Review Article

Skin Appendage Disorders

Skin Appendage Disord 2018;4:187–199
DOI: 10.1159/000484354

Received: June 22, 2017
Accepted: October 18, 2017
Published online: November 29, 2017

Scalp Itch: A Systematic Review

Norma Elizabeth Vázquez-Herrera[a] Divya Sharma[b] Nouf Mohammed Aleid[c] Antonella Tosti[d]

[a]Tecnologico de Monterrey, Campus Monterrey, Monterrey, Mexico; [b]Warren Alpert Medical School of Brown University, Providence, RI, USA; [c]Department of Dermatology, Prince Sultan Military Medical City, Riyadh, Saudi Arabia; [d]Fredric Brandt Endowed Professor of Dermatology, University of Miami, Miami, FL, USA

Keywords
Scalp · Itch · Pruritus · Seborrheic dermatitis · Contact dermatitis · Anxiety · Lichen planopilaris · Lice · Pediculosis · Psoriasis · Trichoscopy

Abstract
Scalp itch is a frequent complaint in the dermatological setting. It is common for the dermatologist to encounter patients with no evident cause of scalp pruritus, making it a distressing situation for both the clinician and the patient. The aim of this paper is to propose a systematic approach to scalp itch, which classifies scalp pruritus into two types: (1) with or (2) without dermatological lesions, and presence or absence of hair loss. Also, it is important to think first about the most common causes and then rule out other, less common etiologies. The acronym SCALLP and the five steps for scalp evaluation (listen, look, touch, magnify, and sample) are useful tools to keep in mind for an assertive approach in these patients.

Introduction

Scalp itch is a frequent complaint in the dermatological setting. Itch, by definition, is the presence of an uncomfortable tingling or uneasy sensation, which is associated with a desire to scratch [1]. Just like skin pruritus, its etiology may be of dermatological or non-dermatological origin. It is common for the dermatologist to encounter patients with no evident cause of scalp pruritus, making it a distressing situation for both the clinician and the patient. The aim of this paper is to review the clinical signs of the most common causes of scalp itch and other less common causes that must be considered in a systematic approach to scalp pruritus.

Materials and Methods

We performed a MEDLINE search through PubMed (1975–2017), using the terms scalp itch and scalp pruritus and included clinical trials, review articles, case series, and case reports to search for the causes of scalp itch.

Pathophysiology

Although various pathogenic etiologies contribute to scalp pruritus, the scalp itself has distinct neuroanatomy and vasculature, specific neuromediators and corresponding receptors, as well as the presence of scalp sebum and microflora, which are all properties that may explain its tendency to be implicated in patients who complain of itch.

KARGER

E-Mail karger@karger.com
www.karger.com/sad

Antonella Tosti
Department of Dermatology and Cutaneous Surgery
University of Miami Miller School of Medicine
1475 NW 12th Avenue, Miami, FL 33136 (USA)
E-Mail ATosti@med.miami.edu

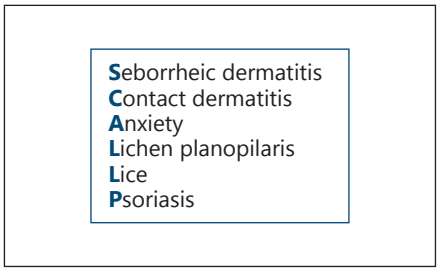

Fig. 1. SCALLP acronym in order to easily recall the most common causes of scalp itch.

Sebum amount and composition vary considerably from individual to individual [2]. Free fatty acids maintain the surface pH and contribute to skin homeostasis [3]. The overall composition of the scalp surface lipids may change in response to dermatological conditions such as atopic dermatitis, seborrheic dermatitis, and psoriasis.

The hair follicle has been recognized as a neuroendocrine organ. It is innervated by multiple structures such as free, lanceolate and Merkel nerve endings, as well as pilo-Ruffini corpuscles. Corticotropin-releasing hormone receptors in the hair follicle have been associated with degranulation and an increased number of perifollicular mast cells [4]. The extent of hair fiber innervations, neuropeptide expression, including interaction with mast cells varies with hair cycling. Cutaneous innervation is also altered by neuropeptides in inflammatory skin diseases [5, 6].

Patient Examination

For evaluating scalp itch, we can follow the five steps proposed by Shapiro and Otberg for evaluation of hair loss: listen, look, touch, magnify, and sample, while focusing on scalp itch [7].

Causes

We propose the acronym SCALLP in order to easily recall the most common causes of scalp itch (Fig. 1). Often, the explanation will be related to one of these causes.

A practical method to classify scalp itch is to consider two types: (1) with or (2) without dermatological lesions. The presence or absence of hair loss may also help with the diagnosis. It is very important to consider that secondary lesions due to scratching, such as crusts and lichen simplex chronicus, that may be present even in cases without a dermatological origin since they are self-inflicted.

In patients with dermatological lesions, we can systematically work through the differential diagnosis using a clinical approach, depending on type of lesion. It is also important to remember that scalp itch may be related to more than one cause, and more than one type of lesion may present. This classification is shown in Figure 2.

Scalp Itch with Skin Lesions

Scalp Itch with Scaling/Erythema

Scalp itch associated with interfollicular and perifollicular scales and/or erythema can be due to the following causes: seborrheic dermatitis, scalp psoriasis, contact dermatitis, early lichen planopilaris (LPP), tinea capitis, dermatomyositis, and xerosis secondary to atopic dermatitis or aging skin. Other less common causes of scalp conditions that may present with scalp itch and hyperkeratosis are pityriasis rubra pilaris, discoid lupus and Langerhans cell histiocytosis [8–10]. Trichoscopy, direct sampling and cultures are useful tools for diagnosis. Histopathology can confirm diagnosis in doubtful cases.

Seborrheic Dermatitis

Seborrheic dermatitis, which affects approximately 16% of the adult population, is one of the most common causes of scalp itch. In a French study, 50% of affected patients complained of scalp itch. Other common symptoms included sensations of prickling, tightness, pain, and burning [11]. Pruritus in seborrheic dermatitis typically disappears or decreases in severity after shampooing. The diagnosis of seborrheic dermatitis may be clinical when scalp itching is associated with flaking in seborrheic body parts such as mid-face and upper chest and back. However, it must be distinguished from psoriasis and contact dermatitis. On trichoscopy, seborrheic dermatitis and contact dermatitis present with arborizing vessels, whereas psoriasis presents with red dots at low magnification and twisted capillary loops at high magnification [12].

Psoriasis

Itch is present in 67–97% of patients with psoriasis [13–18]. Specifically, scalp itch has been reported in up to 80% of patients with psoriasis, and there is positive

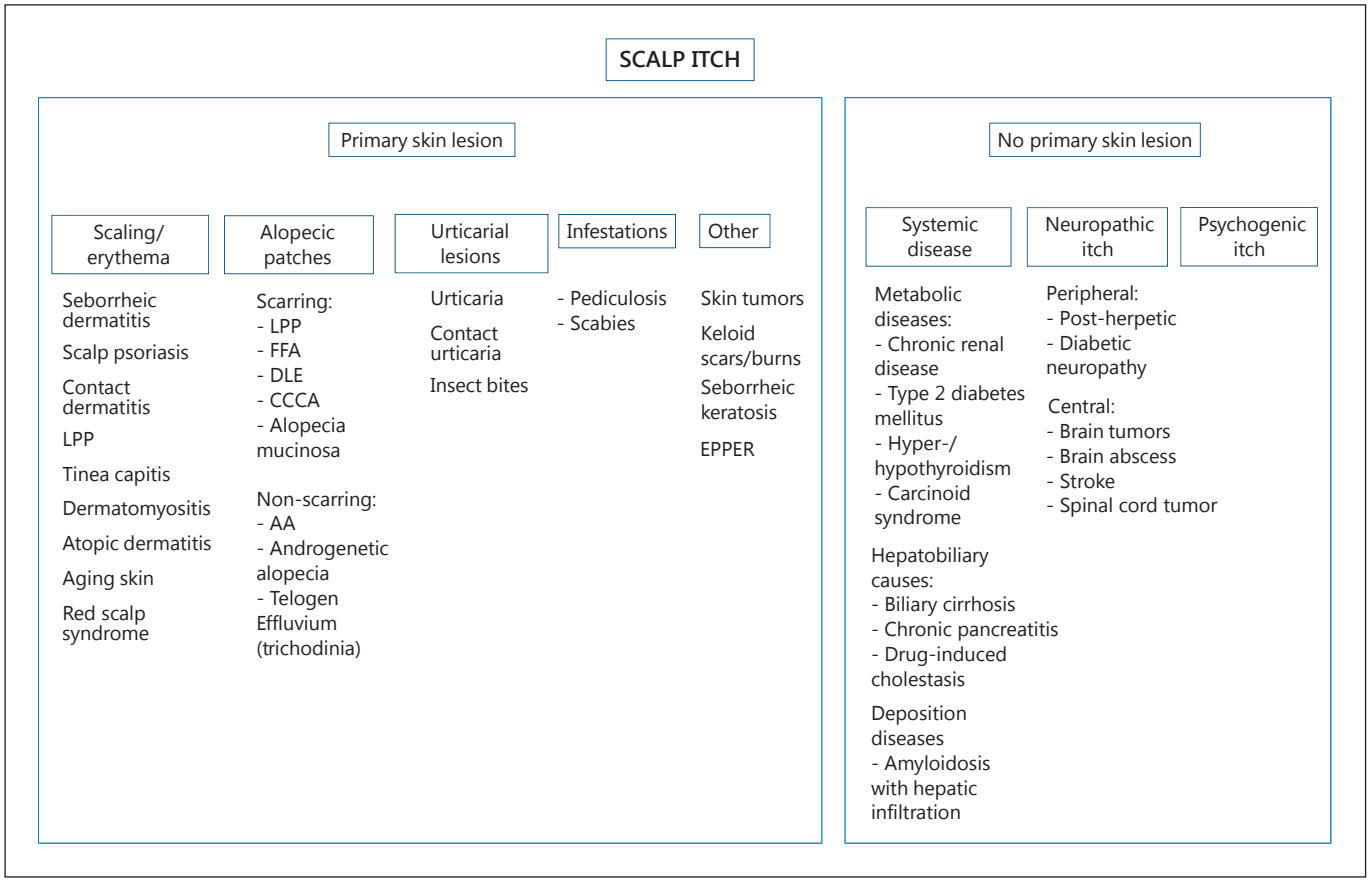

Fig. 2. Proposed algorithm for scalp itching based on presence or absence of primary skin lesions. LPP, lichen planopilaris; FFA, frontal fibrosing alopecia; DLE, discoid lupus erythematosus; CCCA, central cicatricial centrifugal alopecia; EPPER, eosinophilic, polymorphic, and pruritic eruption associated with radiotherapy; AA, alopecia areata.

correlation between the severity of the lesions in scalp psoriasis and the severity of itch [19]. Possible mechanisms that explain pruritus in scalp psoriasis are hyperinnervation, neurogenic inflammation, neuropeptide misbalance, abnormal functioning of the peripheral opioid system, cytokine release, and vascular abnormalities [20, 21]. Regarding quality of life, pruritus is considered by patients as the most important factor that impairs the ability to work, with a negative impact on concentration in 52.5% of patients [15, 22]. The characteristics of pruritus in scalp psoriasis are variable: most of the patients refer intermittent pruritus, and the most important exacerbating factors are stress and temperature [21].

On trichoscopy, in 10× magnification, red dots and globules are present in up to 87% of the patients [23]. At higher magnification, twisted red loops are characteristic; also globules and glomerular vessels may be present [24]. In a comparative dermatoscopic study of 20 patients which scalp psoriasis and 20 patients with seborrheic dermatitis, patients with scalp psoriasis presented with dotted vessels in 50% of cases, glomerular vessels in 65%, red loops in 80%, hairpin vessels in 85%, white scales in 50%, and punctate hemorrhages in 60%. In patients with seborrheic dermatitis, arborizing vessels were present in 80% of cases, atypical red vessels in 65%, and honeycomb pigment in 45% [25]. Scalp psoriasis commonly causes increased hair shedding, and psoriatic alopecia may be present in severe cases.

Contact Dermatitis

Contact dermatitis is another common cause of scalp itch. Clinically, it may present with itch, erythema, and scaling. Arborizing vessels are commonly seen on trichos-

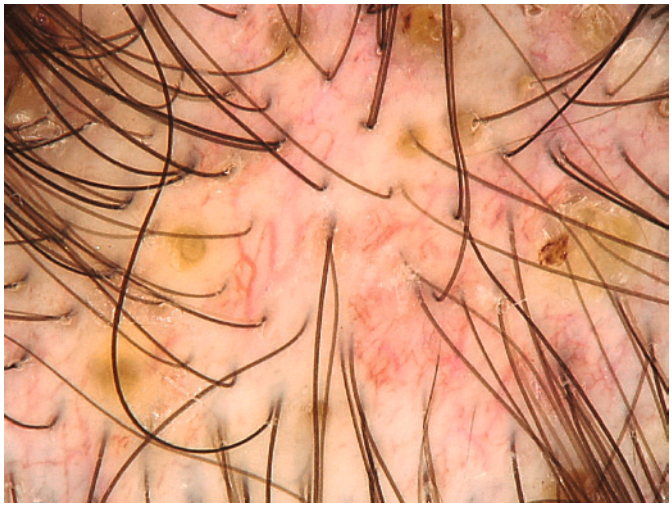

Fig. 3. Red scalp syndrome in a patient with pruritus and scalp erythema associated with papules and pustules. Trichoscopy shows predominantly arborizing telangiectasias, and presence of yellow dots and crusts.

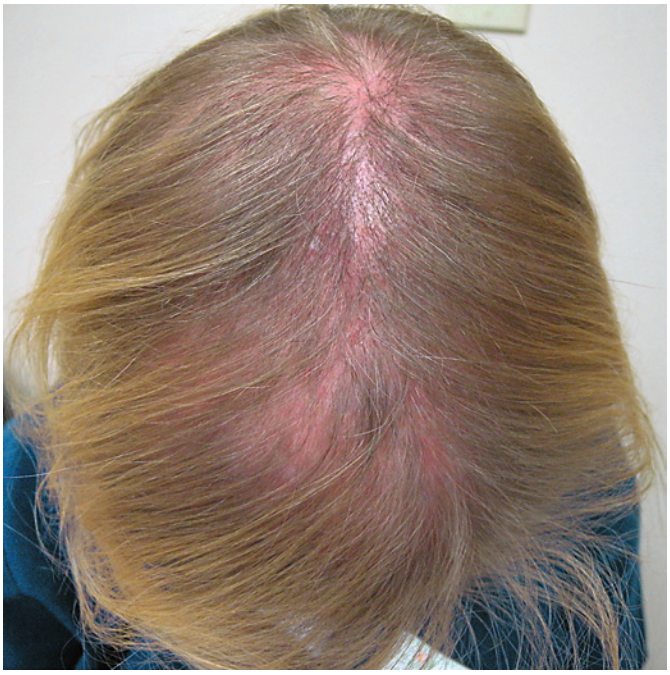

Fig. 4. Patient with dermatomyositis, presenting with scalp erythema and diffuse alopecia.

copy. Contact dermatitis often complicates other scalp disorders, as an interrupted skin barrier predisposes to sensitization. In a study of 1,320 German patients, the most common sensitizers in patients with scalp contact dermatitis were hair dyes (p-phenylenediamine, toluene-2,5-diamine, p-aminophenol, 3-aminophenol, p-aminoazobenzene, and pyrogallo) and cocamidopropyl betaine, which is a common detergent in shampoo [26]. Other common scalp allergens implicated in scalp contact dermatitis include fragrances and preservatives that are typical ingredients of shampoos, conditioners and hairstyling products. In patients whom contact dermatitis is suspected, it is also important to ask about the type of brush that is being used since contact dermatitis associated with nickel, rubber or plastic may be the cause of the problem [26].

Red Scalp Syndrome

The red scalp syndrome is characterized by scalp erythema associated with papules, pustules and telangiectasias (Fig. 3). Patients complain of itching, stinging, and burning. The disorder is considered a variant of rosacea localized to the scalp, which is characteristically resistant to treatment with topical steroids and responds to oral tetracyclines. The red scalp may be associated with androgenetic alopecia [27].

Tinea Capitis

Tinea capitis may cause severe pruritus, erythema, and scaling; alopecia may or may not be present. A prominent inflammatory reaction is typical for kerion lesion and presence of cervical node enlargement suggests diagnosis. Family history, school contacts, history of contact with pets with skin lesions, and the presence of tinea in other body parts of the patient and its family may prompt consideration of this diagnosis [28].

The most important pathogen that causes tinea capitis in the United States is *Trichophyton tonsurans*. In Europe, Brazil, and Mexico, *Microsporum canis*, and in Africa, and Eastern Europe *Trichophyton violaceum* are also common [29–31].

Wood's light may help to identify infection by *Microsporum audouinii* and *M. canis* by the presence of green fluorescence [32]. On dermoscopy, the combination of broken hairs, black dots, comma-shaped hairs and short corkscrew hairs are suggestive of tinea capitis [12]. KOH preparation may be performed; however, false negative results may occur in patients treated with topical or oral antifungals. For culture, the brushing technique is more effective than scraping. Fungal culture may be used for confirmation of diagnosis and species identification. Bi-

Fig. 5. Trichoscopy in a patient with dermatomyositis showing enlarged polymorphous interfollicular vessels, peripilar casts, and short regrowing hairs.

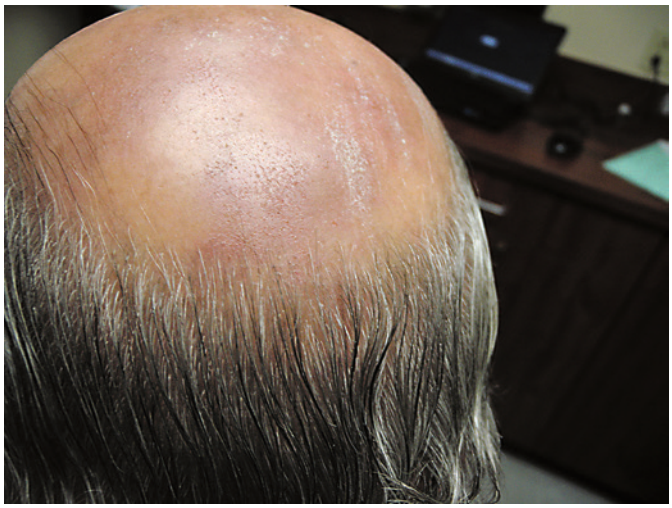

Fig. 6. Severe atopic dermatitis in a patient with androgenetic alopecia.

opsy with PAS stain may also be used to confirm the diagnosis [29].

Dermatomyositis

Dermatomyositis has been linked to pruritus in up to 94% of diagnosed patients [33]. Other scalp manifestations that may be present are erythema, atrophy, scales, and diffuse non-scarring alopecia [34] (Fig. 4). Acute telogen effluvium may also occur and may be diagnosed by history and a positive pull test. A significant decrease in the density of epidermal nerve fibers was found on confocal microscopic evaluation of a skin biopsy in a patient with dermatomyositis, scalp erythema, and scalp itch. This finding suggests that scalp pruritus in this disease may be caused by small fiber neuropathy [35]. Dermoscopic exam shows enlarged polymorphous interfollicular vessels that are very suggestive of the diagnosis [12] (Fig. 5). Other features that may be observed on physical exam include proximal muscle weakness, heliotrope rash, Gottron papules and Gottron sign, V-neck sign, shawl sign, periungual changes, mechanic hands, and photosensitivity [36].

Sensitive Scalp/Atopic Dermatitis

Sensitive scalp is a common condition, which may be related to other scalp diseases such as seborrheic dermatitis, psoriasis and contact dermatitis. In this condition, the scalp is prone to experiencing pruritus and paresthesias including prickling, burning, and tingling when exposed to physical, psychological or hormonal factors. An altered skin barrier has been described, but it has not been linked to a specific immune or allergic mechanism [37]. Diffuse erythema may or may not be present. Three tests that are useful to determine the presence of a sensitive scalp are the stinging, thermal sensitivity, and capsaicin tests [38]. Itch is present in 60% of patients with sensitive scalp. Hair loss has also been associated with scalp sensitivity [39].

The scalp is affected in 49.7% of the patients with atopic dermatitis, and scalp itching is one of the minor diagnostic criteria according to Hanifin and Rajka [40, 41]. Atopic dermatitis may present with erythema and xerosis clinically, and diagnosis may be confirmed by histopathology (Fig. 6).

Scalp Itch with Alopecic Patches

When assessing patients with alopecic patches who present with scalp itch, distinguishing between non-scarring and scarring alopecias is an important aspect of clinical evaluation. Non-scarring alopecias associated with scalp itch include alopecia areata and androgenetic alopecia. Patients with active alopecia areata often complain of

Table 1. Epidemiology, symptoms, clinical manifestations, dermoscopic findings, and histological features of the most common scarring alopecias associated with itch

	History	Symptoms and itching	Clinical features	Dermoscopy	Histological features
Lichen planopilaris (LPP)	Women > men Peak age between 30 and 60 years of age [79] Non-scalp lichen planus in 17–28% of patients with LPP [81]	Shedding/hair loss (100%), scale (80%), and scalp tenderness (72%) [82] Pruritus variable (54.34–90%) [79, 83, 84]	Whitish atrophic or scarring patches on the scalp with complete loss of follicular orifices [80] (Fig. 7)	Absence of follicular openings, peripilar casts, broken hairs Vellus hairs are usually absent [12] (Fig. 8)	Lichenoid dermatitis at the dermoepidermal junction surrounding the follicular infundibulum and the isthmus A perivascular infiltrate may be minimal, superficial and usually perifollicular Epidermal and dermal mucin is usually absent [85]
Frontal fibrosing alopecia	Primarily affects postmenopausal women [86]	Pruritus variable 35–67%[87–90] Burning and pain sensation 21% [89] Pain 17%, burning 8% [90]	Progressive frontotemporal hairline recession, perifollicular erythema, glabellar red dots perifollicular papules, eyebrow loss, eyelashes and body hair loss [87, 91] "Lonely hair" sign [92]	Absence of vellus hair, peripilar casts, broken hairs, "V sign" and black dots [12]	Same as LPP [82]
Discoid lupus	Women > men Young to middle age women (between 20 and 40 years) [81, 82]	Increased shedding (100%), pruritus (65%), and scalp tenderness (26.2%) [83]	Erythematous patches of alopecia, follicular hyperkeratosis, hyperpigmentation or loss of pigment and telangiectasias [81, 82]	Loss of follicular openings, white patches, peripilar casts, keratotic plugs, red dots, enlarged branching vessels [12]	Vacuolar interface alteration of the follicular epithelium with scattered dyskeratotic keratinocytes, cytoid bodies, and a variable dense periadnexal and interstitial lymphocytic infiltrate with dermal mucin Superficial and deep perivasculitis Sebaceous glands are atrophied or absent [82]
Central centrifugal cicatricial alopecia (CCCA)	Most common cause of permanent hair loss in African-Americans Middle-aged black females [85] May be inherited in an autosomal dominant pattern [93]	Symptoms may be trivial or absent: mild pruritus, pain, or tenderness may occur in involved areas [85] Positive correlation between CCCA severity score and peak itch ratings [94]	Flesh-colored, non-inflammatory cicatricial alopecia of the central scalp that enlarges centrifugally [82, 85]	Irregularly distributed pinpoint white dots and irregular white patches and peripilar white-gray halo surrounding the hairs within the patches [12]	Premature inner root sheath desquamation is a characteristic but nonspecific finding Active disease: perifollicular lymphocytic infiltrate surrounds the upper follicle; concentric lamellar fibroplasia occurs around mid- and upper follicles Advanced disease: perifollicular granulomatous inflammation and presence of hair shaft foreign-body giant cells End-stage disease: follicular fibrosis with retention of erector pili [82]

Table 1 (continued)

	History	Symptoms and itching	Clinical features	Dermoscopy	Histological features
Folliculitis decalvans	Young adults of both genders Slight male predominance [95]	Pruritus 68%, trichodynia 30% [95]	Scarring alopecic patches with follicular pustules, crusts and tufted hairs [95] Irregularly shaped, atrophic flesh-colored or ivory-white patches of cicatricial alopecia develop [82, 96] Predominantly involves the vertex and occipital area of the scalp [96]	Polytrichia, perifollicular erythema in a starburst pattern, yellowish tubular scaling, crusting and follicular pustules Chronic lesions: ivory-white and milky-red areas without follicular orifices [12]	Neutrophilic primary cicatricial alopecia [97] Active disease: acneiform infundibular dilatation; variably dense, intrafollicular and perifollicular neutrophilic infiltrate affects the upper and middle parts of the follicle Progression: the infiltrate composed of neutrophils, lymphocytes, and plasma cells extends into the adventitial dermis; granulomatous inflammation occurs Late-stage disease: follicular and adventitial dermal fibrosis [82]

mild itching or burning, which often precedes development of new patches. It may be caused by mast cell release of histamine and tryptase as well as lymphocytic infiltration with release of IL-31 [42]. Patients with androgenetic alopecia often complain of scalp itch and frequently have concomitant seborrheic dermatitis.

In active telogen effluvium, patients complain of trichodynia rather than scalp itch. In a study of 109 patients, 24 patients with androgenetic alopecia, 47 with telogen effluvium, 20 with both conditions, and 18 with telogen effluvium in remission it was concluded that trichodynia is almost exclusive of active telogen effluvium. Trichodynia was significantly more prevalent in the patients with telogen effluvium and those with both telogen effluvium and androgenetic alopecia (50.7%, $p < 0.001$), compared to those who had only androgenetic alopecia without telogen effluvium or telogen effluvium in remission (4.8%) [43].

The most common types of scarring alopecia associated with itch are LPP, frontal fibrosing alopecia, discoid lupus erythematosus, and central cicatricial centrifugal alopecia. Alopecia mucinosa and keratosis follicularis spinulosa decalvans are rare causes.

Table 1 summarizes epidemiology, symptoms, clinical manifestations, dermoscopic findings, and histological features of the most common scarring alopecias associated with itch (Fig. 7, 8).

Urticarial Lesions

In patients with urticarial lesions, consider urticaria, contact urticaria, urticarial vasculitis, and insect bites.

In patients with urticaria, the presence of urticarial lesions on other parts of the body and characteristic histopathological findings may aid the diagnosis. Scalp urticaria usually does not present with the classic findings of erythema and edema, because of the scalp's skin structure. Rather, patients complain of irritation, pruritus, stinging, or burning of the scalp [44, 45].

Contact urticaria occurs with interaction with an irritating/alergenic substance. It has been observed as an adverse effect of utilizing dinitrochlorobenzene (therapy for alopecia areata), ammonium persulfate (a component of peroxide hair bleach), bovine collagen (ingredient in hair conditioner), and henna (dye derived from plant) [46–50]. Contact urticaria with systemic symptoms has also been reported after application of hair conditioner containing bovine collagen [51]. It is therefore very important to ask about exposure to these substances when a patient presents with scalp itch. Awareness of the manifestations of contact urticaria is essential since it may result in more than local injury, with systemic anaphylaxis as a rare, though deadly, complication [52, 53].

The lesions of urticarial vasculitis with exclusive scalp location are extremely rare [54]. Papular urticaria is the urticarial reaction to an insect bite. It may be caused by

Fig. 7. Early lichen planopilaris in a patient with diffuse hair loss.

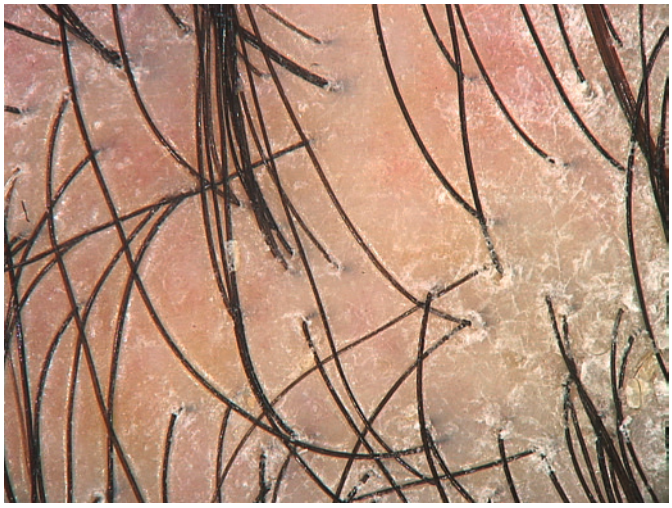

Fig. 8. Trichoscopy of the same patient with lichen planopilaris showing diffuse absence of follicular openings and peripilar casts.

arthropods such as fleas, mosquitoes, and bed bugs. These insects may bite on any body part. The scalp is not the most common location; however, papular urticaria must be considered in the presence of pruritic, erythematous, 3- to 10-mm papules, wheals, and/or vesicles and a history of exposure to bugs.

Infestations

Some patients with scalp itch may present with infestations, with the most common being lice and scabies. Scabies usually spares the scalp in adults, but may be present in this location in children and immunocompromised patients [55].

Pediculosis is a common condition with a worldwide distribution [56, 57]. Patients usually refer intense pruritus of the scalp due to an allergic reaction to the saliva that the parasite produces. Scalp itch takes around 4–6 weeks to manifest if it is the first infestation [58]. In subsequent infections, pruritus may take 24–48 h to develop. Other features of pediculosis and scalp pruritus may include hyperkeratosis, secondary bacterial infection, and cervical lymphadenopathy.

Observing the live louse or nits on the scalp or hair shaft is diagnostic. Head lice can be differentiated from other causes of scalp itching like psoriasis or seborrheic dermatitis, as they usually are associated with yellowish greasy scales. Nits must be distinguished from hair cast (pseudonits). The latter slide easily along the hair shaft in contrast to nits, which are attached firmly to the hair shaft [59–61].

Other Skin Lesions

Other more specific lesions that may be diagnosed clinically or with a biopsy are: seborrheic keratosis, keloid scars, burns, eosinophilic, polymorphic, and pruritic eruption associated with radiotherapy (EPPER), and rarely, skin tumors.

Keloid scars and burn lesions cause peripheral neuropathic itch. They are included in this section for the purpose of clinical classification. Approximately 86% of patients with keloids have itch, which commonly affects the borders of keloid, and pruritus affects 76% of patients with burn lesions [62, 63].

EPPER is a rare entity, which may present in the scalp when radiation has been used in this area. It may present with excoriations, erythematous papules, wheals, and rarely with vesicles, pustules and bullae [64].

Although malignant skin tumors are usually asymptomatic, primary cutaneous follicle center lymphoma was reported in one case as the cause of itchy scalp presenting with infiltrative erythematous plaques [65]. In a study of patients with folliculotropic mycosis fungoides, 58% had head/neck involvement, 73% presented with pruritus and one patient had scalp involvement with the presence of red nodules and alopecia. In this article, they associated pruritus with the presence of a prominent eosinophilic infiltrate [66]. In another case report, a patient with leukemia cutis presented with an itchy erythematous plaque, which was first treated as contact dermatitis without improvement and then achieved complete resolution of the lesion with primary radiation therapy [67].

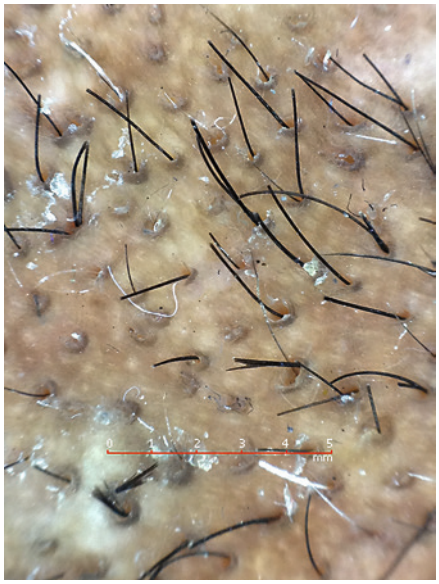

Fig. 9. Lichen simplex chronicus in a patient with chronic rubbing and scratching. Trichoscopy shows prominent follicular openings, empty follicles, broken hairs with trichorrhexis nodosa, and trichoptilosis.

Scalp Itch Associated with Systemic Disease

It is important to exclude systemic causes of scalp itch in patients without obvious dermatological lesions, in cases of secondary lesions that result from rubbing or scratching, such as lichen simplex chronicus, or in cases of lesions associated with systemic disease such as urticarial lesions and vascular or infiltrated plaques (Fig. 9). Several clinical manifestations and dermoscopic patterns, in an apparently normal scalp can lead us to suspect systemic pathologies. Table 2 lists systemic diseases that present with itch not limited to the scalp.

Autoimmune diseases that have been linked with scalp itch are dermatomyositis, systemic lupus erythematosus, scleroderma and Sjögren syndrome. Itch may or may not be associated with skin lesions. If present on dermoscopy, erythema and enlarged interfollicular vessels may aid the diagnosis.

Sarcoidosis may present diffusely with itching folliculitis-like lesions, which may be misdiagnosed as seborrheic dermatitis or as infiltrated asymptomatic plaques. Trichoscopic findings may vary depending on clinical presentation, but presence of yellow-orange spots is characteristic [68, 69].

Table 2. Systemic conditions that present with itching that is not limited to the scalp

Metabolic diseases
Chronic renal disease
Type 2 diabetes mellitus
Hyper-/hypothyroidism
Carcinoid syndrome
Abnormal parathyroid activity
Hepatobiliary causes
Biliary cirrhosis
Chronic pancreatitis
Drug-induced cholestasis
Hepatitis
Sclerosing cholestasis
Cholestasis of pregnancy
Amyloidosis with hepatic infiltration
Paraneoplastic
Hodgkin's lymphoma
Mycoses fungoides
Chronic leukemia, myelomatosis, lymphosarcoma
Solid tumors: lung, colon, brain, breast, gastric, pancreatic, prostate, or laryngeal
Hematological
Iron deficiency
Polycythemia vera
Hemochromatosis
Mastocytosis
Infectious
HIV
Hepatitis C virus infection
Other
Pharmacological
Pregnancy

Scalp Itch without Skin Lesions

Neuropathic Itch

Neuropathic itch is caused by an abnormality in the afferent pathway of the nervous system. It can be central or peripheral. Some skin lesions may also cause this type of itch. Neurological causes of pruritus should be suspected if a patient has sensory symptoms like paresthesia, hypoesthesia and hyperalgesia in the same area and there is no dermatological or systemic cause. In patients with neuropathic pruritus, a full neurological exam is required to test cranial nerve functions and sensations of temperature, touch, and pain. Consultation by a neurologist, EMG, and a nerve conduction study may be required. Also, an MRI may be needed if a brain tumor is suspected [70, 71].

Postherpetic itch is the prototype of peripheral neuropathic itch. Its incidence is underestimated by physicians and is often misdiagnosed as postherpetic neuralgia even though it is common, affecting at least one-third of the patients with herpes zoster. Although it was reported that herpes zoster involving the face and head more commonly have itch, a new multicentric study shows no difference in the severity of itch between different parts of the body affected with herpes zoster. Also, this study shows that the severity of pruritus is related to the severity of neuralgia [72, 73].

Diabetic patients usually have peripheral neuropathic itch, which can affect many sites such as the scalp. However, such scalp itch is relieved when the glucose level is controlled [74]. Keloid and burn scars are other examples of diagnoses associated with peripheral neuropathic itch, but they present with the characteristic skin lesions. Central causes of neuropathic itch are less frequent than peripheral causes. These include brain tumors, abscesses, strokes, and spinal cord tumor [71].

Psychogenic Pruritus

The most difficult part of the assessment of pruritus of the scalp is when a dermatologist does not find any dermatological, systemic, or neurological cause of such complaint. It generally takes several visits to a clinic to determine the cause of pruritus. During this time, the dermatologist must establish a trusting relationship with the patient and should be able to obtain a detailed social history, with questions about the patient's support system, occupation, stressors, and significant life events. In addition, the patient may have a personal or family history of psychiatric complaints or illness. Other parts of the patient's history that may be important include sleep and exercise habits, illicit drug use, and medications taken for mood concerns. This information will help the doctor clarify if there is a psychogenic cause of the pruritus as the itch intensity may vary depending on factors such as stress, illicit drug use, or compulsive behaviors.

Psychiatric consultation is suggested to confirm the diagnosis of psychogenic itch. Notably, when comparing between males and females, psychogenic itch is more prevalent in women, particularly between the age of 35 and 45 years [71]. The patient with psychogenic pruritus normally presents with a healthy scalp or with secondary lesions such as scratch marks and excoriations, without a primary lesion. It is worth mentioning that psychogenic causes may exist along with the dermatological or systemic causes of pruritus and this can make the itch worse. Additionally, chronic pruritus, which does not respond to treatment can cause depression and affect the quality of life of the patient.

In patients with psychiatric illness, scalp itching is common. A retrospective study involving patients with chronic pruritus shows that itching of the patient's face and scalp occurs more often with psychiatric than the non-psychiatric patient. The frequency of scalp and face itching is 16.7% in psychiatric patients, in comparison to 10.1% in non-psychiatric patients [75].

There are several psychogenic causes of pruritus. This includes, but is not limited to, anxiety, depression, obsessive compulsive disorders, schizophrenia, delusion of parasitosis, tactile hallucinations, and somatoform disorders [76, 77]. Stress can cause itching of the scalp, and that is why it is common to see people scratching their scalps when they are in a difficult situation. A community-based study involving 316 patients, assessed the relationship between stressful life events and skin symptoms. Pruritus was found to be the most reported symptom (69.3%) with the scalp being the most affected area in the body (59.5%) [78].

Conclusion

Itch is a common complaint in the dermatological setting. A dermatologist must use an algorithmic approach to investigate the possible cause of scalp pruritus. Also, it is important to think first about the most common causes and then rule out other, less common etiologies. The acronym SCALLP and the five steps for scalp evaluation are useful tools to keep in mind for an assertive approach in these patients.

Statement of Ethics

Verbal photographic and informed consent was obtained from the patients described in this article.

Disclosure Statement

Dr. Antonella Tosti serves in the following roles to the following organizations and/or companies. Consultant: P&G, DS Laboratories, and Fotofinder; Principal Investigator: Incyte; author royalties: Springer and Taylor & Francis; Editor-in-Chief: Karger Publishers; Scientific Board: National Alopecia Areata Foundation. All other authors have no conflicts of interest to disclose. This article has no funding source.

References

1 Tarikci N, Kocatürk E, Güngör F, Topal IGOL, Can PÜ, Singer R: Pruritus in systemic diseases: a review of etiological factors and new treatment modalities. ScientificWorldJournal 2015;2015:803752.
2 Green SC, Stewart ME, Downing DT: Variation in sebum fatty acid composition among adult humans. J Invest Dermatol 1984;83:114–117.
3 de Haan P, Heemskerk AE, Gerritsen A, de Boer EM, Sampat S, van der Raaij-Helmer EM, Bruynzeel DP: Comparison of toxicity tests on human skin and epidermoid (A431) cells using free fatty acids as test substances. Clin Exp Dermatol 1993;18:428–433.
4 Ito N, Sugawara K, Bodó E, Takigawa M, van Beek N, Ito T, Paus R: Corticotropin-releasing hormone stimulates the in situ generation of mast cells from precursors in the human hair follicle mesenchyme. J Invest Dermatol 2010;130:995–1004.
5 Botchkarev VA, Eichmüller S, Peters EMJ, Pietsch P, Johansson O, Maurer M, Paus R: A simple immunofluorescence technique for simultaneous visualization of mast cells and nerve fibers reveals selectivity and hair cycle – dependent changes in mast cell – nerve fiber contacts in murine skin. Arch Dermatol Res 1997;289:292–302.
6 Pincelli C, Fantini F, Giannetti A: Neuropeptides, nerve growth factor and the skin. Pathol Biol (Paris) 1996;44:856–859.
7 Shapiro J, Otberg N: Hair Loss and Restoration, ed 2. Boca Raton, CRC Press, 2015.
8 Lerebours-Nadal L, Beck-Sague CM, Parker D, Gosman A, Saavedra A, Engel K, Dean AG: Severe, disfiguring, pityriasis rubra pilaris in a woman in the Dominican Republic: histopathologic diagnosis and response to antiretroviral therapy. J Int Assoc Provid AIDS Care 2016;15:11–14.
9 Pakula AS, Paller AS: Langerhans cell histiocytosis and dermatophytosis. J Am Acad Dermatol 1993;29:340–343.
10 Moghadam-Kia S, Franks AG: Autoimmune disease and hair loss. Dermatol Clin 2013;31:75–91.
11 Misery L, Rahhali N, Duhamel A, Taieb C: Epidemiology of dandruff, scalp pruritus and associated symptoms. Acta Derm Venereol 2013;93:80–81.
12 Tosti A: Dermoscopy of the Hair and Nails, ed 2. Boca Raton, CRC Press, 2015.
13 Gupta MA, Gupta AK, Kirkby S, Weiner HK, Mace TM, Schork NJ, Johnson EH, Ellis CN, Voorhees JJ: Pruritus in psoriasis. A prospective study of some psychiatric and dermatologic correlates. Arch Dermatol 1988;124:1052–1057.
14 Szepietowski JC, Reich A, Wiśnicka B: Itching in patients suffering from psoriasis. Acta Dermatovenerol Croat 2002;10:221–226.
15 Reich A, Hrehorów E, Szepietowski JC: Pruritus is an important factor negatively influencing the well-being of psoriatic patients. Acta Derm Venereol 2010;90:257–263.
16 Yosipovitch G, Goon A, Wee J, Chan YH, Goh CL: The prevalence and clinical characteristics of pruritus among patients with extensive psoriasis. Br J Dermatol 2000;143:969–973.
17 Bilac C, Ermertcan AT, Bilac DB, Deveci A, Horasan GD: The relationship between symptoms and patient characteristics among psoriasis patients. Indian J Dermatol Venereol Leprol 2009;75:551.
18 Prignano F, Ricceri F, Pescitelli L, Lotti T: Itch in psoriasis: epidemiology, clinical aspects and treatment options. Clin Cosmet Investig Dermatol 2009;2:9–13.
19 Kim T-W, Shim W-H, Kim J-M, Mun J-H, Song M, Kim H-S, Ko H-C, Kim M-B, Kim B-S: Clinical characteristics of pruritus in patients with scalp psoriasis and their relation with intraepidermal nerve fiber density. Ann Dermatol 2014;26:727–732.
20 Carstens E, Akiyama T: Itch: Mechanisms and Treatment. Boca Raton, CRC Press Book, 2014.
21 Kim T-W, Shim W-H, Kim J-M, Mun J-H, Song M, Kim H-S, Ko H-C, Kim M-B, Kim B-S: Clinical characteristics of pruritus in patients with scalp psoriasis and their relation with intraepidermal nerve fiber density. Ann Dermatol 2014;26:727–732.
22 Hrehorów E, Salomon J, Matusiak L, Reich A, Szepietowski JC: Patients with psoriasis feel stigmatized. Acta Derm Venereol 2012;92:67–72.
23 Haliasos EC, Kerner M, Jaimes-Lopez N, Rudnicka L, Zalaudek I, Malvehy J, Hofmann-Wellenhof R, Braun RP, Marghoob AA: Dermoscopy for the pediatric dermatologist part I: dermoscopy of pediatric infectious and inflammatory skin lesions and hair disorders. Pediatr Dermatol 2013;30:163–171.
24 Miteva M, Tosti A: Hair and scalp dermatoscopy. J Am Acad Dermatol 2012;67:1040–1048.
25 Xu C, Chen D, Liu J, Liu Y, Sun Q: Roles of dermoscopy in the diagnosis and differential diagnosis of scalp psoriasis and seborrheic dermatitis (in Chinese). Zhonghua Yi Xue Za Zhi 2014;94:3467–3470.
26 Hillen U, Grabbe S, Uter W: Patch test results in patients with scalp dermatitis: analysis of data of the Information Network of Departments of Dermatology. Contact Dermatitis 2007;56:87–93.
27 Oberholzer PA, Nobbe S, Kolm I, Kerl K, Kamarachev J, Trüeb RM: Red scalp disease – a rosacea-like dermatosis of the scalp? Successful therapy with oral tetracycline. Dermatology 2009;219:179–181.
28 Silverberg NB: Scalp hyperkeratosis in children with skin of color: diagnostic and therapeutic considerations. Cutis 2015;95:199–204, 207.
29 Trovato MJ, Schwartz RA, Janniger CK: Tinea capitis: current concepts in clinical practice. Cutis 2006;77:93–99.
30 Rebollo N, López-Barcenas AP, Arenas R: Tinea capitis (in Spanish). Actas Dermosifiliogr 2008;99:91–100.
31 Ginter-Hanselmayer G, Weger W, Ilkit M, Smolle J: Epidemiology of tinea capitis in Europe: current state and changing patterns. Mycoses 2007;50:6–13.
32 Fuller L, Barton R, Mohd Mustapa MF, Proudfoot L, Punjabi S, Higgins E: British Association of Dermatologists' guidelines for management of tinea capitis 2014. Br J Dermatol 2014;171:454–463.
33 Shirani Z, Kucenic MJ, Carroll CL, Fleischer AB, Feldman SR, Yosipovitch G, Jorizzo JL: Pruritus in adult dermatomyositis. Clin Exp Dermatol 2004;29:273–276.
34 Kasteler JS, Callen JP: Scalp involvement in dermatomyositis. Often overlooked or misdiagnosed. JAMA 1994;272:1939–1941.
35 Hurliman E, Groth D, Wendelschafer-Crabb G, Kennedy W, Kavand S, Ericson M, Hordinsky M: Small-fibre neuropathy in a patient with dermatomyositis and severe scalp pruritus. Br J Dermatol 2017;176:209–211.
36 Muro Y, Sugiura K, Akiyama M: Cutaneous manifestations in dermatomyositis: key clinical and serological features – a comprehensive review. Clin Rev Allergy Immunol 2016;51:293–302.
37 Farage MA: Perceptions of sensitive skin: changes in perceived severity and associations with environmental causes. Contact Dermatitis 2008;59:226–232.
38 Misery L, Sibaud V, Ambronati M, Macy G, Boussetta S, Taieb C: Sensitive scalp: does this condition exist? An epidemiological study. Contact Dermatitis 2008;58:234–238.
39 Misery L, Rahhali N, Ambonati M, Black D, Saint-Martory C, Schmitt A-M, Taieb C: Evaluation of sensitive scalp severity and symptomatology by using a new score. J Eur Acad Dermatol Venereol 2011;25:1295–1298.
40 Shi M, Zhang H, Chen X, Guo Y, Tao J, Qi H, Gan J, Jiang A, Yu H, Liang J, Chen J, Yang L, Zhuang Y, Yan M, Gu Y, Yu X, Feng X, Yao Z: Clinical features of atopic dermatitis in a hospital-based setting in China. J Eur Acad Dermatol Venereol 2011;25:1206–1212.
41 Hanifin JM, Raika G: Diagnostic features of atopic dermatitis. Derm Venereol Suppl 1980;92:44–47.
42 Yamakoshi T, Andoh T, Makino T, Kuraishi Y, Shimizu T: Clinical and histopathological features of itch in patients with alopecia areata. Acta Derm Venereol 2013;93:575–576.
43 Baldari M, Montinari M, Guarrera M, Rebora A: Trichodynia is a distinguishing symptom of telogen effluvium. J Eur Acad Dermatol Venereol 2009;23:733–734.

44 Zhai H, Zheng Y, Fautz R, Fuchs A, Maibach HI: Reactions of non-immunologic contact urticaria on scalp, face, and back. Skin Res Technol 2012;18:436–441.
45 O'goshi K, Iguchi M, Tagami H: Functional analysis of the stratum corneum of scalp skin: studies in patients with alopecia areata and androgenetic alopecia. Arch Dermatol Res 2000;292:605–611.
46 McDaniel DH, Blatchley DM, Welton WA: Adverse systemic reaction to dinitrochlorobenzene. Arch Dermatol 1982;118:371.
47 Fisher AA, Dooms-Goossens A: Persulfate hair bleach reactions. Cutaneous and respiratory manifestations. Arch Dermatol 1976; 112:1407–1409.
48 Pasche-Koo F, Claeys M, Hauser C: Contact urticaria with systemic symptoms caused by bovine collagen in a hair conditioner. Am J Contact Dermat 1996;7:56–77.
49 Uehara S, Inomata N, Suzuki A, Matsuura M, Aihara M: Severe contact urticarial syndrome due to oxidative hair dye containing para-aminophenol and sodium-methyl-oleoyl-taurate. J Dermatol 2014;7:560–561.
50 Majoie IML, Bruynzeel DP: Occupational immediate-type hypersensitivity to henna in a hairdresser. Am J Contact Dermatitis 1996;7: 38–40.
51 Pasche-Koo F, Claeys M, Hauser C: Contact urticaria with systemic symptoms caused by bovine collagen in a hair conditioner. Am J Contact Dermatitis 1996;7:56–57.
52 Geo yon Krogh C, Maibach HI: The contact urticaria syndrome – an updated review. J Am Acad Dermatol 1981;5328–342:328–342.
53 Burla MJ, Brody AM, Welch RD, Favot MJ: Anaphylactic reaction after ongoing exposure to hair glue: a novel case report. J Emerg Med 2015;48:e5–e7.
54 Berg RE, Kantor GR, Bergfeld WF: Urticarial vasculitis. Int J Dermatol 1988;27:468–472.
55 Lewis EJ, Connolly SB, Crutchfield CE, Rest EB: Localized crusted scabies of the scalp and feet. Cutis 1998;61:87–88.
56 Chosidow O: Scabies and pediculosis. Lancet 2000;355:819–826.
57 Mimouni D, Ankol OE, Gdalevich M, Grotto I, Davidovitch N, Zangvil E: Seasonality trends of pediculosis capitis and *Phthirus pubis* in a young adult population: follow-up of 20 years. J Eur Acad Dermatol Venereol 2002; 16:257–259.
58 Devore CD, Schutze GE: Head lice. Pediatrics 2015;135:e1355–e1365.
59 Di Stefani A, Hofmann-Wellenhof R, Zalaudek I: Dermoscopy for diagnosis and treatment monitoring of pediculosis capitis. J Am Acad Dermatol 2006;54:909–911.
60 Zalaudek I, Argenziano G: Images in clinical medicine. Dermoscopy of nits and pseudonits. N Engl J Med 2012;367:1741.
61 Haliasos EC, Kerner M, Jaimes-Lopez N, Rudnicka L, Zalaudek I, Malvehy J, Hofmann-Wellenhof R, Braun RP, Marghoob AA: Dermoscopy for the pediatric dermatologist part I: dermoscopy of pediatric infectious and inflammatory skin lesions and hair disorders. Pediatr Dermatol 2013;30:163–171.
62 Gauffin E, Öster C, Gerdin B, Ekselius L: Prevalence and prediction of prolonged pruritus after severe burns. J. Burn Care Res 2015; 36:405–413.
63 Lee S, Yosipovitch G, Chan Y, Goh CL: Pruritus, pain, and small nerve fiber function in keloids: a controlled study. J Am Acad Dermatol 2004;51:1002–1006.
64 Lee DJ, Kim YC: A pustular form of eosinophilic, polymorphic, and pruritic eruption associated with radiotherapy. J Am Acad Dermatol 2011;65:e51–e53.
65 McCrary WJ, Hurst MD, Hiatt KM, Singh ZN, Wirges ML: Acute alopecia with underlying pruritic erythema. J Am Acad Dermatol 2015;73:893–894.
66 Lehman JS, Cook-Norris RH, Weed BR, Weenig RH, Gibson LE, Weaver AL, Pittelkow MR: Folliculotropic mycosis fungoides: single-center study and systematic review. Arch Dermatol 2010;146:607–613.
67 Stang K, Alite F, Steber J, Emami B, Surucu M: Leukemia cutis of the face, scalp, and neck treated with non-coplanar split field volumetric modulated arc therapy: a case report. Cureus 2015;7:e430.
68 Pellicano R, Tiodorovic-Zivkovic D, Gourhant J-Y, Catricalà C, Ferrara G, Caldarola G, Argenziano G, Zalaudek I: Dermoscopy of cutaneous sarcoidosis. Dermatology 2010;221:51–54.
69 Torres F, Tosti A, Misciali C, Lorenzi S: Trichoscopy as a clue to the diagnosis of scalp sarcoidosis. Int J Dermatol 2011;50:358–361.
70 Yosipovitch G, et al: Itch: Basic Mechanism and Therapy. Boca Raton, CRC Press, 2004, 213–239.
71 Yo GI, Sipovitch L, Samuel L: Neuropathic and psychogenic itch. Dermatol Ther 2008; 21:32–41.
72 Oaklander AL, Bowsher D, Galer B, Haanpää M, Jensen MP: Herpes zoster itch: preliminary epidemiologic data. J Pain 2003;4:338–343.
73 Lee H-J, Kim G-W, Kim W-J, Mun J-H, Song M, Kim H-S, Ko H-C, Kim M-B, Kim B-S: Clinical characteristics of postherpetic pruritus: assessment using a questionnaire, von Frey filaments and Neurometer. Br J Dermatol 2015;172:1672–1673.
74 Scribner M: Diabetes and pruritus of the scalp. JAMA 1977;237:1559.
75 Ferm I, Sterner M, Wallengren J: Somatic and psychiatric comorbidity in patients with chronic pruritus. Acta Derm Venereol 2010; 90:395–400.
76 Ständer S, Weisshaar E, Mettang T, Szepietowski JC, Carstens E, Ikoma A, Bergasa NV, Gieler U, Misery L, Wallengren J, Darsow U, Streit M, Metze D, Luger TA, Greaves MW, Schmelz M, Yosipovitch G, Bernhard JD: Clinical classification of itch: a position paper of the international forum for the study of itch. Acta Derm Venereol 2007;87:291–294.
77 Rishnan A, Koo J: Psyche, opioids, and itch. Dermatol Ther 2005;18:314–322.
78 Gupta MA, Gupta AK: Stressful major life events are associated with a higher frequency of cutaneous sensory symptoms: an empirical study of non-clinical subjects. J Eur Acad Dermatol Venereol 2004;18:560–565.
79 Lyakhovitsky A, Amichai B, Sizopoulou C, Barzilai A: A case series of 46 patients with lichen planopilaris: demographics, clinical evaluation, and treatment experience. J Dermatolog Treat 2014;6634:1–5.
80 Kang H, Alzolibani AA, Otberg N, Shapiro J: Lichen planopilaris. Dermatol Ther 2008;21: 249–256.
81 Otberg N: Primary cicatricial alopecias. Dermatol Clin 2013;31:155–166.
82 Ross EK, Tan E, Shapiro J: Update on primary cicatricial alopecias. J Am Acad Dermatol 2005;53:1–37; quiz 38–40.
83 Tan E, Martinka M, Ball N, Shapiro J: Primary cicatricial alopecias: clinicopathology of 112 cases. J Am Acad Dermatol 2004;50:25–32.
84 Chieregato C, Zini A, Barba A, Magnanini M, Rosina P: Lichen planopilaris: report of 30 cases and review of the literature. Int J Dermatol 2003;42:342–345.
85 Whiting DA: Cicatricial alopecia: clinicopathological findings and treatment. Clin Dermatol 2001;19:211–225.
86 Kossard S: Postmenopausal frontal fibrosing alopecia. Scarring alopecia in a pattern distribution. Arch Dermatol 1994;130:770–774.
87 Vañó-Galván S, Molina-Ruiz AM, Serrano-Falcón C, Arias-Santiago S, Rodrigues-Barata AR, Garnacho-Saucedo G, Martorell-Calatayud A, Fernández-Crehuet P, Grimalt R, Aranegui B, Grillo E, Diaz-Ley B, Salido R, Pérez-Gala S, Serrano S, Moreno JC, Jaén P, Camacho FM: Frontal fibrosing alopecia: a multicenter review of 355 patients. J Am Acad Dermatol 2014;70:670–678.
88 Dlova NC, Jordaan HF, Skenjane A, Khoza N, Tosti A: Frontal fibrosing alopecia: a clinical review of 20 black patients from South Africa. Br J Dermatol 2013;169:939–941.
89 Banka N, Mubki T, Bunagan MJK, McElwee K, Shapiro J: Frontal fibrosing alopecia: a retrospective clinical review of 62 patients with treatment outcome and long-term follow-up. Int J Dermatol 2014;53:1324–1330.
90 Samrao A, Chew A-L, Price V: Frontal fibrosing alopecia: a clinical review of 36 patients. Br J Dermatol 2010;163:1296–1300.
91 Ladizinski B, Bazakas A, Selim MA, Olsen EA: Frontal fibrosing alopecia: a retrospective review of 19 patients seen at Duke University. J Am Acad Dermatol 2013;68:749–755.

92 Tosti A, Miteva M, Torres F: Lonely hair: a clue to the diagnosis of frontal fibrosing alopecia. Arch Dermatol 2011;147:1240.
93 Dlova NC, Jordaan FH, Sarig O, Sprecher E: Autosomal dominant inheritance of central centrifugal cicatricial alopecia in black South Africans. J Am Acad Dermatol 2014;70:679–682.
94 Bin Saif GA, McMichael A, Kwatra SG, Chan YH, Yosipovitch G: Central centrifugal cicatricial alopecia severity is associated with cowhage-induced itch. Br J Dermatol 2013;168:253–256.
95 Vañó-Galván S, Molina-Ruiz AM, Fernández-Crehuet P, Rodrigues-Barata AR, Arias-Santiago S, Serrano-Falcón C, Martorell-Calatayud A, Barco D, Pérez B, Serrano S, Requena L, , Grimalt R, Paoli J, Jaén P, Camacho FM: Folliculitis decalvans: a multicentre review of 82 patients. J Eur Acad Dermatol Venereol 2015;29:1750–1757.
96 Otberg N, Kang H, Alzolibani AA, Shapiro J: Folliculitis decalvans. Dermatol Ther 2008;21:238–244.
97 Olsen EA, Bergfeld, Cotsarelis WFG, Price VH, Shapiro V, Sinclair R, Solomon A, Sperling L, Stenn K, Whiting DA, Bernardo O, Bettencourt M, Bolduc C, Callendar V, Elston D, Hickman J, Ioffreda M, King L, Linzon C, McMichael A, Miller J, Mulinari F, Trancik R; Workshop on Cicatricial Alopecia: Summary of North American Hair Research Society (NAHRS)-sponsored Workshop on Cicatricial Alopecia, Duke University Medical Center, February 10 and 11, 2001. J Am Acad Dermatol 2003;48:103–110.