

## Hair and scalp disorders. II.

### Patchy alopecia.

In the previous issue we talked about diffuse alopecia. In this issue we talk about patchy alopecia, according its classification (see pag. 602 of the Book of Pediatric Dermatology, EJPD, n.4, 2002).

#### **Congenital, non scarring, patchy alopecia**

Talking about the newborn (pag. 594 of the Book, EJPD, n. 4, 2002), we already mentioned the most frequent causes of patchy alopecia at birth, namely aplasia cutis, sebaceous nevus and triangular congenital alopecia.

Rarely, congenital disorders or other nevi can be responsible for a patch or plaque devoid of

hair. Among the congenital disorders *tuberous sclerosis* can be responsible for fibrous plaques level with the scalp. The latter can be present at birth or appear in the first period of life on the forehead and scalp, making the clinical diagnosis of tuberous sclerosis easier. From a pathological point of view, the fibrous plaques as shagreen plaques share the same findings of angiofibromas (4).

From a clinical point of view (Fig. 1211, 1212), fibrous plaques are characterized by a thickened skin with soft-elastic consistence and sparse and fine hair, which can be also lighter.



Fig. 1211



Fig. 1212

Fig. 1211, 1212: Tuberous sclerosis: fibrous plaque with thinning of the hair (Fig. 1211). In the young man in Fig. 1212 the diagnosis, besides the fibrous plaque of the right frontoparietal region, is based on angiofibromas of the nose (arrows).



Fig. 1213: Cerebriform epidermal nevus with thinning of the hair.

Among nevi *epidermal nevus* (Fig. 1213) and *nevus siryngocystoadenoma papilliferum* (Fig. 1214) are less rare. The latter can complicate a preexisting sebaceous nevus or be present at birth or appear in the first period of life on normal skin.

Plaques and nodules with more or less severe hypotrichosis can be caused by benign tumors such as hemangioma and malignant disorders such as Langerhans cell histiocytosis. The latter may be present at birth -this is why they are mentioned in this chapter- or appear later on.



Fig. 1215: Alopecia areata in the younger sister on the left and "reactive" trichotillomania in the elder one.



Fig. 1214: Congenital retroauricular nevus siryngocystoadenoma papilliferum.

#### **Non scarring, acquired patchy alopecia with normal skin**

This group includes very frequent disorders such as alopecia areata, trichotillomania (Fig. 1215) and traumatic alopecia. Also secondary syphilis is responsible for multiple, small, roundish patches of alopecia.

*Traumatic alopecia* (Fig. 1216) is frequent in children and favored by long hair. It is due to the belligerence, which is not rare in males also in prepuberal age. Traumatic alopecia is clinically characterized by an irregular, often elongated



Fig. 1216: Traumatic alopecia. The diagnosis is supported by the history and its elongated shape.

area devoid of hair, sometimes with normal but shorter hair, because pulled away, inside the affected area. Rarely the trauma responsible for hair loss is due to the usage of containing and stretching devices, usually in girls.

*Alopecia areata* is an autoimmune disorder as supported by family history and presence of serum autoantibodies. Alopecia areata usually starts in the first decade.

Clinically, it is characterized by one or more roundish patches devoid of hairs and preceded by localized paresthesias as more frequently reported by teenagers and adults. The fully developed patch is characterized by normal skin with exclamation mark-like hairs, namely hairs that progressively taper from the free extremity to the follicular opening (Fig. 1217). The latter

are frequently localized inside or at the periphery of the patch.

The clinical course of alopecia areata is scarcely influenced by treatments. On the other hand, it is related to the severity of the disorder, namely to the percentage of hair loss. This means that a single, small patch of alopecia areata often spontaneously recovers. When the hair loss is more than 20%, a chronic waning and waxing clinical course is likely. Finally, when the hair loss is complete, the latter is usually irreversible. In the phase of regrowth (Fig. 1218) some diagnostic difficulty may arise with trichotillomania, due to the presence of hairs inside the patch, and with vitiligo, due to the possible presence of transitory white hairs (Fig. 1219).

Fig. 1217, 1218, 1219: Alopecia areata: the presence of exclamation mark-like hairs (Fig. 1217), which namely taper towards the follicular opening, is characteristic of the disorder. In the phase of regrowth some diagnostic difficulty may arise with trichotillomania, due to the presence of hairs inside the patch, which in Fig. 1218 are characterized by a target-like appearance of difficult explanation, and with vitiligo, due to the possible presence of transitory white hairs (Fig. 1219). In both cases the history of a previous complete alopecia is a clue to the diagnosis of alopecia areata.

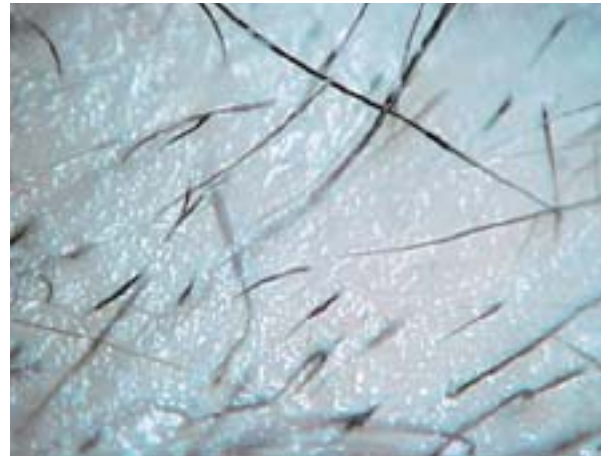


Fig. 1217



Fig. 1218



Fig. 1219

Alopecia areata localized on the nape of the neck has a more severe prognosis. This site is also involved in other types of hair loss, such as that one of the third month of life and moniletrix (pag. 594 of the book, EJPD, n. 4, 2002). Finally, in almost all cases of alopecia areata affecting the nape of the neck the midline angioma of this site comes back visible.

In the most severe cases of alopecia areata, psychotherapy aimed at accepting the disease,

improving the self-esteem, which is decreased due to the disease, and finally, increasing socialization, is probably more important of the symptomatic treatment aimed at stimulating the regrowth of the hair. Alopecia areata should be differentiated from other forms of hair loss with normal scalp skin, namely traumatic alopecia, trichotillomania and luetic alopecia.

*Trichotillomania* is a tic and not a mania, at least in the prepuberal age, when its meaning is



Fig. 1220



Fig. 1221



Fig. 1222

Fig. 1220, 1221, 1222: Trichotillomania is characterized by a geometrical shape, sometimes roundish (Fig. 1221) and thus apparently spontaneous, more often square (Fig. 1220) or serpiginous (Fig. 1222) etc., and thus non spontaneous. Trichotillomania is also characterized by the normality of the skin of the scalp and by the presence inside the patch of hairs with different length. The latter feature is more useful for the differential diagnosis.

the same of the suction of the thumb. The child, especially before sleeping in a twilight state, plays with his/her hair or with the hair of his/her mother till to pull away the hair. This play is done overtly and many times the child is not aware of his/her play. When the child is in school age, it is enough to explain the mechanism of the hair loss and to stimulate his/her esthetical sense in order to overcome the problem. The importance of trichotillomania is different in teenagers and adults, who do not confess their maneuvers and therefore need a psychiatric approach.

Trichotillomania is clinically characterized by a geometrical area, sometimes roundish (Fig. 1221) and therefore apparently spontaneous, more often square (Fig. 1220), rectangular, spindle or serpiginous (Fig. 1222) and therefore non spontaneous, namely non round or oval as in alopecia areata. The geometrical shape is due to the repetitive movement of the play, always persevering on the same sites. Besides the geometrical shape, trichotillomania can be differentiated from alopecia areata thanks to the presence of hairs of different length inside the patch. The latter are normally growing hairs that are not long enough to be caught and pulled out. The skin of the scalp is perfectly normal as in alopecia areata.

In *secondary syphilis* the hair loss can be, especially in women, the symptom leading the patient to the physician. Syphilis should be suspected when there are multiple, small sized patches with indefinite borders, diffuse on the entire scalp, with a moth-eaten appearance. Usually, other concomitant symptoms or signs make the diagnosis easier. The specific treatment leads to rapid regrowth of the hair.

#### **Non-scarring, patchy, acquired hair loss with scaling skin**

This group includes disorders not always directly affecting the hair as ringworm. In fact the presence of thick scales can be associated to a more or less important hair loss level with the scaling area.

Nowadays, *ringworm*, namely ringworm reminiscent of a tonsure, is more often due to *Microsporum canis* in Europe (Fig. 1223). The hairs, which are filled in by mycelium hyphae

and surrounded by small spores muff-like arranged, break 3-4 millimeters from the follicular opening. Their proximal stumps are hardly visible among the scales covering the floor of the patch. However, when scraping on the floor of the patch aimed at performing a fresh examination, the proximal stumps of the broken hairs, 1-2 millimeters long and thicker get evident. From a clinical point of view, the patch of tonsuring ringworm is often regularly round, with clear-cut borders. Tonsuring ringworm is treated with griseofulvin at a dosage 20-25 mg/kg per day for 30-40 days.

Many dermatological disorders can be responsible for a scaling dermatitis with clear-cut borders, simulating ringworm also due to the rarefaction of hair, which can be total. The term "pseudotinea amiantacea" is merely descriptive and does not help to understand the etiology and therefore the prognosis of the different responsible disorders, namely psoriasis, atopic dermatitis, seborrheic dermatitis, ichthyosis, Darier's disease and other rarer disorders. All these disorders usually affect other sites and in this case the differential diagnosis should be better done on the glabrous skin. On the other hand, when only the scalp is affected, the differential diagnosis is much more difficult.



Fig. 1223: "Non inflammatory", tonsuring ringworm of the scalp, due to *Microsporum canis*.

**Table 1. Differential diagnosis of the scaling disorders of the scalp.**

	<i>Age</i>	<i>Type of scaling</i>	<i>Size of scales</i>	<i>Border</i>	<i>Lack of hairs</i>
<i>Tonsuring ringworm</i>	school	patchy	dandruff-like	clear-cut	yes
<i>Psoriasis</i>	whatever	patchy	micaceous	clear-cut	possible
<i>Atopic dermatitis</i>	first years	diffuse	pityriasis-like	indefinite	no
<i>Seborrheic dermat.</i>	peripuberal	diffuse	dandruff-like	indefinite	no
<i>Darier's disease</i>	peripuberal	diffuse	dandruff-like	clear-cut	no
<i>Ichthyosis</i>	first years	diffuse	adherent	indefinite	no

*Psoriasis* can only affect the hairline or the entire scalp giving raise to a scaling helmet. More often psoriasis affects the scalp with one (Fig. 1224) or more round patches, with clear-cut borders. In the latter case the scales are thicker than in tonsuring ringworm and whitish. The scales get even more white with the methodic scratching. Sometimes, the background erythema is yet visible under the scales, getting more visible when removing the scales for therapeutic purposes. The scales are sometimes so thick that the hairs cannot cross them.



Fig. 1224: Psoriasis of the scalp: single patch devoid of hairs, which are not able to cross the scales.

*Atopic dermatitis* can sometimes be responsible for scaling lesions with rarefaction of the hair, even after the first months, about which we already talked (pag. 600 of the book, EJPD, vol. 12, n. 4). In this case the scaling dermatitis of the first months does not regress completely within the first year as in most cases. On the other hand, the scaling lesions wax and wane for years.

Atopic dermatitis of the scalp in children aged more than one year often affects the central area (Fig. 1225) of the scalp. It is characterized by



Fig. 1225: Atopic dermatitis of the scalp with severe thinning of the hair.

dry lesions with pityriasis-like scales and with indefinite borders. The lesions of atopic dermatitis, which are often itchy, usually affect also other sites.

*Seborrheic dermatitis* of the scalp (Fig. 1226), besides the so-called cradle-cap, about which we already talked in the chapter devoted to the child aged 2 to 6 months (pag. 600 of the book, EJPD, vol. 12, n. 4), may affect children in the peripubertal age, sometimes from the end of the first

decade with lesions reminiscent of atopic dermatitis, but usually less itchy.

All types of *ichthyosis* can be associated to a scaling dermatitis of the scalp and thinned hair. The scales are more thick in X-linked (Fig. 1227) and non bullous congenital erythrodermic ichthyosis, particularly in the first years of life.

*Darier's disease* starts in the peripubertal age with yellowish scales mainly affecting the hairline (Fig. 1228), often with follicular keratosis.

Fig. 1226, 1227, 1228: Scaling dermatitis of the scalp with thinning of the hair can be caused by different disorders such as seborrheic dermatitis (Fig. 1226), ichthyosis, especially X-linked ichthyosis (Fig. 1227) and Darier's disease (Fig. 1228).



Fig. 1226



Fig. 1227



Fig. 1228

**Non scarring, patchy, acquired hair loss with inflamed skin**

Eosinophilic folliculitis (see pag. 336 of the Book, EJPD, vol. 6, 1996), is a non infectious disorder. Children can be affected and at this age the scalp can be mainly affected (Fig. 1229, 1230).

Eosinophilic folliculitis usually starts in the second semester of life, lasts 6-9 months and then spontaneously regresses. The clinical course is characterized by subintractable crops of lesions lasting 7-10 days with a free interval of several days or weeks. Characteristically, the suppurative lesions of the scalp do not respond to antibiotics and to whatever treatment and spontaneously regress.

*Pemphigus vulgaris* (pag. 302 of the Book, EJPD, vol. 7, n. 3) is not the most frequent autoimmune bullous dermatosis in children. It is in fact overcome in frequency both by dermatitis herpetiformis and linear IgA dermatosis (2). In

contrast with these two latter autoimmune bullous disorders, pemphigus vulgaris can be transmitted through the placenta (2) from the mother affected or anyway carrying anti-desmoglein autoantibodies. In this case, as in herpes gestationis, blisters occur in the newborn from the first days of life and then spontaneously regress in a few weeks.

In pemphigus the scalp is frequently affected, also in children, and the chronic inflammation can be responsible for hair loss.

The appearance of a persistent, exudating lesion of the scalp (Fig. 1231) can be a clue to the right diagnosis of an erosion of the oral cavity (Fig. 1232). The latter is another site of difficult diagnosis, where pemphigus vulgaris can be initially localized. The clinical diagnosis is later confirmed by a cytodiagnostic method - Tzanck test-, by histological examination and, finally, by direct and indirect immunofluorescence.



Fig. 1229



Fig. 1230

Fig. 1229, 1230: Eosinophilic folliculitis: the more frequently affected age is the second semester. At this age the scalp is characteristically affected. The lesions, which are reminiscent of toxic erythema of the newborn, are typically recurrent.

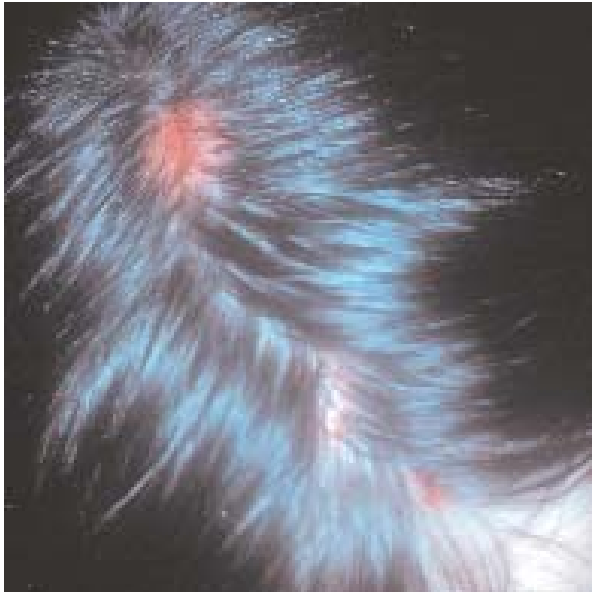


Fig. 1231



Fig. 1232

Fig. 1231, 1232: Pemphigus vulgaris in a 7-year-old girl. The disease started in the oral cavity (Fig. 1232). The appearance of persistent exudating lesions of the scalp (Fig. 1231) made the diagnosis easier.

**Non scarring, patchy acquired hair loss with suppuration in progress**

Between the already mentioned tonsuring ringworm and kerion, about which we will talk in this issue, there are *intermediate forms of ringworm* characterized by a superficial, erythe-

matous inflammation of the scalp (Fig. 1233, 1234), with suppurating folliculitis, without evident hair loss, with indefinite borders, with torpid clinical course. These intermediate forms of ringworm can be hardly differentiated from pyoderma.



Fig. 1233



Fig. 1234

Fig. 1233, 1234: Between tonsuring ringworm and kerion there are intermediate forms of ringworm characterized by superficial inflammation without evident hair loss. *Microsporon canis* (Fig. 1233) is the most frequent cause in Europe.



Fig. 1235



Fig. 1236

Fig. 1235, 1236: Pyoderma can affect primarily the scalp (Fig. 1235) or secondarily to other itching disorders of the scalp, such as pediculosis (Fig. 1236) or atopic dermatitis.

In *pyoderma* of the scalp the infection can be primary (Fig. 1235), but it is usually secondary to an itchy disorder, such as pediculosis (Fig. 1236) and atopic dermatitis.

The diagnosis of pyoderma should be suspected in presence of suppurating lesions with rapid

progression and tendency to extend until antibiotics are given.

Among the other suppurating infections affecting the scalp we should mention *varicella*. The diagnosis of the latter is much more easy on the glabrous skin. However, in doubtful cases the involvement of the scalp is searched for in order to confirm the diagnosis.

*Suppurating hydroadenitis* of the scalp (Fig. 1237) affects children aged less than 1 year. It is characterized by 5 mm to 2 cm in size nodules rapidly undergoing suppuration. A drainage is useful besides antibiotics in the largest lesions.

#### **Non scarring, patchy, acquired hair loss with plaques or nodules**

Many granulomatous and malignant disorders can be responsible for patchy hair loss, due to the intradermal compression of the hair follicle. These disorders can be also responsible for scarring. This is why they are also considered among the scarring forms.

An infiltrated plaque with thinning of the hair can be due to follicular mucinosis. The latter can be idiopathic or, more rarely, expression of lymphoma. The infiltrated plaque with thinned hair is soft-elastic and non painful. Histologically, a lymphocytic infiltrate penetrates the hair



Fig. 1237: Hydroadenitis of the scalp in the unweaned child is characterized by a suppurating nodule (arrow).



Fig. 1238

Fig. 1238, 1239: The most frequent mycotic granulomas in Europe are kerion, which is usually due to *Microsporon Canis* (Fig. 1238) and chronic mucocutaneous granuloma, mainly the diffuse granulomatous type III form (Fig. 1239).

follicle and is responsible for its severe degenerative alterations. Inside the follicle an amorphous substance metachromatically staining with toluidine blue accumulates.

Kerion is the most frequent granulomatous disorder in children. However, granulomatous lesions can be also caused by other disorders such as chronic mucocutaneous candidiasis and leishmaniasis.

*Kerion* starts from the onset with an infiltrated plaque (Fig. 1238) or it complicates a tonsuring ringworm. We are dealing with an intensely erythematous plaque, devoid of hair, very painful and rapidly suppurating. Kerion spontaneously healed even in preantibiotic and pregriseofulvin era. It was the only tinea capitis healing before puberty. However, healing was slow -in months- and with thinning of the hair and scarring sequelae in the most severe cases. Although non significantly, griseofulvin decreased the severity and duration of the disease, mainly due to the host reaction.

*Chronic mucocutaneous candidiasis* affects the scalp both in type I, which is autosomal dominant, and in type III (Fig. 1239).

*Leishmaniasis* of the scalp (Fig. 1240) gives raise to a granulomatous plaque with a torpid clinical course. It does not suppurate, although a



Fig. 1239

characteristic central area of softening is often appreciated. Usually, the hair are not thinned. However, they can get thinned in the most severe cases. The torpid clinical course of a granuloma of the scalp in a child aged a few years in an endemic area should raise the suspicion of leishmaniasis.



Fig. 1240: Leishmaniasis of the scalp in a 5-year-old child coming from an endemic area.

The specific infiltrate of *Langerhans cell histiocytosis* can be responsible for papules and nodules.

Papules, which are more frequent, are the characteristic cutaneous lesions of multisystem histiocytosis type Letterer-Siwe. The characteristic papules are distributed in seborrheic regions such as scalp (Fig. 1241), mainly on retroauricular areas, inguinal folds and central area of the trunk, both on the chest and the back. The characteristic papules are about 1 mm in size, even though they can reach 2-3 mm, closed each other but not confluent and purpuric. The purpuric appearance is not due to liver failure, because also the pure cutaneous forms present purpuric lesions. Sometimes, the papules of the scalp are founded on erythematous skin.

The differential diagnosis from seborrheic dermatitis is easy for the skilled physician, because in seborrheic dermatitis there are never isolated papules.

Moreover, in Langerhans cell histiocytosis there can be nodules and plaques with thinned hair. The latter can be found in multisystem form type Letterer-Siwe and in the self-healing form type Hashimoto-Pritzker, which is charac-

terized by significant polymorphism of the lesions.

In the multisystemic form yellowish, xanthomatous nodules can be seen in the remission phase of the disease (Fig. 1242) after the end of treatment. In the self-healing form type Hashimoto-Pritzker, which is characteristic of the newborn period, there can be infiltrated, erythematous and cyanotic plaques and nodules (Fig. 1243).

Yellow plaques and nodules can be also detected in juvenile xanthogranuloma (Fig. 1244). However, in this case the yellowish color is preceded by a red-brownish color in the histiocytic phase preceding the xanthomization of the lesion.

Juvenile xanthogranuloma as well as other benign proliferations of the first months of life, spontaneously regresses. The regression takes a couple of years, therefore it is more rapidly than mastocytosis and most hemangiomas (see pag. 417 of the Book, EJPD, 1999).

Among the less rare causes of nodules with thinned hair, there are *epidermal cysts*, often familial, firm-elastic, spherical, that can be seen starting from the second decade.



Fig. 1241: Multisystem Langerhans cell histiocytosis with its characteristic purpuric papules.



Fig. 1242: Multisystem Langerhans cell histiocytosis in phase of remission with xanthomized nodules (arrows).



Fig. 1243: Nodules with thinned hair in a newborn with Hashimoto-Pritzker self-healing histiocytosis.

*Pilomatrixoma*, on the other hand, can be seen beginning from the newborn period. It is responsible for plaques and nodules with irregular surface, firm. The nodules can undergo inflammation reaching a diameter 2-3 times larger, to turn back in months to the previous size.



Fig. 1244: The diagnosis of juvenile xanthogranuloma of the scalp is easy in the phase of xanthomization.

Nodules with thinned hair of the scalp can be also due to malignancies such as neuroblastoma and lymphoma. Type B lymphomas, especially those ones with “common” phenotype (Fig. 1245, 1246), may start with cutaneous plaques, localized on the scalp, hard, very large (1). The



Fig. 1245

Fig. 1245, 1246: Lymphoblastic lymphoma B with “common” phenotype starting on the skin of the scalp (Fig. 1245) and after chemotherapy (Fig. 1246).



Fig. 1246

clinical diagnosis should be confirmed by histological examination.

### Scarring, patchy hair loss due to congenital disorders

We already mentioned congenital disorders responsible for non scarring hair loss. There are also congenital disorders responsible for scar-

ring alopecia. Among the latter there is *incontinentia pigmenti*. In this disorder, the typical cutaneous and dental lesions are sometimes associated with an area of scarring alopecia, usually on the vertex (Fig. 1247, 1248). In severe *epidermolysis bullosa dystrophicans recessiva* a scarring alopecia with multiple patches or even



Fig. 1247

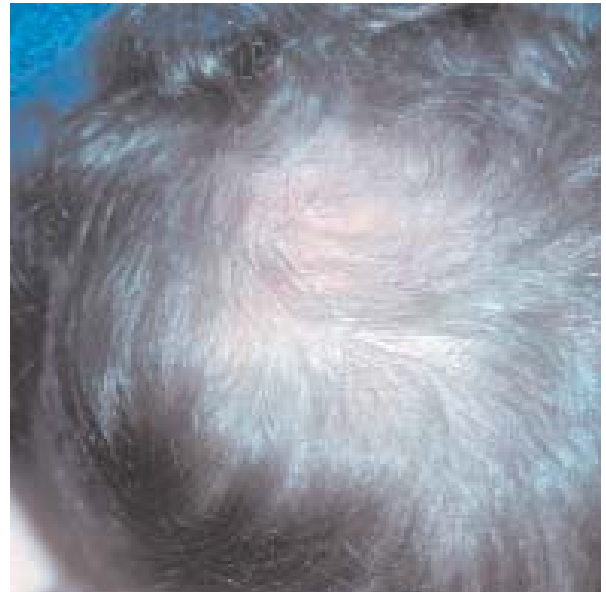


Fig. 1248

Fig. 1247, 1248: Incontinentia pigmenti in pigmented phase (Fig. 1247), with scarring alopecia on the vertex (Fig. 1248).

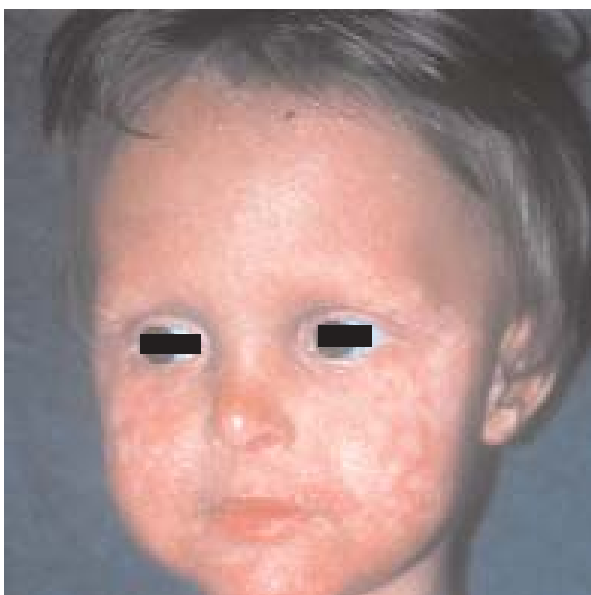


Fig. 1249



Fig. 1250

Fig. 1249, 1250: Rothmund-Thomson syndrome: hypopigmentation and alopecia of the eyelashes, eyebrows and vertex.



Fig. 1251



Fig. 1252

Fig. 1251, 1252: Tick bite (Fig. 1251) can be responsible for a large necrosis (Fig. 1252) and scarring alopecia.

diffuse is frequent. In *Rothmund-Thomson syndrome* a diffuse scarring alopecia is associated to photosensitivity and hypopigmented lesions (Fig. 1249, 1250), besides the characteristic phace. In *keratosis follicularis spinulosa decalvans* diffuse hypotrichosis, which is usually present at birth, can be associated in the post-puberal age to inflammatory lesions with residual scarring.

#### Scarring, patchy hair loss due to acquired disorders

Trauma is the most frequent cause of circumscribed scarring alopecia of the scalp. The latter is usually characterized by a white line lacking hair and follicular openings (Fig. 1206 of the Book, EJPD, vol. 12, pag. 607, 2002).

Third degree burns of the head can be responsible, although rarely, of scarring with thinning of the hair. Deep radiotherapy for angiomas or brain tumors can be responsible for scarring alopecia or sometimes for a diffuse and persistent thinning of the hair.

Tick bite (Fig. 1251) causes lesions of the hair ranging from a transitory alopecia around the granuloma to a scarring definitive alopecia, when the inflammation caused by the substances stimulating the flogosis injected by the tick lead to a large necrosis of the skin (Fig. 1252).

Scarring alopecia due to folliculitis decalvans and acne keloidea of the nape of the neck are not usually observed before the second decade.

Autoimmune disorders such as lichen pilare, lupus erythematosus and scleroderma can be responsible for scarring alopecia. In children localized cutaneous scleroderma (Fig. 1253),



Fig. 1253: Localized cutaneous scleroatrophy of the scalp with greater evidence of dermal vessels.



Fig. 1254



Fig. 1255

Fig. 1254, 1255: Alopecia parvimaiculata is reminiscent of alopecia areata (Fig. 1254). However, at a closer observation, atrophy gets evident, leading to the right diagnosis.

mainly linear or “en coup de sabre” is the most frequent autoimmune disorder, finally leading to thinning of the skin with more evident dermal vessels (Fig. 1253).

Numerous benign and malignant tumors can be responsible for scarring alopecia. Hemangioma is the most frequent in children. It can be responsible, when its regression is complete, for persistent thinning of the hair.

Alopecia parvimaiculata, which is typical of childhood (3), is characterized by small areas of alopecia with inflamed and atrophic skin and irregularly angular, round or oval shaped (Fig. 1254, 1255). Small epidemics and familial cases

were observed, but no infectious agent was identified. Some Authors (2) believe that alopecia parvimaiculata is a childhood form of pseudoarea of Brocq, even taking into account the few pathological findings available. According to the latter the hairs of the same follicular unit are not all affected by inflammation and consequent atrophy. This is why a regrowth of hairs in a few weeks was observed.

With regard to its diagnosis, the history rules out atrophic residua of multiple insect stings and of primary or secondary suppurating bacterial infections, whereas laboratory examinations rule out mycosis and secondary syphilis.

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