23.13 Hair and nail disorders 5724 David de Berker

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ESSENTIALS Nails grow continuously throughout life, except after exceptional physiological or traumatic events when they are shed. All other less disruptive influences result in changes in the colour, thickness, tex- ture, and growth of nails, and may also affect the periungual tissues. The most common local diseases affecting the nail are psoriasis, fungal nail infections, periungual eczema, and viral warts. Trauma is a common cause of changes in toenails. Looking at the nails is an important part of the general examination, since changes such as clubbing or splinter haemorrhages can indicate systemic disease. Hair growth in a healthy person is determined by body site, gender, and age. Within these parameters there are accepted norms. Disease can affect hair growth by direct action on the fol- licle or by indirect effects sustained through generalized physio- logical disturbance. Clinicians might be asked to assess specific diseases of the scalp with implications for hair growth, or specif- ically to address pathological patterns of hair growth where there may be underlying systemic disease. Common diseases of the scalp include psoriasis, eczema, fungal infection, alopecia areata, and the scarring alopecias. Telogen effluvium is the most common hair problem related to general medical or surgical upset, char- acterized by variable shedding of hair about 6–10 weeks after a period of significant physiological disturbance. Where scalp hair loss presents in association with increased hair on the body or at sites associated with masculinity (hirsutism), a pathological source of androgen should be sought. A reduction in scalp hair is a normal part of ageing in both sexes. Disorders of the nails The nail grows from the matrix and is supported by the nail bed until it reaches the free edge. At the proximal and lateral margins, it is em- bedded in the nail folds. Local and systemic diseases can alter the ap- pearance and function of all four structures (Fig. 23.13.1). The most common local diseases affecting the nail are psoriasis, fungal nail infections, periungual eczema, and viral warts. Tumours other than viral warts are rare. They include squamous cell carcinoma and ma- lignant melanoma. Systemic diseases manifested in the nail include cardiovascular, respiratory, and gastrointestinal diseases leading to clubbing; vascular phenomena (e.g. splinter haemorrhages, cyan- osis) and changes in nail growth as a result of general metabolic fac- tors influencing nail matrix function. Psoriasis Psoriasis affects 1.5–3% of the population, and nail involvement is found in up to 90% of patients at some time. Clinical features The most common manifestations are pitting, onycholysis, ‘oily spots’, transverse

ridging of the dorsal surface, splinter haemorrhages, and subungual hyperkeratosis. Pits represent surface defects in the nail due to foci of psoriatic epithelium (Fig. 23.13.2). Onycholysis is separation of the nail from the nail bed arising because psoriasis of the 23.13 Hair and nail disorders David de Berker Nail bed Lateral nail fold Cuticle Nail plate Cuticle Proximal nail fold Nail matrix Distal interphalangeal joint Lunula Fig. 23.13.1 Anatomy of the normal nail.

23.13 Hair and nail disorders 5725 nail bed reduces adherence of the nail. Subungual hyperkeratosis is thickening of the skin of the nail bed with psoriatic scale that cannot be lost because of the overlying nail. An oily spot is psoriasis in the nail bed. It is termed ‘onycholysis’ if it extends to the free edge. These signs might allow diagnosis of psoriasis at other sites. Even in the presence of obvious psoriasis elsewhere, fungal infection should be sought if the nail features are not typical, because treatable infection can be superimposed and might warrant active therapy. Treatment Using local therapy, pitting can be concealed with lacquers, or may respond to a potent topical corticosteroid applied over 2–3 months to the proximal nail fold. Severe pitting and other changes might occasionally justify a trial of injection of triamcinolone acetonide, 0.1 ml of 2.5–5 mg/ml, into the proximal nail fold, with preliminary local anaesthetic. Onycholysis is difficult to manage and is made worse by trauma and picking. Patients should avoid leverage at the free edge by keeping the nails short and by wearing gloves during wet or dirty work. Debris caught beneath the nail should be removed with a soft nail brush. Excavation with a pointed tool (a common cleaning technique) makes the condition worse. Topical calcipotriol or potent corticosteroid ointment can be helpful and needs to be applied under the free edge at night. Clipping the nail back to the point of separation from the nail bed can facilitate treatment of nail bed psoriasis with topical therapy. Occlusion of the nail with tape is also advocated. Systemic therapy (e.g. methotrexate, ciclosporin, acitretin, or biologics) and psoralen plus ultraviolet A (UVA) can help and might remove the need for inconvenient local treatments. Fungal nail infection The prevalence of fungal nail disease is up to 13% in urban areas in developed countries, but less than 1% in rural Democratic Republic of Congo. It largely affects toenails associated with enclosed foot- wear; raised humidity probably explains the difference between European and African prevalence rates. The principal pathogens are dermatophyte fungi, which infect skin and nail; Trichophyton rubrum and Trichophyton interdigitale are the most common (see Chapters 8.7.1 and 23.10). The nondermatophyte fungi (e.g. Fusarium sp., Scopulariopsis brevicaulis) and yeasts (e.g. Candida spp.) are uncommon pathogens. Onychomycosis is more common in damaged nails. Tinea pedis often coexists between the fourth and fifth web space or as a moccasin infection (a diffuse, scaling fungal infection affecting the sole of the foot). Clinical features Onychomycosis presents in one of four patterns (Fig. 23.13.3). Classic onychomycosis is where the nail thickens, becomes yellow, and is undermined by subungual hyperkeratosis. It may involve just the distal and lateral margins of the nail or be throughout, being named distal, lateral, subungual, or total dystrophic onychomycosis, respectively. Superficial white onychomycosis (Fig. 23.13.4) is rela- tively more common in children and is the variant best treated with topical therapy. Proximal white subungual onychomycosis may pre- sent with a white proximal nail plate and no destruction in the early stages of the disease; this pattern is more common in patients who are immunosuppressed (e.g. organ transplant recipients, those with (a) (b) Fig. 23.13.2 (a) Psoriasis with pitting of the nail and splinter haemorrhages in the nail bed. (b) Psoriasis with marked subungual hyperkeratosis. (b) (a) (d) (c) Fig. 23.13.3 Four most common variants of onychomycosis: (a) distal lateral onychomycosis; (b) total dystrophic onychomycosis; (c) superficial white onychomycosis; (d) proximal white subungual onychomycosis.

section 23 Disorders of the skin 5726 HIV infection). Candida spp. are most commonly found colonizing the damp undersurface of an onycholytic fingernail, where warmth and humidity are common in affected individuals (e.g. caterers). There might also be factors influencing local or systemic immunity (e.g. peripheral ischaemia, Raynaud's, and diabetes mellitus). Diagnosis Mycological confirmation should normally be obtained before starting systemic antifungal therapy. A large sample of discoloured nail plate, with underlying soft debris, is required for a reliable result; this is best taken using heavy-duty nail clippers. Infection can be confirmed through definite identification on microscopy, with identification of the fungus through subsequent culture. Microscopy alone is indicative, but not conclusive as the fungus can be nonviable or already treated. Polymerase chain reaction can also be used to type the fungus, but is at risk of detecting clinically irrelevant antigen in the absence of viable pathogen. The importance of making a clear diagnosis before treatment is greatest where systemic therapy is ad- vocated. Both of the main agents have been reported to cause severe reactions, such as Stevens–Johnson syndrome and toxic epidermal necrolysis. Drug interactions are a particular risk with itraconazole. Treatment Terbinafine and itraconazole are systemic therapies for dermato- phyte onychomycosis. Terbinafine is slightly more effective than itraconazole in dermatophyte infections, but possibly less effective against Candida spp. Topical therapy is usually less effective than systemic therapy, being of most value in superficial or mild distal infections. Avulsion is usually warranted only in nondermatophyte infections or when systemic therapy is ineffective or contraindicated. Relapse can be diminished by vigorous treatment of local tinea pedis and avoidance of trauma through correction of orthopaedic abnor- malities and the wearing of broad-fitting footwear. Nails in systemic disease Clubbing This is a classic sign of various disorders (Box 23.13.1), but is seen in some healthy people. It is usually more obvious on the fingers than the toes; increased longitudinal curvature and loss of the angle between the nail at its proximal margin and the nail fold is seen. This margin is 'boggy' or fluctuant. There may be associated cyanosis if clubbing is caused by cyanotic heart disease or certain pulmonary diseases. Classic changes of geometry change the angle (Lovibond's angle) at the junc- tion of the proximal nail fold and nail plate. Normally, this is less than 160 degrees, but in clubbing it increases to become more than 180 degrees. Clinically, this corresponds to the loss of a window visible be- tween the dorsal aspects of corresponding contralateral fingers when held against each other (Schamroth's sign). Prominent clubbing can be part of hypertrophic pulmonary osteoarthropathy (HPOA) where hypertrophy of the upper and lower extremities resembles that seen in acromegaly, with additional pseudoinflammatory painful changes of the large limb joints with associated radiological and neurovascular changes. Secondary HPOA is typically associated with lung car- cinoma, mesotheliomas of the pleura, and, less commonly, bronchi- ectasis. Primary HPOA is associated with mutations in the HPGD and SLCO2A1 genes which are involved in prostaglandin metabolism and prostaglandin transmembrane transport, respectively. The diagnostic features of clubbing are shown in Fig. 23.13.5. Splinter haemorrhages These represent blood escaping from the longitudinal capillaries in the nail bed beneath the nail (Fig. 23.13.2a). They can have a local or systemic cause. The most common causes are trauma (e.g. manual labour) and nail psoriasis, in which nail bed vessels are more nu- merous and fragile. Significant systemic causes are uncommon; they include infarction of the vessels (e.g. microemboli in endocarditis) and vessel damage related to other causes of vasculitis. The likelihood of a systemic dis- order is greater when there are associated nail fold infarcts than when nail bed changes are found in the absence of nail dystrophy. Nail fold vessels They might become prominent with dilatation, tortuosity, and haemorrhage. This can be associated with increased length and a ragged appearance of the cuticle. These changes are seen in con- nective tissue diseases, in particular

systemic sclerosis, dermato- myositis, and systemic lupus (see Chapter 23.7). Beau’s lines Any severe illness can lead to a transverse arcuate depression on each nail, often most prominent on the thumbs. Because the average Fig. 23.13.4 Superficial white onychomycosis affecting the big toe and the third toe. Involvement of the upper surface of the big toe is exacerbated by the overlapping second toe. Box 23.13.1 Systemic causes of clubbing • Idiopathic/congenital • Cardiovascular — Congenital cyanotic heart disease — Infective endocarditis • Respiratory — Bronchiectasis — Bronchial carcinoma — Empyema — Fibrosing alveolitis • Gastrointestinal — Liver disease — Inflammatory bowel disease — Malabsorption

23.13 Hair and nail disorders 5727 rate of fingernail growth is 2–4 mm per month, the position of the line indicates the approximate date of onset of the original illness. A milder event may produce just a pale transverse line in the nail (see Mee’s lines, next). Thyroid disease Localized separation of the nail from the nail bed (onycholysis) is sometimes seen in thyrotoxicosis. In hypothyroidism, nail thickening is more common. A form of clubbing termed ‘thyroid acropachy’ occurs in Graves’ disease. Koilonychia (spooning of the nails) This is occasionally seen in iron deficiency anaemia. It is common in the big toes of normal babies and infants, and has been reported in mechanics performing oily work. Rickshaw pullers tend to develop traumatic toenail koilonychia. Acquired colour changes None of the classic variants of white transverse bands or zones repre- sent firm clinical signs. The three main signs reflect vascular changes within the nail bed, unlike the fourth which represents abnormal nail production (Table 23.13.1). Terry’s nail A white nail bed obscures the lunula and extends to the distal 2– 3 mm of the nail bed, where there is a red-brown appearance. This is sometimes associated with cirrhosis, in which setting it was origin- ally described; it is also seen in normal ageing. Uraemic half and half nail This is similar to Terry’s nail, but only 50% of the proximal nail bed is involved and associated with uraemia in some instances. Muehrcke’s bands These are thin, transverse white bands seen usually within the prox- imal part of the nail bed, but which move distally to some degree. They appear sensitive to blood albumin levels and can be reversed if it is normalized. This association is not invariable and there is some overlap with the appearance of Mee’s lines where the change is in the nail plate and not the nail bed. Mee’s lines These are transverse bands resembling Muehrcke’s bands but the white change appears within the nail plate rather than the nail bed. This re- flects a period of abnormal nail production that usually corresponds to (b) (a) 160

> “ 180 Schamroth’s window open in normal digit Phalangeal depth ratio Nail-fold angles Normal Clubbed Clubbed Normal Schamroth sign Normal B A C D Clubbed A B C D Schamroth’s window closed in the clubbed digit DPD IPD DPD IPD
> Fig. 23.13.5 Diagnostic features of clubbing. (a) The normal angle between the nail plate and the proximal nail fold is less than 160 degrees, increasing to greater than 180 degrees in clubbing. If the depth of the finger is measured at the interphalangeal joint (interphalangeal finger depth, IPD), it is greater than the depth as measured at the edge of the proximal nail fold (distal interphalangeal finger depth, DPD) in a normal finger. In clubbing it is the reverse. (b) When two contralateral fingers are apposed on their dorsal surfaces, a diamond-shaped window is revealed in normal fingers, bordered by the proximal nail fold and proximal nail (Schamroth’s window). This is lost in

clubbing. Table 23.13.1 Systemic causes of nail discolouration Nail colour Location Causes Yellow Nail plate Yellow nail syndrome White Nail bed Hypoalbuminaemia Brown Nail bed Chronic renal disease Blue Nail bed Mepacrine Lunula Wilson’s disease Red Lunula Congestive cardiac failure, rheumatoid arthritis

section 23 Disorders of the skin 5728 some form of poisoning. The most common is therapeutic poisoning in the form of chemotherapy, but criminal poisoning with arsenic has been detected by this sign. Associations between acquired nail colour changes, drugs, and systemic disease are listed in Table 23.13.1. Disorders of the hair Hair arises from follicles distributed almost universally over the body surface, within the skin. In some areas it becomes dark and relatively thick in diameter such as in the axillae, groin, and beard. On the scalp it is variably pigmented and of moderate bore. On the rest of the body it is generally pale, fine, and termed vellus hair. Vellus hair is short. Length is determined by the period of growth of any single hair before the hair follicle transforms out of the growth phase (anagen) and into the resting phase (telogen), during which the hair is shed and the follicle gradually returns to the growing phase. The characteristics of scalp disease, features of follicular change, and hair shaft morphology can all be assessed by the use of a dermatoscope, which when used on the scalp is referred to as trichoscopy. Follicles that have an anagen phase of 1000 days or more will grow hair far longer than those where it is limited to 60 days and this re- flects the situation for the scalp and eyebrow, respectively. If scalp hair follicles were grafted to the eyebrow, they would retain their scalp identity and the person would grow extremely long eyebrows! Scalp psoriasis Scalp psoriasis can present in isolation. Equally, where signs else- where are equivocal, scalp changes should be sought to explore and clarify the diagnosis. Typically, there are zones of redness with mod- erate demarcation and variable amounts of scale. Scale can be either light and yellow or composed of dense, adherent white material that exposes pin point bleeding of the scalp when picked off. There is a predilection for the margins of the scalp, the creases behind the ears, and within the external auditory canal. There is rarely marked hair loss, but when inflammation is intense there may be shedding and, if associated with prolonged periods of dense scale, scarring can occur. Diagnosis can be confirmed by scalp biopsy where the main differ- ential is with eczema or discoid lupus erythematosus. The main local scalp diseases, including psoriasis, as well as systemic diseases and treatments affecting hair growth are shown in Box 23.13.2. Treatment Patients mainly complain of itch, scale that drops onto clothing, and the appearance within the hair line. Itch can be relieved short term by washing with tar-based shampoos, but this does little more than wash away the surface scale and only provides a weak antipruritic dose of tar coupled with the detrimental irritant effects of shampoo. It does not address the underlying inflammatory process. Alcohol- based steroid preparations delivered through a nozzle are popular because they leave no residue on the hair and are easy to apply. However, the alcohol can sting in the cracked inflamed skin and the barrier function of the diseased skin is further compromised by the solvent properties of the alcohol. The most effective local treatments entail making a mess of the hair in order to treat the scalp as for psor- iasis elsewhere. This can include thick emollient based products con- taining tar, salicylic acid, and coconut oil massaged carefully into the scalp and left overnight before washing out. An overnight shower cap can protect the pillow and enhance the emollient effect on the scalp. Vitamin D-based products, topical steroid as foam, gel or shampoo, and dithranol can all be used on the scalp. Systemic therapies used for

widespread psoriasis also help the scalp, while phototherapy provides less benefit as the scalp is photoprotected by hair. Eczema Scalp eczema This is usually part of atopic eczema and will be found with a typical history and appearance of the disease elsewhere (see Chapter 23.6). The treatments are as for the skin, with an emphasis on avoidance of irritants (use conditioner in place of shampoo and avoid alcohol- based topical products), the use of some emollient (ideal with short hair, but otherwise use steroids in lotion or cream base), and some steroid creams, gels, or foams. Allergic contact scalp dermatitis This can present acutely, typically in connection with paraphenylenediamine (PPD) in hair dyes. Less aggressive presentations are with allergy to ingredients in other hair cos- metics. Paraphenylenediamine allergy can be sufficiently florid as to appear as an acute cellulitis of the scalp, descending onto the forehead. There may be significant reversible hair shedding in the after- math of such an episode. The allergen is determined by patch testing (see Chapter 23.6). Irritant scalp dermatitis This presents with gradual widespread scalp itch, dryness, and light scale with only slight inflammation. It is usually due to excess use of shampoo in someone who has a low threshold for skin irritancy— which can be part of atopy or acquired with age and loss of sebum. Seborrhoeic dermatitis of the scalp This is also termed pityriasis capitis. It barely itches, but presents as scaling or as ‘heavy dandruff’. Redness and scale affect the nasolabial folds, eyebrows, sternum, and sometimes interscapular skin. In the Box 23.13.2 Local scalp diseases and systemic diseases and treatments affecting hair growth The main local scalp diseases affecting hair growth are: • Psoriasis • Eczema • Fungus • Alopecia areata • Scarring alopecia: lichen planopilaris and discoid lupus erythematosus Systemic diseases/treatments affecting hair growth are: • Telogen effluvium • Endocrine: androgen secreting pathologies, hypopituitarism, hypo/ hyperthyroidism, hypoparathyroidism, acromegaly, hyperprolactina emia, Cushing’s syndrome • Iron deficiency • Malnutrition • Severe chronic illness • Genetic syndromes • Drugs (e.g. antimitotic, anticoagulants, oral contraceptives)

 scalp it might be difficult to distinguish from psoriasis, with light, yellow, slightly greasy scale. Overgrowth of normal skin malassezia yeasts plays a part in the disease and management can be directed at reducing the concentration of this yeast on the skin and scalp with antifungal shampoos containing azoles or selenium sulphide. Topical hydrocortisone can reduce the inflammatory component. Fungal scalp disease Fungus can primarily affect the hair shaft, or the scalp, or both. Where the scalp is heavily involved, it can respond either mainly with scaling or with inflammation, with some common ground between both presentations. An intense inflammatory response can produce a raised, oedematous boggy mass known as a kerion. This is usually found in children in contact with farm animals or pets where the zoophilic fungi Trichophyton verrucosum or Microsporum canis are encountered. Transmission between hu- mans is of anthropophilic fungi where the inflammatory response is usually less. Patterns include discoid areas of hair loss, diffuse scale, diffuse pustules, kerion formation or a scalp with little scale and inflammation, and hairs that snap at the scalp surface revealing a ‘black dot’ of residual hair shaft. Changing urban patterns of fungal scalp disease means that this variant due to Trichophyton tonsurans is now the most common in western medicine. Diagnosis is by scalp scraping and hair pluck for mycology. A differential diag- nosis can be sought by scalp biopsy, but this is seldom necessary in a child. Treatment is with systemic griseofulvin, itraconazole, or terbinafine. All are effective, but griseofulvin requires longer treat- ment and has more side effects. However, in many countries it is the only systemic agent licensed in children who are the most common sufferers. Contact tracing within the extended family is important to try to prevent relapse through reinfection. Infectivity in the early phases of treatment can be reduced with use of antifungal sham-

poos. A kerion can give rise to long-term scarring hair loss, due to the intensity of the inflammation. Alopecia areata The classic pattern of alopecia areata is with small discoid areas (‘areata’) of hair being shed in a scattered distribution on the scalp or body. The eyebrows, and lashes are also commonly affected and the beard area in men. Hair loss might progress through a phase where there are residual white hairs (being lost last) followed by a smooth scalp with retained normal hair follicle openings and no apparent inflammation (Fig. 23.13.6). Scalp biopsy reveals a T-lymphocyte inflammatory process engulfing the dermal papilla of the hair fol- licle. This autoimmune attack on the follicle arises in genetically sus- ceptible people. It which switches off anagen, leading to shedding of hair. This attack is not always overwhelming and some follicles might continue to produce small, short-lived, slightly dystrophic hairs with partial pigmentation. These are known clinically as ‘ex- clamation mark’ hairs because they are distally thicker and more pigmented than they are proximally. Details of the residual follicular openings and exclamation mark hairs are best detected using a dermatoscope (Fig. 23.13.7). Typically, no tests are needed to estab- lish the diagnosis. Where all the hair on the scalp is lost, the clinical pattern is re- ferred to as alopecia totalis, which can extend to all hair throughout the body, known as alopecia universalis. The likelihood of spontan- eous regrowth of hair is between 60 and 80% for small ‘areata’, but 10% or less for extensive patterns. There is a statistical association with autoimmune thyroid disease. Treatment Treatment might suppress the immune attack on the follicle for as long as therapy is maintained, but any clinical response is prone to relapse once treatment is stopped. Topical (lotion, gel, foam, shampoo), locally injected, and sys- temic steroids can all be used, with greater efficacy in proportion to penetration and concentration of steroid. Local side effects of atrophy become prominent with time. The side effects of systemic steroid mean that this is not a good long-term treatment choice. Topical immunotherapy is the term applied to the other main treat- ment option where the patient is sensitized to either diphencyprone or squaric acid dimethylbutyl ester. These chemicals are then ap- plied to the affected scalp at regular intervals to provoke an allergic contact dermatitis. Hair might grow in response to this in 50–60% of patients, with less than 20% in more extensive patterns of loss. Relapse after treatment is common. These chemicals have no license for medical use and there are problems with sensitization of medical Fig. 23.13.6 Alopecia areata with smooth nonscarred scalp retaining normal skin markings. Fig. 23.13.7 Scalp dermoscopy illustrates exclamation mark hairs within the affected area and normal follicular openings distinguishing it from a scarring alopecia.

 staff and contamination of work areas, which limits acceptance. Wigs, hair pieces, and psychological support can be very helpful and are not to be underestimated. Scarring alopecia Lichen planopilaris (LPP) and discoid lupus erythematosus (DLE) These are two of the more common forms of scarring alopecia. Lichen planopilaris is characterized by perifollicular purplish discoloration and scale. The interfollicular skin can be normal. Meanwhile, discoid lupus erythematosus has a more coarse pattern of scale and is more likely to affect the scalp between follicles. Where hairs have been lost, the follicles might be plugged with scale (Fig. 23.13.8). Both condi- tions can leave scarring which appears as multiple hairs aggregated within one follicular opening and loss of normal follicle openings in adjacent scalp. With time, these areas might become smooth, hypo or hyperpigmented. Both conditions may have signs elsewhere to help confirm the clinical diagnosis (see Chapters 23.5 and 23.7). Confirmation of the diagnosis is by scalp biopsy with testing for immunofluorescence. False positive immunofluorescence can be a problem on the scalp. Serological tests are done to exclude systemic lupus erythematosus or in preparation for systemic medication. Treatment Both conditions can be progressive and

irreversible and hence short- term early treatment with systemic steroid can be justified as a means of containing active disease before establishing a less toxic regimen for maintenance. Systemic hydroxychloroquine and acitretin are common options in this order for discoid lupus erythematosus and ciclosporin is an option for lichen planopilaris. Doses can be min- imized by coincident, locally injected, or topical potent steroid. Sun avoidance can be important for some discoid lupus erythematosus patients where photo-exacerbation is noted. Systemic diseases and hair changes Telogen effluvium (TE) is the most common hair problem related to general medical or surgical upset. It is characterized by pronounced shedding of hair about 6–10 weeks after a period of significant physiological disturbance such as major blood loss, high fevers, or a period of active inflammatory bowel disease. The patient might not make the association with recent illness and such clues need to be actively sought when someone presents with hair loss. Anagen effluvium describes a more acute pattern where a toxic event im- mediately switches off the hair follicle. The typical example of this is cancer chemotherapy, where treatment is often directed at prolif- erative cells, inadvertently including those of the dermal papilla of the hair follicle. Other drugs such as retinoids, some anticonvulsants, and proges- togens might contribute to hair loss. The list is much longer, but the data to support commonly cited drugs, such as statins, hormone re- placement therapy, and atenolol are less clear. Treatment This entails looking for any active contributing inflammatory or other systemic precipitating factor and correcting it. The iron status might have some independent relevance and should be maintained within the normal range. Usually, the hair will return to normal. The exception is when the individual is of an age where normal patterns of balding are evolving. In this instance, they might find that the hair pattern returns one or two grades down the line of age-related patterned balding. Where scalp hair loss presents in a woman with increased hair on the body or at sites associated with masculinity (hirsutism), a pathological source of androgen should be sought. The history is extremely important in terms of establishing the time course of change and whether the problem is part of a familial pattern. Disturbed menses will increase the index of suspicion of a definable endocrine cause. In someone with recent alteration of menses and evolving male-type hair changes, the concern is of an androgen se- creting tumour. Where there is a family history and problems have been established over years, often since puberty, the diagnosis is more likely to be polycystic ovary disease. Both can be screened by checking the free testosterone levels. Where this is elevated, further endocrinological, and possibly gynaecological assessment is war- ranted. The rash of secondary syphilis can cause a patchy pattern of hair loss scattered over the scalp like numerous ‘glades in a wood’. FURTHER READING Disorders of the nails Baran R, et al. (eds) (2012). Baran & Dawber’s diseases of the nails and their management, 4th edition. Wiley-Blackwell, Chichster. Daniel CR 3rd, Zaias N (1988). Pigmentary abnormalities of the
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Revision #1
Created 2026-01-22 16:42:56 UTC by Omar Ayman
Updated 2026-01-22 16:42:56 UTC by Omar Ayman