

Practical paediatric cases

Prof. Sébastien Barbarot (Dermatology Department, Nantes University Hospital)

Case 1

Acquired scalp pustules in a child with alopecia => ringworm (kerion type)

Beware of two misleading situations:

- Abscessed kerion **placed in the hands of surgeons**, which becomes scarred
- **"Impetigo-like crusting without alopecia"**

A study by Stéphanie Mallet on ringworm in children weighing less than 10 kg showed that local treatment was **insufficient** and that terbinafine was very well tolerated in this population.

He showed us the new algorithm with withdrawal of ITRACONAZOLE from the market (see FMC 13).

As a reminder, for microsporic ringworm in children weighing under 20 kg = those who cannot swallow a tablet and must take 50 mg of an unscored tablet => TERBINAFINE ½ tab for six weeks.

Ringworm does not require shaving and there is no need to stay home from school (transmission by close contact).

Case 2

A six-month-old infant with scaly dermatitis and erosive papules on the scalp + erosive areas in the neck and buttock folds => Langerhans cell histiocytosis, a disease not to be missed in the presence of *erosive bipolar dermatitis*, CD1a + immunostaining, 100% of systemic forms involve the scalp.

Case 3

Congenital diffuse alopecia, small hairs that **broke very quickly**, **follicular keratosis**, was growing well, moniliform dysmorphia => **monilethrix, great phenotypic variability.**

Case 4

A two-year-old child with an angioma that bled occasionally in the **medial occipital** region, millimetre ulceration and firm nodule **without skin aplasia** => **SUSPECT DYSRAPHISM here, dermal sinus with abnormal closure of the cranial cavity.** Sometimes a blind fistula, sometimes extending into the brain stem. It is to be differentiated from aplasia cutis congenita of the scalp with the hair collar sign, **MRI if collar sign, large size or hair malformation**.

Case 5

Five-years old, frizzy, tangled hair, autosomal recessive uncombable hair syndrome, **TCHH PADI 3 gene, no syndromic form**, isolated hair anomaly.

Not to be confused with woolly hair syndrome + PPK, risk of arrhythmogenic cardiomyopathy => **Naxos-Carvajal syndrome.**

Case 6

Woolly hair naevus linked to mosaic mutations in HRAS or KRAS, associated cancer risk debated.

Prof Barbarot recalled the results of ritlecitinib in severe alopecia areata: 25% achieve a SALT score < 20. He reminded us of the importance of corticosteroid therapy in acute alopecia areata.

Importance of anatomical and clinical comparison

Prof. Kolivras (Dermato-pathologist in Brussels)

The same histological image will have different interpretations depending on the clinical situation, hence the importance of dialogue between the clinician and the pathologist.

First example with alopecia areata

Peribulbar mononuclear infiltrate with follicular miniaturisation and increase in the % of catagen and telogen hairs.

- Case of secondary syphilis mimicking alopecia areata.
- Psoriasis and lupus erythematosus can also mimic it.

Do not hesitate to request ANAs, syphilis serology and mycological samples.

Second example with lichen planopilaris and its differential diagnoses in anatomical pathology

Pseudopelade of Brocq is a form of lichen planopilaris

Folliculitis decalvans can mimic LPP **but** polytrichia visible in anatomical pathology

Central centrifugal cicatricial alopecia gives **spectacle-like** images = **fusion of two hair shafts**.

Syphilis in anatomical pathology is rich in plasma cells

Post-transplant (context, questioning)

Androgenetic alopecia: follicular miniaturisation and increase in the % of catagen and telogen hairs

Hormone-induced alopecia due to breast cancer or taxane chemotherapy

Morphea and systemic amyloidosis.

Androgenetic alopecia

Dr. Pascal Reygagne (Sabouraud Centre, Paris)

Rather than talking **about** androgenetic alopecia (AGA), we need to take into account age, psychological profile and the fact that treatment is chronic. Do not forget **Hamilton's score/stage.**

If dermoscopy reveals more than 20% fine hair, treatment with minoxidil 5% is started. Otherwise, we reassure the patient that there is nothing wrong to date and we will see them again in one year.

A 14-year-old child with a **frizzy** appearance **=> poor prognosis**, put on minoxidil 2% then 5% if not sufficient (off-label).

Another case of a young adult with moderate AGA, either finasteride 3 mg three times a week + minoxidil 2% or finasteride 1 mg daily + minoxidil 5%.

=> Start one treatment at a time to find out which one does what.

For finasteride, the ANSM leaflet must be given to patients with the side effects, **3% sexual dysfunction**. Question about depression? Doubtful.

It is important to take before-and-after photos because patients do not realise how effective treatments are.

The next case was a 30-year-old man with moderate or more severe AGA. We can combine minoxidil + finasteride then switch to oral minoxidil 2.5 mg per day but take weight, pulse and blood pressure +/- dutasteride 1 mg/week on Sundays. As a last resort, micrograft hair transplant.

Discuss and discourage PRP, LED, MESOTHERAPY and MICRONEEDLING – does not work.

Topical finasteride is on its way (see study results).

Avoid transplanting too early... from the age of 28-30.

The following case involved Hamilton stage VI and VII AGA => classic wig.

In women, the diagnosis is less straightforward, usually with a **preserved frontal border** and a Christmas tree pattern. Look for menstrual disorders, hirsutism, late-onset acne and hyperseborrhoea, and carry out a work-up if there are any associated signs, then seek an endocrinological opinion if there are abnormalities.

If the work-up is abnormal: anti-androgen treatment, contraception with anti-acne pills such as Triafemi/Diane 35/Jasmine + spironolactone 100 mg/day; minoxidil can be added if not sufficient.

If oily hair: contraception and spironolactone, minoxidil 2% then 5% at six months, spironolactone can be increased to a maximum of 150 mg/day and minoxidil changed to oral 0.5 or 1 mg max as a reimbursed compound.

Offer spray make-up or tinted powder, typically for special occasions.

Warning: minoxidil can cause oedema of the extremities, hypotension and hypertrichosis. In the event of allergy to topical minoxidil, it can be given orally. There is no increased risk of breast cancer in women taking spironolactone.

In conclusion: early treatment is essential. The earlier we start treatment, the earlier we can delay the onset of baldness.

Alopecia areata

Philippe Assouly (Sabouraud Centre, Paris)

Alopecia areata occurs two out of three times before the age of 30 and is chronic and capricious.

Quality of life is not proportional to the surface area affected and it improves over time.

It grows back better at the front than at the back, and long-standing alopecia areata has a poor prognosis because it reduces the size of the bulb over time.

It would appear that SALT 10 is better for assessing patients' quality of life.

Topical corticosteroids should always be offered as lotions as they carry fewer systemic risks than other galenic formulations. Patients can be offered the option of applying the lotion to one side only if they have already tried this treatment previously, for a minimum of three months.

After kenacort, the hair starts to grow after three weeks. 0.5 ml of kenacort are pumped in, followed by 2 ml of NaCl 0.9 isotonic serum. There is no eye risk if kenacort is applied to the eyebrows.

Phototherapy works and can relieve an acute situation, as can systemic corticosteroid therapy, which remains the most effective treatment for unblocking a situation or blocking a flare-up.

The original baricitinib study in 1,200 adult patients with a SALT score > 50 had a SALT score < 20 as the primary endpoint. A SALT score < 10 was achieved in 20% of patients **but was suspensive**. Quality of life was not evaluated. We lose half of the patients by halving the dose.

The original Litfulo study with 50 mg per day in 718 patients, including adolescents aged > 12 years, showed 40% SALT 20 at 12 months, 30% SALT 10 at 12 months and 22% SALT 10 for alopecia totalis at 12 months.

There are no topical forms of JAK inhibitors in alopecia areata.

Lastly, the initial combination of a JAK inhibitor and systemic corticosteroid therapy may be of interest.

Terbinafine-resistant dermatophytoses: what can be done?

According to the Hot Topics presentation. Terbinafine-resistant dermatophytoses: new challenges.

Dr. Alicia Moreno-Sabater

The emergence of terbinafine **resistance in dermatophytoses has become an almost daily problem**. Nearly 8% of trichophyton strains are resistant to terbinafine, mainly Trichophyton indotineae. In fact, almost 68% of strains analysed in French mycology laboratories are resistant.

Therapeutic management is as follows:

1. Start at 250 mg/day
2. If treatment fails, increase the dose to 500 mg/day (i.e. two tablets)
3. If (2) fails, switch to itraconazole 100 mg/day
4. If (3) fails, increase the dose of itraconazole to 200-400 mg/day

It is important to remember that **resistance to itraconazole** is currently very low. Of course, some reference centre laboratories can carry out antifungal susceptibility tests. The list is available on the website of the French Society of Medical Mycology. However, the response time is long (one month).

The second explanation for a possible failure of itraconazole is low serum levels, particularly when taken at 100 mg/day. Absorption of itraconazole is increased when taken after a heavy meal or with a drink such as Coca-Cola.

The relapse rate for *T. indotinae* infections is very high, whatever the form of the disease, whether widespread or localised. There is no way of predicting the occurrence of a relapse.

Lastly, as an alternative to itraconazole, there is voriconazole with various protocols (200 mg/day for two to four weeks or 400 mg x two days, etc.). Fluconazole is not effective, and the combination of itraconazole and terbinafine does little to help.

Balanitis, balanitis and... balanitis

Donzel C, *et al*. Balanoposthites associées à *Streptococcus agalactiae* : étude rétrospective multicentrique de 37 patients.

Donzel C, *et al*. Balanite à *Streptococcus agalactiae* secondaire à un traitement par inhibiteur de SGLT2 description d'une première série de 4 cas.

Salle R, *et al*. Balanite chronique associée à *Corynebacterium glucuronolyticum* : description de 3 premiers cas.

Salle R, *et al*. Balanite syphilitique de Follmann : une manifestation rare de syphilis primaire.

Several posters presented different infectious causes of balanitis.

A retrospective study of 37 patients with balanitis and/or posthitis and a positive bacteriological sample for Group B Streptococcus showed that the pathogenicity of the bacterium was established in 64% of cases, with improvement in symptoms following topical antibiotic treatment (mupirocin or fusidic acid) and/or amoxicillin. All patients were uncircumcised, with more frequent dribbling. These results are consistent with the maceration risk factors for balanitis.

Tubular glucose reuptake inhibitors are a new class of oral hypoglycaemic agents marketed in 2015. Three drugs are already approved in Europe (dapagliflozin, canagliflozin, empagliflozin). **SGLT2 inhibitors are known to cause candidal balanitis.** Dermatologists at Ambroise-Paré Hospital compiled four cases of patients aged between 56 and 83, all taking SGLT inhibitors (but one was non-diabetic), with bacterial balanitis caused by *Streptococcus agalactiae,* presenting as erythematous, fissuring balanoposthitis, with partial phimosis that progressed favourably during treatment with oral antibiotics (amoxicillin 3 g/day for five days and/or mupirocin twice a day for seven days). Mycological samples were negative. Treatment with SGLT inhibitors had been in place for several months to one year.

Streptococcus agalactiae should not be considered as purely saprophytic at the balanopreputial level. It is a potential pathogen in patients with balanitis and as such should be investigated and treated if necessary.

Three cases of balanitis caused by *Corynebacterium glucuronolyticum* have been described in young men (aged 30-40) who had sex with men with chronic signs of balanitis (erythema, smegma, scales) and a negative STI and mycological work-up. The role of this gram-negative bacterium has not yet been established (cause or secondary infection of pre-existing balanitis). The outcome is favourable with standard treatment for non-specific balanitis and, if necessary, amoxicillin.

Syphilitic balanitis of Follmann is a clinical variant of primary syphilis that should not be overlooked due to the resurgence of syphilis. This is a superficial diffuse erosive balanitis teeming with Treponema pallidum, corresponding to an equivalent of syphilitic chancre. It is extensive and painful and can therefore be misleading (herpes, fixed pigmented erythema or caustic balanitis). In addition, syphilis serology may initially be negative. It is therefore important to carry out an interrogation and repeat the syphilis serology test or take a PCR sample if available.

Hailey-Hailey disease: towards new therapeutic possibilities?

Ly S, *et al*. Maladie de Hailey-Hailey ano-vulvaire : efficacité du ruxolitinib topique.

Walls B, *et al*. Efficacité de l'aprémilast dans un cas de maladie de Hailey-Hailey.

Oillarburu, *et al*. Efficacité du dupilumab dans la maladie de Hailey-Hailey.

Decaestecker, *et al*. Efficacité du dupilumab dans la maladie de Hailey-Hailey : 2 cas.

Hailey-Hailey disease (HHD) is an autosomal dominant genodermatosis linked to a mutation in the ATP2C1 gene encoding an ATPase; it is responsible for pruritic, painful and malodorous erosive plaques in the skin folds and perianal area. It has a major impact on the quality of life of patients. Treatment options are varied but are often poorly tolerated and disappointing (topical corticosteroids, topical calcineurin inhibitors, isotretinoin, naltrexone, surgery).

Ly *et al*. reported the very rapid efficacy of ruxolitinib, a JAK inhibitor, in the treatment of localised anovulvar HHD. Functional symptoms were controlled within 48 hours and clinical lesions within one month. The patient continues to apply the maintenance treatment several times a week.

Walls *et al*. from Bichat Hospital used apremilast 30 mg twice daily, after an initial titration period, in a 26-year-old patient with refractory disease involving the genital, axilla and inguinal regions. The treatment was well tolerated. A major improvement in symptoms was reported after two weeks of treatment. After one month of treatment, only post-inflammatory pigmentation was observed. The response was maintained at the five-month reassessment.

The Toulouse team reported the results of a French multicentre study that included 17 patients with severe HHD mainly treated with dupilumab at the dose recommended for atopic dermatitis with some efficacy. At the same time, the Rouen team reported the efficacy of dupilumab in two forms of adult HHD within one month.

Skin barrier abnormalities could lead to inflammation of the Th2 pathway involving IL-4 and IL-13; this would explain the efficacy of dupilumab and inhibitors of the JAK-STAT pathway.

But Doctor, it was a natural product!

Bernier C, *et al.* Phytophotodermatose grave induite par une tisane

Boulhilat A, *et al.* Erythème pigmenté fixe bulleux généralisé à l'ashwagandha (superfood)

We often tend to forget that **natural products, especially plants, can cause skin reactions, in the form of drug-induced dermatitis.**

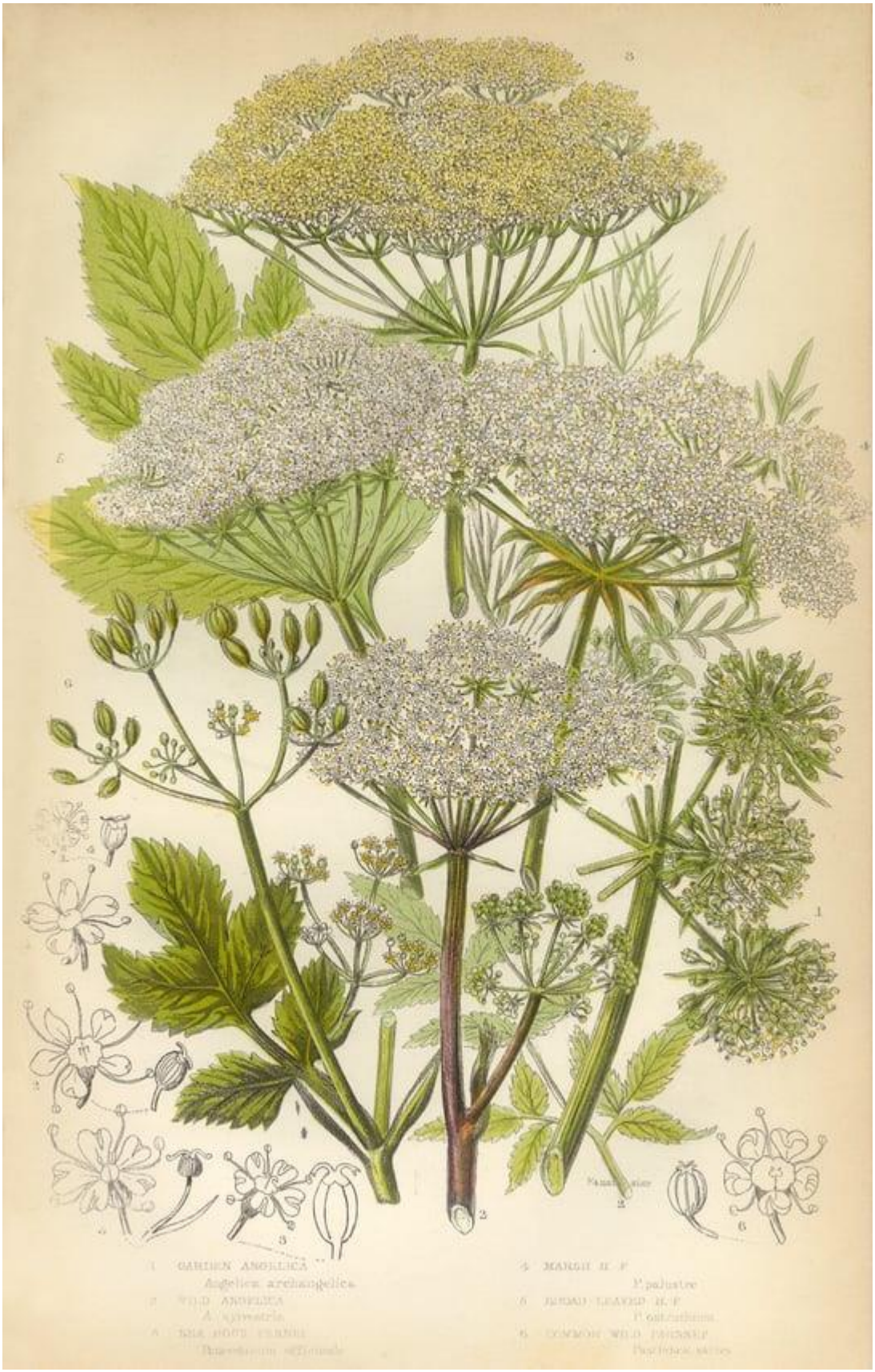

Figure 1. *Angelica archangelica* – garden angelica

Here we have the bitter experience of a 60-year-old woman who developed a **serious case of phytophotodermatosis with second-degree burns and hospitalisation, after drinking an herbal tea containing garden angelica (*Angelica archangelica)***, a member of the Apiaceae family (Fig.1). The patient had consumed this herbal tea the day before, in early September, and dozed off on a beach in Brittany for two hours the following day. On waking from her nap, she developed diffuse erythema limited to the exposed areas, which progressed to painful skin detachment over the following days, requiring hospitalisation in a burns unit. The presence of furocoumarins explains this phytophotodermatosis picture.

Figure 2. *Withania somnifera* – ashwagandha or Indian ginseng

Dermatologists in Rabat reported a case of **fixed bullous erythema covering 80% of the skin's surface four days after taking ashwagandha**, confirmed by a reintroduction test. Ashwagandha or Indian ginseng *(Withania somnifera*) is a plant in the Solanaceae family. It is part of the traditional ayurvedic pharmacopoeia in India, and has been gaining popularity in the West through social media since COVID-19. Available in capsule or powder form, it is thought to act on stress-related diseases.

In short, stick to chamomile and verbena...

Management of atopic dermatitis in 2024: French recommendations issued by the Atopic Eczema Research Group and the Centre for Evidence in Dermatology

Based on the European recommendations published in 2022, French recommendations have been drawn up by the Atopic Eczema Research Group (GREAT) and the Centre for Evidence in Dermatology (CPD), adapted for France and the specific features of our healthcare system, and updated in the light of the new therapies available. Here is a summary of some of these new recommendations.

Comprehensive management

- Consider comprehensive management, in particular an assessment of the impact on family life, and offer psychological care to patients and their close family and friends if severe AD is having repercussions for daily life
- Corticophobia should be discussed at the 1st consultation, to limit any fears that may arise
- Patient education programmes are suggested

Contributory factors

- Avoid irritating clothing (wool, synthetics)
- Avoid smoking
- Water softeners are not effective
- Do not limit physical activity
- In the absence of an immediate reaction, there is no need for allergy testing or a systematic diet as part of the management of atopic dermatitis
- It is advisable to start diversifying the diet of babies from the age of four months, with no preventive elimination diet (expert opinion)
- Allergy testing
- if symptoms appear within two hours of eating food, in the form of urticaria, angioedema, flushing or digestive problems, an immediate IgE-mediated reaction must be ruled out ⇒ in which case allergy testing should be requested, including specific IgE assays, skin tests and a reintroduction test
- if moderate to severe AD is resistant to well-administered treatment, with suspected worsening after food intake
- There are no recommendations concerning the use of probiotics, food supplements, antioxidants or vitamin D
- Borage oil and evening primrose oil are not effective in the treatment of AD

Local treatments

- Strong topical CSs for the body, moderate topical CSs for the face, once a day until the non-regressing plaques diminish
- Use the fingertip unit rule
- In the event of frequent flare-ups, proactive treatment is recommended after remission of the flare-up, twice a week on the areas previously affected

- The use of topical calcineurin inhibitors in adults and children:
- is recommended in the event of repeated use, and on skin areas at risk of atrophy with topical CSs: face including eyelids, skin folds, ano-genital region
- in the event of an acute flare-up, initial treatment with a topical CS is suggested before treatment with calcineurin inhibitors to improve tolerance
- after remission, proactive treatment twice a week on the affected areas is recommended
- Use emollients every day, preferring a balm in winter and a lotion in summer
- Antiseptics are not recommended
- Take short, warm baths or showers, using cleansing products that are free of allergens and irritants, with a pH between 5 and 6

Systemic treatments

- If AD is poorly controlled (SCORAD > 50 and/or major impact on quality of life)
- Or if the patient is unable to undergo suitable local treatment
- Or if the amount of topical CSs required to control the disease over the long term is > 4 tubes of 30 g per month for adults
- Cyclosporin

In adult patients, as a short course of treatment, to get over a hurdle, and therefore for a limited period of time

High dose (4 to 5 mg/kg) from the outset

BP and kidney function monitoring

- Methotrexate (off-label indication)

15-20 mg/week, preferably via the SC route

Liver monitoring

- Biotherapies

Dupilumab (IL-4 and IL-13 inhibitor) is recommended for adults and children aged six months and over with AD requiring systemic treatment

Lebrikizumab (IL-13 inhibitor, SC, 500 mg S0 and S2, then 250 mg every fortnight) and tralokinumab (IL-13 inhibitor, SC, 600 mg S0 then 300 mg every fortnight) are

recommended for adults and adolescents aged 12 and over with AD requiring systemic treatment

These treatments do not require any pre-therapeutic assessment or biological monitoring

The risk of conjunctivitis must be known

- JAK inhibitors (JAKi)

Abrocitinib (100 or 200 mg/day) is recommended for adults and adolescents aged 12 and over with AD requiring systemic treatment

Baricitinib (2 or 4 mg/day) is recommended for adults and children aged two and over with AD requiring systemic treatment

Upadacitinib (15 or 30 mg/day) is recommended for adults and adolescents aged 12 and over with AD requiring systemic treatment

Initial hospital prescription

Pre-therapeutic assessment and biological monitoring (CBC, liver and kidney function tests, lipid profile)

Patients > 65 years of age or who smoke or have smoked for a long time or have other risk factors for cardiovascular disease or malignant tumours can be treated with JAKi only if there is no alternative and at half the dose, and if they are informed of the risks.

Caution is required in patients with venous thromboembolic risk factors.

The HAS recommends the use of biotherapies and JAKi as second-line treatment if cyclosporin has failed, is not tolerated or is contraindicated.

When should treatment be stopped in the event of complete remission?

- Cessation as soon as remission is achieved with cyclosporin: maximum treatment duration of one year
- After 16 weeks (four months) of treatment with lebrikizumab and tralokinumab: space out the injections every four weeks
- After six months of remission, 1) for methotrexate, space out in stages until the minimum effective dose is reached; 2) for dupilumab, space out the injections; and 3) for JAKi, reduce to half-doses

Cutaneous manifestations of Mycoplasma pneumoniae infections

According to the oral presentation of Guyomard V, *et al*. Cutaneous manifestations associated with *Mycoplasma pneumoniae* infection in adults in France during the 2023 epidemic: a multicentre observational study

And according to the posters of Nadé R, *et al*. Necrotic livedo revealing cold agglutinin disease secondary to lung disease caused by mycoplasma, and Couissi I, *et al*. Sneddon-Wilkinson subcorneal pustular dermatosis associated with a mycoplasma infection

***Mycoplasma pneumoniae* (MyPn) is a Gram-negative bacterium responsible for respiratory infections. It affects children aged between five and 17 as well as young adults and occurs worldwide, particularly in temperate climates. An upsurge in cases of *M. pneumoniae* has been observed worldwide since autumn 2023.** This increase can be explained by the low level of viral and bacterial exposure in previous years, due to the preventive measures taken during the COVID-19 pandemic. **MyPn infections can be accompanied by mucocutaneous manifestations (10-25% of cases), for which a dermatologist may be asked to give an opinion.**

A national observational study (MYCADO study) in autumn/winter 2023-2024 showed that 4.5% of patients with a confirmed MyPn infection had cutaneous manifestations (n = 60/1,309). Of the 60 cases included, the most common, in order of frequency, were erythema multiforme (EM) (n = 33, 55%), maculopapular exanthema (n = 13, 22%), urticaria (n = 7, 12%) and others (purpura, impetigo, n = 7). Among the EM cases, the cutaneous manifestation was considered severe in 88% of patients, with mucosal involvement in 97% – oral (94%), ocular (79%) and genital (39%) – and involvement of at least two sites in 79% of cases. Systemic corticosteroid therapy was required in a third of cases.

Patients with dermatological involvement were significantly younger, less frequently obese, more frequently diagnosed with *M. pneumoniae* infection by serology vs. PCR alone, and had fewer associated severe respiratory forms (less use of oxygen therapy), but greater use of systemic corticosteroid therapy.

In this cohort, dermatological forms affected younger patients and were dominated by EM, as expected, with single or multiple mucosal involvement.

In addition, there can be clinical polymorphism, including the possibility of extensive necrotic livedo due to cold agglutinins induced by a MyPn infection, which progresses favourably with macrolide, acetylsalicylic acid and local corticosteroid therapy, or the occurrence of Sneddon-Wilkinson syndrome four days after a MyPn infection, which also progresses favourably with antibiotic therapy and local treatment. Dapsone can also be offered if necessary.

Yellow urticaria is not serious, while blue urticaria is serious

According to the communication of Prof Aurélie Du-Thanh, What's new in clinical dermatology?

And the communication of Louise Gouvion. Not everything in life is rosy (Top 12 interns)

Yellow urticaria is a rarely described dermatological manifestation reflecting the accumulation of conjugated bilirubin in skin tissue. It has the same symptoms as urticaria but is distinguished by its yellow colour. The pathophysiological mechanism of yellow urticaria in liver damage is thought to be the accumulation of conjugated bilirubin in skin tissue due to vasodilation and increased capillary permeability caused by urticaria. However, the yellow colour has no prognostic value for the urticaria itself.

Acute bluish urticaria is one of the clinical manifestations of immediate IgE-mediated hypersensitivity reactions to dye, especially patent blue, a dye used in sentinel node detection. It is thought to affect 1 to 3% of patients. In clinical terms, there may be bluish eyelid oedema, acute bluish or blue-green urticaria, or even anaphylactic reactions that may lead to cardio-respiratory arrest. The reaction occurs within 30 minutes of the injection. Skin tests can be carried out (prick, tuberculin) to confirm the diagnosis and recommend definitive exclusion of the product, but these tests are difficult to interpret and are not always reliable. **The alternative is sentinel node detection using an isotope technique combined with indocyanine green.**

Efficacy of GLP-1 agonists in hidradenitis suppurativa: an anti-inflammatory mechanism?

According to the communication of Gouvrion L, *et al*. Therapeutic effect of GLP-1 analogues on hidradenitis suppurativa. Retrospective series of 66 patients.

The placing on the market of glucagon-like peptide-1 (GLP-1) agonists (GLP1a) represented a therapeutic revolution for patients with type 2 diabetes and obesity. Weight reduction is induced both by action on the brain (reduced appetite, increased sensation of satiety) and on the stomach (slower gastric emptying). Two drugs currently have MA for obesity (semaglutide and liraglutide). In addition, these drugs may have anti-inflammatory properties, which are currently being studied. **Given that obesity is a major comorbidity associated with hidradenitis suppurativa (HS), GLP-1a could be of therapeutic interest in HS via weight reduction or direct anti-inflammatory action.**

A French multicentre retrospective study of 66 HS patients exposed to dulaglutide, liraglutide or semaglutide assessed the impact on the course and severity of HS, as well as the specific efficacy of each drug, by comparing patients on dulaglutide with those on liraglutide or semaglutide.

Of the 66 patients included, 48 were on semaglutide, 13 on dulaglutide and five on liraglutide. Seventy-one percent of patients were also on metformin, and 47% were not on any "specific" treatment for HS.

A total of 53% (n=35) improved their disease (HS-PGA score) six months after initiation of treatment and 61.5% (n=40) at maximum follow-up time (median follow-up for 18.5 months). Sixty percent of patients also had fewer relapses between three and six months. Interestingly, the only factor significantly associated (univariate analysis) with improvement in HS was the use of semaglutide or liraglutide compared with dulaglutide. Severity and weight loss in kg had no impact. According to a multivariate analysis, once again only GLP-1a treatment was associated with an improvement in HS.

Given the lack of effect of weight loss after six months, GLP-1a could be effective because of their anti-inflammatory properties, in particular their modulatory action on pro-inflammatory adipokines, as well as on cytokines such as TNF-alpha and IL-23, IL-17, etc. In addition, they reduce local dysbiosis and over-activation of the toll-like receptors involved in the innate immune response.

GLP-1a could play a role in the management of HS, but robust comparative prospective studies will need to be undertaken to position this treatment.

Dermatological habits in the Caribbean and voluntary depigmentation among adolescent girls in Africa

According to the poster of Boulay *et al*. Common problems and the impact of lifestyle habits in the Afro-Caribbean population of Martinique, Guadeloupe and French Guiana: epidemiological study of 749 patients

According to the poster of Seudjip Nono *et al*. Voluntary skin depigmentation among adolescents in rural areas

According to the poster of Soumé *et al*. Voluntary depigmentation in schools in Conakry: clinical aspects and attitudes of pupils

Seven hundred forty-nine people in Guadeloupe, Martinique and French Guiana (phototype III or higher) responded to an online questionnaire disseminated via social media and informational posters in pharmacies in 2024. The aim was to identify the **habits and frequent skin problems of the Caribbean and South American population**.

In order of frequency, skin problems included hyperpigmentation, acne, dermatosis papulosa nigra, dandruff, atopy and traction alopecia.

The frequency of shampooing varied from once a week or once a fortnight to once a month (40%, 38% and 22%). Hair was natural in 76% of cases; if it was braided, the braids were redone every two to three weeks (38%) or once a month (29%). Straightening was thermal (55%) or chemical (24%). Fifty-five percent said they did both.

A third of those questioned applied sun protection every day.

Only 23% were convinced that the dermatologists in the study region were trained in pigmented skin.

Voluntary skin depigmentation is a real scourge in Sub-Saharan Africa and constitutes a major health problem. Two studies on this practice among children and teenagers from Africa (Congo and Guinea) were presented at JDP 2024.

A study of children and teenagers (aged 10-19) in a rural area of Congo unfortunately showed that depigmentation is still common. It was practised by **almost two out of 10 people questioned (17%), mainly young teenage girls aged between 14 and 16, with hydroquinone being used in 71.5% of cases**. The classic motivations were found: having fair skin, beautiful skin; the influence of the environment through imitation and the media or magazines.

In Conakry, the capital of Guinea, a study of registered users (median age 17) found **similar motivations**. **The depigmenting treatments used were topical corticosteroids (60%), followed by mercury derivatives (28%) and fruit acids (11%)**. Hydroquinone was not mentioned. **More worrying was the fact that in eight out of 10 cases, these products came from supermarkets, and in less than one in two cases from pharmacies**. **Complications reported from the misuse of depigmenting products included dyschromia (54%), acne (31%) and stretch marks (18%).**

Voluntary depigmentation is a reality in schools in Sub-Saharan Africa. It is started early by teenage girls, with skin complications already observed. **Greater awareness is needed to put an end to this, in order to protect the health of teenagers.**

CANCER PREDISPOSITION FOR DERMATOLOGISTS: WHEN SHOULD IT BE CONSIDERED? HOW CAN IT BE PROVEN? HOW SHOULD IT BE MANAGED?

Speakers: Frédéric Caux, Eve Maubec, Virginie Bubien

Reed's syndrome or familial cutaneous and uterine leiomyomatosis

It should be considered in the event of **leiomyomas** (painful pinkish papules) around the age of 30 (10-77) and when large, multiple uterine fibroids develop around the same age (18-53).

It is an autosomal dominant disease with high penetrance.

The diagnosis is confirmed by genetic analysis of the FH gene.

The prognosis varies between and within families.

The risk of **kidney cancer** is estimated at 15%, often with aggressive potential, and appears around the age of 40. Multiple leiomyosarcomas as well as atypical uterine leiomyosarcomas have been described.

Appropriate management: - gynaecological consultation + uterine ultrasound from the age of 20, then annually

- Renal MRI from the age of eight, then annually.

Birt-Hogg-Dubé syndrome

It should be considered in the presence of **fibrofolliculoma/trichodiscoma** (pathognomonic whitish papules) on the face, neck and upper trunk from the age of 20. Lung cysts occur in 90% of cases, and pneumothoraces in 25% of cases before the age of 40.

It is an autosomal dominant disease with high penetrance.

The risk of **kidney cancer** is estimated at 20%, often before the age of 50, and is bilateral and multifocal. In 45% of cases, there are also kidney cysts.

Appropriate management:

- Genetic counselling (heterozygous mutation of the FLCN gene)
- Treatment of skin tumours by destruction (surgery, laser, electrocoagulation)
- Annual dermatological monitoring (increased risk of melanoma)
- Lung assessment with thoracic CT scan
- Prevention: stop smoking, avoid hyperpressure (diving, music)
- Screening for kidney tumours using imaging (MRI and ultrasound) from the age of 18 in the USA

Muir-Torre syndrome

It should be considered in the presence of **profuse sebaceous lesions** (sebaceous adenomas, sebaceomas, sebaceous carcinomas, keratoacanthomas and BCCs with sebaceous differentiation) outside the cheeks and forehead and in young patients (under the age of 50).

It is one of the manifestations of **Lynch syndrome**, which is a predisposition to multiple, early, often not very aggressive visceral cancers (colon 50%-60%, endometrium 30%-40%, stomach 10%, ovaries 8%, urothelium 5%, lymphomas, central nervous system).

It is caused by autosomal dominant mutations in DNA mismatch repair (MMR) genes (MSH2, MSH6, MLH1, PMS2). Paraclinical diagnosis is based on analyses of tumours (PCR, MMR protein immunostaining) and blood (search for germline mutations in MMR genes).

Differential diagnoses:

- Drug-induced eruptive forms (cyclosporine, azathioprine, sirolimus)
- Familial adenomatous polyposis with MUTYH mutation: sebaceous lesions (adenomas, hyperplasias, carcinomas). This is an autosomal dominant disease.

Appropriate management:

- Genetic counselling with screening of relatives
- Surgical excision if necessary, use of prolonged retinoids
- Cancer screening
    - colon: colonoscopy every two years from the age of 25, or five years before the age at onset of the earliest familial cancer
    - endometrium: ultrasound + smear +/- hysterography every year from the age of 30, discuss prophylactic hysterectomy.
    - other: mammography, urology and upper endoscopy every two years.

Cowden syndrome

It should be considered in the presence of **storiform collagenoma**.

Other mucocutaneous signs are observed in more than 95% of cases and begin around the age of 20: papillomatosis of the gums, tongue and lips, trichilemmomas on the face and storiform fibroma.

Extracutaneous signs include:

- Thyroid manifestations (>70%) such as heteromultinodular goitre with adenomas, follicular carcinomas (15%).
- Digestive manifestations (>70%) such as oesophageal glycogenic acanthosis, extensive polyposis with varied histologies and colonic adenocarcinomas.
- Mammary manifestations (>65%) such as fibrocystic disease, adenofibromas and adenocarcinomas (60%).
- Genitourinary manifestations (>40%) such as ovarian cysts and cancers, multiple early uterine fibroids and renal carcinomas.
- Neurological manifestations (40%) such as macrocephaly (58 cm in women and 60 cm in men), epilepsy, meningiomas, vascular anomalies and Lhermitte-Duclos disease.

In 2006, the criteria for diagnosis were classified as

- Pathognomonic criteria: facial trichilemmomas, acrokeratoses, facial papules, oral papillomatosis, Lhermite-Duclos disease.
- Major criteria: breast cancer, thyroid cancer, endometrial cancer and macrocephaly.
- Minor criteria: breast hamartomas, thyroid, digestive tract, lipomas, fibroids, mental retardation, uterine fibroids, kidney tumours.

To raise the possibility of a diagnosis:

- in an individual, there must be more than six facial papules with three trichilemmomas or facial papules + oral papillomatosis or acrokeratoses + oral papillomatosis or more than six palmoplantar keratoses or two major criteria or one major criterion and three minor criteria or four minor criteria.
- in a family where the diagnosis has already been made, there must be one pathognomonic criterion, one major criterion or two minor criteria.

Genetic analysis shows the PTEN mutation in 80% of cases.

These patients often have a social cognition disorder.

Gorlin syndrome

The diagnosis should be considered if there are two major criteria or one major criterion and two minor criteria.

The major criteria are:

- > Two BCCs before the age of 20
- Maxillary cysts
- Cerebral calcifications
- More than three palmar or plantar pits
- Fused or flared bifid ribs
- Family history (first-degree relative) of Gorlin syndrome

The minor criteria are:

- Macrocephaly
- Congenital malformations (cleft lip/palate, frontal bossing, hypertelorism)
- Other skeletal abnormalities (scoliosis, *pectus excavatum*, syndactyly, Sprengel deformity)

- Radiological bone abnormalities (*sella turcica*, vertebrae, hands and feet, etc.)
- Ovarian fibroids
- Medulloblastoma

The genes most often involved are PTCH1, SUFU (risk of medulloblastoma) and PTCH2.

Susceptibilty to melanoma

Melanoma is a multifactorial disease.

When should an oncogenetic consultation be requested?

Patients should be referred if there is/are:

- At least two invasive cutaneous melanomas in the same family branch (5 to 10%)
- At least two invasive cutaneous melanomas in the same subject before the age of 75 (9%)
- An invasive cutaneous melanoma and another cancer in the same subject (melanoma of the eye, pancreatic cancer, kidney cancer, central nervous system cancer or mesothelioma)
- An invasive melanoma in a young subject (<40 years).

The French recommendations for monitoring patients are as follows:

- Patients with CKDN2A, CDK4 or BAP1 mutations: six-monthly clinical monitoring for life, with total body photography once a year and, if possible, video microscopy at M0, M3 and M12.
- Patients without mutations should be monitored every six months if there are associated risk factors (large number of naevi, atypical naevi, high sun exposure, phototype I or II, MC1R or MITF variants). Other annual examinations.

In terms of screening for other cancers associated with melanoma, it is important to investigate:

- Pancreatic cancer in CDKN2A-mutation carriers: MRI + endoscopic ultrasound
- Eye melanoma in BAP1-mutation carriers: Annual fundus examination from the age of 11
- Kidney cancer in BAP1-mutation carriers; annual MRI from the age of 30

There are other tumours associated with BAP1. These include BAPoma, mesothelioma, BCC, hepatocellular carcinoma, cholangiocarcinoma and meningioma.

HOT TOPICS

TREATMENT STRATEGY FOR BULLOUS PEMPHIGOID (BP)

Speaker: Marina Alexandre

- Option 1: Strong local corticosteroid (CS) therapy 30 to 40 g/day to be continued for 15 days after clinical control, then gradually tapered off. If the patient weighs less than 45 kg, limit topical CSs to 20 mg.

- Option 2: Strong local CS + methotrexate (MTX) regimen

Strong local CS 10 to 30 g/day with tapering over three months + MTX from the outset (10 – 12.5 mg/week) for nine months.

Recommended if your patient is in good health and cognitive condition. This regimen avoids the need for cortisone, and there seems to be less relapse.

- Option 3: Medium-potency local CS + doxycycline 200 mg/day

Use if CI for CS therapy or if regimen 1 is not possible

- Option 4: General CS regimen, 0.5 mg/kg/day, with gradual tapering off over four to six months.

If these options fail, **omalizumab** can be offered. Seventy-seven percent complete remission with control within 10 days and a relatively low number of relapses. This treatment seems beneficial if there is an increase in circulating IgE (>70%), positive ELISA testing for IgE BP180-NC16A antibodies (in 58-77% of BP cases) and IgE deposits by DIF (in 18-41% of cases).

The other hope for BP is **dupilumab**, because BP patients have high TH2 polarisation and overexpress IL-4 and IL-13. In the studies, dupilumab was combined with local CSs. Complete remission was achieved in 35-44% of patients, with median disease control within 14 days and with good clinical tolerance.

Contact allergy news

Speaker: Angèle Soria

The most common contact allergens continue to be metals (nickel, cobalt and chromium), fragrances (the main source being cosmetics) and methylisothiazolinone (MIT).

New allergens include:

- Essential oils

- Vegetable oils (nigella, hemp, tamanu). Patients should be questioned directly, as they do not always think to mention this, and their oil should be tested.

- Isothiazolinones. This is especially true in Europe, as MIT has been banned from no-rinse cosmetics in France. In France, they are mainly found in cleaning products, leather and leather care products.

- Acrylates and derivatives. They are highly allergenic in their liquid form, but not at all in their polymerised (hard) form. They are mainly found in nail and eyelash cosmetics, medical devices for diabetes, dressings, surgical adhesives, orthopaedic prostheses (cements), dentistry and certain everyday connected objects.

- Epoxy resins found in children's play equipment, causing eczema and facial oedema.

Allergic cheilitis is often secondary to the presence of tin in toothpastes and lip balms containing waxes/butters (beeswax, candelilla wax and shea butter).

Eyelid eczema is often caused by eye drops. This eczema affects the conjunctiva, the eyelids and sometimes the cheeks. One study showed that certain eye drops are more responsible: antibiotic > antiglaucoma > preservative. Patients often have multiple sensitivities. They can easily be tested by administering ROATs (two applications per day on a 5x5 cm area on the forearm, without covering the area, for 10 days). Applications should be stopped as soon as eczema occurs.

We can interpret the patch tests of our patients on dupilumab, which will not be the case with JAKis.

BEST OF POSTERS AND COMMUNICATIONS

Speakers: Arnaud Porquet, Eléna Prospero, Maha Habibi, Maelle Pacé, Sébastien Barbarot

1. **TRICHOPHYTON MENTAGROPHYTES Genotype VII** should be considered as an STI in MSM. Several cases were described after a tantric massage.

Treatment with terbinafine 250 mg/day for one month is very often insufficient. They must therefore be monitored and the treatment extended until lesions have resolved. No resistance to terbinafine has been described.

2. A patient with **DARIER'S DISEASE** who did not respond to conventional treatments responded to treatment with baricitinib and IL-17.

3. Treatment of **XANTHELASMAS** with intra-lesional injections of sodium heparin. One session was held per week, with a maximum of 10 sessions. Improvements appeared after five to six weeks. The treatment was well tolerated, with haematomas and post-inflammatory pigmentation occurring as adverse effects in 40% of cases (although this was in a North African population).

4. **DISSEMINATED ACTINIC POROKERATOSIS** treated with a cholesterol compound (2% simvastatin, 2% cholesterol, Excipial lipo cream QS 100 g). Patients had a significant improvement in quality of life and few adverse effects were reported.

5. Study on prebiotics for the **PRIMARY PREVENTION OF AD** exclusively before birth.

The only protective factor against AD is to reduce antibiotic use in newborns. Questions still remained about water softeners, probiotics and prebiotics.

This study showed that there is now solid evidence to rule out a clinically significant effect of perinatal prebiotics on the prevention of AD at one year of age.

RINGWORM IN CHILDREN

Speaker: Sophie Leducq

Use your Wood's lamp to diagnose *Microsporum canis*, which produces a bluish fluorescence.

Your dermatoscope is also a weapon for diagnosis, for identifying the germ in question and for providing follow-up. Trichoscopy reveals comma, corkscrew, zigzag and barcode hairs. Trichophytic ringworm (endothrix parasitism) produces comma and corkscrew hairs on trichoscopy, while microsporic ringworm (ectothrix parasitism) produces zigzag and barcode hairs.

From a therapeutic point of view, what's new in 2024 is the possibility of prescribing itraconazole in community medicine on a compassionate basis. However, in November 2024, the marketing of itraconazole syrup was discontinued, which poses a problem for children who cannot swallow capsules.

Recommendations for microsporic ringworm are changing in December 2024:

- **For children weighing between 10 and 20 kg,** prescribe terbinafine for six weeks (1/4 tablet once a day with a meal, i.e. 62.5 mg/day). If the ringworm has not cleared after six weeks of treatment, it can be continued for a further two weeks.

- **For children weighing more than 20 kg**, prescribe itraconazole for six weeks (one capsule once a day outside of mealtimes, i.e. 100 mg/day).

GERIATRIC DERMATOLOGY

Speaker: Priscille Carvalho

Chronic eczema in patients over 65 taking multiple medications (> 5)

A study of 646 patients showed that stopping the responsible drug did not improve the eczema and could be dangerous by aggravating the co-morbidity for which the treatment had been prescribed.

Most often, a calcium channel blocker is responsible for the eczema. We need to treat our patients by avoiding long-term local CSs, which aggravate dermatoporosis. Treatment with dupilumab is possible with good efficacy and few adverse effects.

Drug-induced dermatitis in the elderly

A French allergology review showed that the average age of patients was 82, that 64% were taking more than five medications and that 30% were self-medicating. So we have to ask them because they do not think to tell us. Thirty-two percent of them had had a drug allergy (often in childhood) and 21% could not remember the clinical expression of this allergy.

Thirty-five percent had severe drug-induced dermatitis, including 18 cases of DRESS syndrome. Twenty-one percent of these drug-induced dermatitis cases were preventable.

SURGICAL TREATMENT FOR PILONIDAL SINUS

Speaker: Elise Pommaret

This is an acquired condition that tends to affect men around the age of 20, decreases after the age of 25 and is rare after the age of 45. It is a foreign body granuloma, with hairs being the foreign body.

We should look for the dimple(s), look down to the anus and investigate other sites of hidradenitis suppurativa.

Initial pilonidal sinus abscesses should not be treated surgically, as in 65% of cases there will be no recurrence.

There are several surgical techniques – wide "lay open" excision, limited "deroofing" excision and wound closure – but there are also **minimally invasive techniques**:

- SilaT, which involves curettage followed by destruction of the sinus via thermocoagulation using a 1470 nm laser fibre
- EPSiT. This is like laparoscopy

The two methods are equivalent in terms of results, but EPSiT is more expensive.

IMMUNOLOGY AND PERSONALISED MEDICINE IN DERMATOLOGY

Speaker: Michel Gilliet

Prof Michel Gilliet presented the results of his research into the creation of a molecular map of inflammatory dermatoses. The aims are to help diagnose uncertain cases and erythroderma, fine-tune treatment and gain a better understanding of switches in certain conditions.

They used NanoString technology. They extracted DNA from a skin biopsy taken with a 4 mm punch and then hybridised the probes (NanoString). They defined inflammatory profiles for 20 inflammatory dermatoses.

This will pave the way for individualised treatments, enable the therapeutic target to be chosen rationally based on the dominant molecular profile, and help gain a better understanding of non-responders and immune shifts.

These techniques are already used in day-to-day practice in his department in Switzerland.

WHAT'S NEW IN DERMATOLOGICAL THERAPY?

Speaker: Carle Paul

1. In psoriasis, the pressure on treatment with systemic agents needs to be eased:

- There is **no risk of MTX-related liver fibrosis**, except in obese patients, those with metabolic syndrome, alcoholics, and diabetics. There is therefore no point in performing fibroscans.

- In HIV patients whose infection is well controlled, tolerance of biotherapies is satisfactory. IL-17, IL-23 or 12/23 inhibitors should be preferred to TNF inhibitors.

- Conventional systemic treatments (IL-17, IL-23 or 12/23 inhibitors) should be continued in the event of surgery. A therapeutic window is required with TNF inhibitors if there is a high risk of infection.

- There is a risk of reactivation of latent tuberculosis with TNF and JAK inhibitors, but not with IL-17 and IL-23 inhibitors. It is therefore recommended that **latent tuberculosis should not be treated if IL-17 or IL-23 inhibitors are used**.

2. Local CSs increase the risk of osteoporosis and fracture. There is a dose effect and a duration effect. We therefore need alternatives to local CSs for the treatment of our chronic inflammatory diseases.

3. New treatment option for hand eczema: delgocitinib cream twice a day.

4. Can JAK inhibitor or dupilumab therapy be spaced out after monitoring in AD? No for JAK inhibitors: patients relapse within four weeks. On the other hand, yes for dupilumab: when treatment is stopped, the benefits are maintained over 36 weeks for the majority of patients.

5. Comparative assessment of the safety of JAK inhibitors and dupilumab in real life. Dupilumab mainly causes ophthalmic complications, whereas JAKis increase the risk of skin infection, acne-like rashes, acne and blood disorders (anaemia, neutropenia, thrombocytopoenia, etc.). Longer-term data are required to assess the risk of rare or delayed adverse effects.

6. Baricitinib should be maintained on a long-term basis when benefits are obtained for alopecia areata.

GAME CHANGER - THERAPEUTIC REVOLUTIONS THAT CHANGE (PATIENTS') LIVES / OPINION (OF DERMATOLOGISTS) - EPISODE 1

Speakers: Delphine Staumont-Sallé, Pascal Joly, Caroline Robert, Denis Jullien, Olivier Chosidow

In this excellent session, five speakers addressed five current topics by summarising some key publications.

The first speaker was **Delphine Staumont-Sallé**, who gave a presentation on **prurigo nodularis (PN).** She described two pivotal clinical trials on dupilumab – *PRIME* and *PRIME2* – which clearly demonstrated the efficacy and tolerance of this treatment in PN. This led to a first MA in 2024 for this first-line indication. She also reminded us of the clinical definition which includes chronic pruritus with signs of scratching and multiple pruritic lichenified skin lesions. She addressed the **burden of this disease, which is much greater than initially recognised and multidimensional**. Concluding her session, Dr Staumont-Sallé talked to us about **upcoming new therapeutic options including nemolizumab, an IL-31 inhibitor that has been very promising in phase 3 studies.**

The second speaker, **Pascal Joly**, talked to us about the **use of rituximab in pemphigus.** He presented all the steps that led to the identification of rituximab as a first-line treatment for pemphigus. It was the first treatment that very quickly reduced the use of systemic corticosteroid therapy compared to standard treatments. He also described the major randomised head-to-head study versus mycophenolate clearly demonstrating the superiority of rituximab. Long-term follow-up studies have demonstrated prolonged remission in more than 60% of patients after seven years. Lastly, he presented a **recent study that demonstrated the use of anti-DSG antibodies as a marker of disease relapse in order to be able to only treat patients who need it during follow-up. It is interesting to know that the long-term cost is shown to be lower than systemic corticosteroid therapy.**

The third speaker was **Caroline Robert**, who shared her excellent expertise on **metastatic melanoma.** She first told us about the first anti-BRAF therapy discovered (vemurafenib). However, the duration of response to this treatment was very short. We realised over time

that the combination with MEK inhibitors increased the response and delayed the development of resistance. She then provided an overall picture of the available immunotherapies, including dual immunotherapy with ipilimumab + nivolumab, anti-PD1 treatment (pembrolizumab and nivolumab) and the new combination of nivolumab + relatlimab (anti-LAG3 treatment). She presented us with portraits of patients in complete remission following single or double immunotherapy. **Resistance to treatment in 50% of patients remains a significant challenge for the future.**

The fourth speaker was **Denis Jullien**, who presented the **latest developments in biological treatments for psoriasis.** He started with the basics of the original treatment including methotrexate, ciclosporin and acitretin. The drugs that emerged subsequently were TNF and IL-12/23 inhibitors. Then, the discovery of the Th1 pathway led to the development of IL-17 and -23 inhibitors. These drugs helped achieved PASI 90-100 rather than PASI 75. **However, many challenges still remain, such as preventing the development of psoriatic arthritis or simply completely curing the disease.** Many oral medications are also currently used, such as JAK inhibitors and deucravacitinib.

The last speaker was **Olivier Chosidow**, with the subject of interest being **infectiology.** He told us about prospects for treating fleas, including ivermectin. First, he mentioned the treatments originally studied, namely malathion, which showed twice the efficacy of d-phenothrin, a reference treatment. Then, oral ivermectin treatment causing parasite death via damage to the neuromuscular junction was presented. A landmark 2010 study showed high efficacy compared to topical malathion. **Current challenges are numerous: malathion has been withdrawn from the market, pyrethrins can have resistance, and doubts about systemic absorption and the first cases of resistance to topical ivermectin have been published. Lastly, it was mentioned that pediculosis remains difficult to treat and that oral ivermectin at 400 micrograms/kg/day on days 1 and 8 is highly effective and should have its place in therapy for severe or treatment-resistant cases.**

NEW DEVELOPMENTS IN VASCULAR MALFORMATIONS

Speaker: Anne Dompmartin

Professor Anne Dompmartin is a world-renowned paediatric dermatologist specialising in vascular anomalies. These are broadly divided into vascular tumours and vascular malformations. Today's topic focused on the latter, i.e. vascular anomalies. **Most are sporadic, while a few are hereditary**. **With hereditary lesions, there is polymorphism explained by the "double-hit" phenomenon, which explains the appearance of the lesions.** Thus, in the same family, some individuals have very few lesions and others are covered with them, because the phenotypic expression is different. The mutation must be present in the tissue. **Sporadic unifocal venous malformations are the most frequent form encountered clinically. It has been observed that 50% of patients carry TIE2 mutations and 20% the PIK3CA mutation**. Multifocal clinical forms exist but are very rare. Studies on the signalling pathways of vascular tumours have identified several elements such as activation of the PI3K/AKT/mTOR pathway which leads, for example, to Klippel-

Trenaunay syndrome, and activation of the RAS/MAPK/ERK pathway, producing multiple vascular malformations.

The management of these disorders is multidisciplinary, involving in particular a dermatologist, an interventional radiologist and a surgeon. A precise diagnosis is established using Doppler ultrasound, MRI and/or arteriography. Treatment options depend on the type of tumour and include surgery and sclerosing therapy.

In terms of the drugs used, we have **rapamycin – or sirolimus**, a selective immunosuppressant initially intended for kidney transplant patients. A phase 3 study demonstrated 50% improvement for symptomatic slow-flow arteriovenous malformations with a favourable safety profile. Next, **alpelisib** is a PIK3CA inhibitor that significantly reduces tumour size. The next agent is **trametinib**, a MEK inhibitor (also used for advanced BRAF-mutated melanoma), which is more effective than sirolimus. However, in several countries it cannot be combined with a BRAF inhibitor in cases of benign disease, and this considerably reduces its tolerance. Next, **thalidomide** is an anti-VEGF anti-angiogenic agent occasionally prescribed in these conditions. Treatment is tailored to the severity of the clinical presentation. Lastly, **lenalidomide** can also be used and is better tolerated. However, it is less angiogenic and the results are less significant.

Prof Dompmartin ended her talk by reviewing the key messages. First, several genetic anomalies have been discovered, allowing us to understand the pathophysiology and better target treatments. Then, precisely diagnosing the type of vascular anomaly is essential and allowed by the clinical examination; it is fundamental for PIK3CA mutations and the genetic analysis of tissue. Next, even though targeted treatments have provided a whole new therapeutic perspective for these disorders, **many questions still remain unanswered. For example, it remains unclear which patients will benefit most from therapies and how they can be combined with interventional treatments**. To conclude, it was mentioned that **vascular anomalies are particular benign lesions where the genetic anomalies are those of cancer, but within endothelial cells with a stable genome. Management should therefore be personalised and multidisciplinary**.

WHAT'S NEW IN INTERNAL MEDICINE?

Speaker: Patrice Cacoub

Dr Cacoub is a world expert in autoimmune and inflammatory diseases. He divided his presentation into autoimmune diseases first, then vasculitis. He started with the **discovery of the role of *CD19 CAR T-cells* in the treatment of autoimmune diseases. In a large review, patients treated with this method were brought into complete remission despite initially having had a highly refractory disease.** The results even showed that it is possible to achieve negative serum testing results for lupus patients. However, they still maintain a good vaccine response. This in itself is an "immunological reset", as Dr Cacoub mentioned. This review also investigated short-term safety, which is good. Another publication informed us that *CAR T-cells* cause depletion of *CD-19 and CD-20* B-cells compared to rituximab. **The US FDA, however, issued a significant risk advisory in a**

companion publication, reporting 22 cases of T-cell-mediated cancer, the majority of which occurred within one year of *CAR T-cell* therapy. Therefore, attempts should also be made to use related strategies targeting B-cells, but with a better safety profile. Dr Cacoub mentioned new bispecific CD3-CD19 treatments such as teclistamab, which has demonstrated very interesting results in rheumatoid arthritis refractory to all treatment. He then pointed us to a publication on the treatment of hereditary angioedema. The authors used *CRISPR-Cas9* to modify the kallikrein gene, inducing complete disease remission with a single dose of treatment. ***CRISPR-Cas9* therapy is therefore very promising in several diseases for the future.** Next, for the treatment of giant cell arteritis, the GIACTA study reported very interesting results for tocilizumab. The presentation concluded with the recent comparative study of infliximab versus cyclophosphamide in the treatment of Behçet's disease (ITAC trial). Infliximab demonstrated a 1.5-fold greater response with a significantly lower relapse rate than cyclophosphamide.

WHAT'S NEW IN PAEDIATRIC DERMATOLOGY?

Speaker: Stéphanie Mallet

Dr Mallet is highly recognised within the French paediatric dermatology community. She began her presentation with some key studies on dupilumab in atopic dermatitis. The first study presented demonstrated that **dupilumab reduces the risk of bacterial infection in atopic dermatitis patients**. This disproves the popular belief that it increases the risk of infection. Next, a study on the **role of dupilumab in eczematous *GVHD* demonstrated a clear response in severe and refractory cases.** Again in atopic dermatitis, a study then demonstrated that **methotrexate and ciclosporin had positive responses but that methotrexate maintained a longer-lasting response after treatment was stopped**. Both treatments had a good safety profile. Then, the **ALLEGRO study on ritlecitinib 30 and 50 mg in severe alopecia areata demonstrated significant hair regrowth versus the placebo. This is a suspensive treatment**.

In vascular anomalies, an interesting article on the long-term follow-up of patients with *PHACE* syndrome reported generally minor aesthetic sequelae. However, a large majority of patients still had neurological symptoms such as migraines and learning disabilities. Next, for **PIK3RI-PROS syndrome, a study on the role of alpelisib demonstrated a reduction in lesions in 75% of cases with rapid clinical improvement.** In *BASCULE* syndrome (*blue-white-red vasomotor dermatosis*), two large-scale studies have recently been published. We now understand that this involves vasomotor dysfunction and seems to be associated with several neuropsychological disorders. However, the precipitating factors are difficult to identify according to the authors. Electrocardiograph does not appear to be necessary in the assessment.

Next, a case of hereditary epidermolysis bullosa treated with an innovative gene therapy demonstrated complete regression of ocular involvement. Subsequently, a **review of biological agents in congenital ichthyosis revealed rather disappointing results**. Netherton syndrome demonstrated the best response, although it remained modest. **For giant congenital nevi, series of cases treated with trametinib (a MEK inhibitor) have**

demonstrated promising results in reducing pain and progression. Interestingly, these treatments appeared to be better tolerated as monotherapy in these patients than in patients with melanoma. Lastly, Dr Mallet closed the session by talking about **chronic visible dermatoses. In the majority of cases, adolescents felt stigma associated with the visibility of their disease. In terms of prevalence, Verneuil's disease ranked first and hyperhidrosis second. This was also highly correlated with bullying and social isolation. Unfortunately, only a minority of patients spoke openly about it.**

INTERACTIVE SESSION ON SKIN CANCER: ONCOLOGY IN PRACTICE

Speakers: Barouyr Baroudjian, Céleste Lebbé, Ines Nakouri, Safa Idoudi, Pauline Tétu, Emilien Ezine, Mouna Bennani

This session was dedicated to **the management of melanoma**. In the form of an interactive quiz, the speakers addressed different aspects in the diagnosis and treatment of melanoma.

First, in the **initial management of a pigmented lesion suspected of melanoma, the ideal biopsy is an elliptical excisional biopsy with margins of 1 to 2 mm (without definitive margins).** This allows for an adequate evaluation of all melanoma criteria (Breslow thickness, presence of ulceration and microsatellitosis) and helps guide subsequent management. A partial biopsy may be appropriate for large-diameter pigmented lesions on the face or acral area, but it should be interpreted with caution.

Next, the speakers reviewed the **recommendations for surgical margin revision.** When the diagnosis of melanoma *in situ* is confirmed, a 0.5 cm margin is usually adequate. For lentigo maligna, especially on the head, a surgical technique allowing histological control of the periphery is interesting and provides for a better evaluation (slow Mohs or "spaghetti" technique). For invasive melanomas with a Breslow thickness of 1 mm or less (T1), revision with 1 cm margins is suggested. For Breslow thicknesses of 1 to 2 mm (T2a and T2b), revision can vary between 1 and 2 cm and above 2 mm (T3 and T4), a revision of 2 cm is recommended. These surgical treatment recommendations come largely from the French skin cancer group (GCC, https://www.cancer-et-peau.com/) but have also been adopted by several international expert groups (ESMO, NCCN, Cancer Council Australia, etc.).

Then, the speakers discussed the sentinel node (SN) technique. This technique has three objectives: adequate staging (positive SN = Stage III according to AJCC 2018), prognosis of our patients (positive SN = worse prognosis) and guidance for subsequent management including follow-up planning and indications for adjuvant treatment. The sentinel node technique is indicated for melanomas with a Breslow thickness of 1 mm and above or ulcerated melanomas with a Breslow thickness below 0.8 mm. For melanomas with a Breslow thickness between 0.8 and 1 mm, it is optional and must be tailored to patients. The Melanoma Institute of Australia's interactive website is useful for assessing the risk of SN involvement and helps us guide our patients (https://www.melanomarisk.org.au/SNLLand). Usually, the SN technique is recommended for patients for whom we are considering adjuvant therapy. It is often omitted in very elderly

patients and those with a contraindication for general anaesthesia or ECOG 3. In addition, it is sometimes abandoned by some teams for Stage IIB or IIC melanomas (Breslow thickness of 2 mm with ulceration or 4 mm or more) in BRAF patients negative for V600E. These patients (Stage IIB/IIC) already have the indication for one year of adjuvant treatment with a PD1 inhibitor (pembrolizumab or nivolumab). However, their estimated prognosis is less accurate without knowing the SN status and the discussion of benefits/disadvantages is less adequate.

For practical guidelines for monitoring patients with melanoma, again, we can refer to the GCC website. In short, for thin melanomas, simple monitoring is recommended with clinical, dermoscopic and lymph node examinations at least twice a year for three years and once a year for life thereafter. For Stage IIB, IIC and III melanomas, monitoring is more frequent and a nodal ultrasound, PET-CT scans or chest/abdomen/pelvic scans and brain MRI are added. As far as possible, screening for melanoma in relatives is desirable. Genetic testing is desirable in the following situations: two invasive melanomas before the age of 75 in two 1st or 2nd degree relatives or in the same individual. It is also suggested when (in the same patient or the parental branch) an invasive melanoma is combined with one of the following diagnoses: ocular melanoma, pancreatic cancer, kidney cancer, mesothelioma or CNS tumour.

The results of adjuvant treatment studies with targeted dual therapy in BRAF+ patients (dabrafenib + trametinib) and anti-PD1 immunotherapy (nivolumab and pembrolizumab) were revised to include recent updates from ESMO 2024. These studies demonstrate the benefits in terms of recurrence-free survival (10 to 15% better) and distant recurrence-free survival (10% decrease). In the management of Stage III patients, it is important to consider the metastatic load within the SN. With inframillimetric sentinel node involvement, the benefits of adjuvant therapy are debated and the adverse effects of treatments often outweigh the benefits. So, for Stage III patients, new biomarkers should be investigated and integrated into the decision. A multidisciplinary team (MDT) meeting is strongly recommended in the management of more advanced melanoma cases.

Lastly, **the new role of neoadjuvant treatment in Stage IIIb melanoma (clinically palpable or imaged nodes) was addressed.** In all Stage III cases, the diagnosis must be confirmed histologically by a lymph node biopsy, usually guided by radiology or ultrasound. Two neoadjuvant treatment options have been reported in the last two years: anti-PD1 treatment and dual immunotherapy. In the first treatment regimen, the patient receives three doses of pembrolizumab (SWOG 1801) prior to lymph node dissection surgery and then completes their year of usual adjuvant treatment. In the new regimen, the patient instead receives two doses of dual immunotherapy (ipilimumab + nivolumab, reversed doses) before dissection. For cases with a major pathological response (complete or near-complete), subsequent adjuvant therapy is omitted. It is important to note that these two types of treatment have demonstrated significant benefits, i.e. a 30 to 40% reduction in subsequent events (local or distant recurrence). Grade 3 or 4 side effects range from 15% (SWOG 1801) to 30% (NADINA). Thus, this neoadjuvant approach is becoming the new standard of practice for Stage IIIB melanomas.