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Infections, Infestations and Neoplasms of the Scalp

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Abstract

This chapter reviews common cutaneous infections, infestations, and neoplasms of the scalp. Infections of the scalp are subdivided into three major groups. The most seen are: (1) Bacterial: Folliculitis, folliculitis decalvans, tufted hair folliculitis and acne keloidalis nuchae. (2) Fungal: Tinea capitis, favus and kerion celsi. (3) Protozoal: Syphilitic alopecia. Pediculosis capitis is the most common worldwide infestation of the scalp. The neoplasms of the scalp are large group of different diseases due to arising different origin. In the following section, trichilemmal cyst, proliferating trichilemmal cyst, nevus sebaceous and cylindroma are discussed in detail.

Keywords: infection, infestation, neoplasm, scalp

1. Introduction

Scalp diseases are one of the most seen reasons for admission to dermatology clinics. There is a wide spectrum of different aetiologies for scalp lesions. This chapter will review cutaneous infections, infestations and neoplasms of the scalp. Examples of bacterial infections include folliculitis, folliculitis decalvans, tufted hair folliculitis and acne keloidalis nuchae. There will be some detailed information about fungal infections, such as tinea capitis, favus and kerion celsi. The neoplasms of the scalp represent a varied group of dermatoses. This review focuses on trichilemmal cyst, proliferating trichilemmal cyst, nevus sebaceous and cylindroma.

2. Bacterial infections of scalp

There are several types of infections of the scalp including chronic scalp folliculitis [SF], folliculitis decalvans [FD], tufted folliculitis [TF], acne nuchae keloidalis [ANK] and dissecting

cellulitis [DCS]. These infections have similar features like chronic scarring folliculocentric pustules localised to the scalp. Many of these conditions show the presence of *Staphylococcus aureus* [SA] and response to antibiotic therapy [1].

2.1. Scalp folliculitis

Folliculitis is a pyoderma that begins within the hair follicle. It is classified according to the microbial aetiology, including bacteria, viruses and fungi, as well as many other non-infectious ones. The most seen folliculitis of scalp are *Staphylococcus aureus* [*S. aureus*] folliculitis, Herpes simplex virus folliculitis and dermatophytic folliculitis [Tinea capitis]. Some of the predisposing factors are hyperhidrosis, maceration, friction, overweight, medications such as corticosteroids and halogenated compounds, as well as occlusive hair care products and topical hydrocarbons, such as oils and tars. In addition, immunodeficiencies such as HIV/AIDS and diabetes mellitus are also predisposed to folliculitis [2].

S. aureus is the most common cause of folliculitis. The major cause is either contagion or auto-inoculation from a carrier focus, usually nasal or perianal region. The typical lesion of folliculitis is a small inflamed, dome-shaped papule or pustule which can be drained spontaneously. Both pruritus and pain can be seen. Systemic symptoms, such as fever or lymphadenopathy, may occur when the involvement is widespread. Biopsy is rarely needed to distinguish between fungal or viral folliculitis. Biopsy shows neutrophils in the dermis and follicular wall damage. Many of the patients may carry *S. aureus*, so nasal and perineal cultures should be taken. Other forms of folliculitis can be identical in appearance, so this possibility should always be taken into consideration. Treatment options include topical antibiotic or disinfectant solutions such as mupirocin cream, triclosan [2%] or chlorhexidine [1%] and antistaphylococcal systemic antibiotics [1, 2].

2.2. Folliculitis decalvans

Folliculitis decalvans [FD] is a rare type of cicatricial alopecia and was first described by Quinquaud in 1881 [3]. It is an inflammatory disease characterised by follicular pustules and haemorrhagic crusting, leading to scarring hair loss. Although in most patient FD started in the vertex, other sites such as the occipital or midscalp area may also be affected. At the periphery of lesion, follicular pustules continue to form. Tufting of hairs may be seen [4]. Clinically, the majority of the patients were generally healthy, without any systemic symptoms or any signs of immunosuppression. Although the exact cause remains unknown, *S. aureus* is usually cultured from these pustules [5]. The disease shows a chronic and relapsing course. Histologically, early lesions show dense perifollicular inflammatory infiltrates consisting mostly of neutrophils. In later stages, follicular rupture, lymphocytes, histiocytes and plasma cells are seen, as well as perifollicular and interstitial dermal fibrosis [6, 7]. Differential diagnosis of FD consists of follicular degeneration syndrome or central centrifugal scarring alopecia.

As FD is usually associated with infection of *S. aureus* [5, 6], systemic antibiotics are the mainstay of disease [e.g., cephalexin, minocycline, tetracycline, clindamycin, rifampicin, ciprofloxacin].

Because of its high lipid solubility, and it is said to be the best antistaphylococcal antibiotic, rifampicin has been successfully used in combination with various other antibiotics [5, 7]. Rifampicin is not recommended for lone use. Rifampicin 300 mg b.i.d and clindamycin 300 mg b.i.d is the preferred regimen. Shampooing with antibacterial wash products and topical corticosteroids may also be useful. Varying results have been reported after treatment with prednisolone, isotretinoin, human immunoglobulin and more recently biologics [e.g., infliximab and adalimumab] [8, 9], neodymium: yttrium aluminium-garnet [Nd:YAG] laser [10] and photodynamic therapy [PDT] [11]. There is very limited evidence that FD can be treated with dapsone, minoxidil or radiation therapy [12].

2.3. Tufted hair folliculitis

Tufted hair folliculitis [THF] was first described by Smith and Sanderson in 1978 [7]. It is characterised by scarring bacterial folliculitis of the scalp associated with multiple bundles of hair emerging from a single dilated follicular orifice in a 'doll's hair' pattern. Infection with *S. aureus* is thought to be initial causative factor [13]. Tufting of hair is caused by clustering of adjacent follicular unit due to a fibrosing process and to retention of telogen hairs within a dilated follicular orifice [14]. The patients were 20–60 years old, the peak incidence occurring in 30 years [4]. It affects male more frequently than female.

Clinically, it presents erythematous, infiltrated plaque of cicatricial alopecia and enlarged follicular openings with tufts containing 20–30 apparently normal hair shafts (**Figure 1**). The lesions are usually found in the occipital and parietal areas [8]. Frequently reported subjective symptoms are pruritus, pain and scales adherent to the scalp and hair. Regional lymph node enlargement [occipital, pre- or retroauricular] may also be noticed [9]. *S. aureus* is most often cultured from the lesions [10]. Underlying differences in follicular anatomy or host response may be responsible for the lesion [15]. Histopathological studies reveal scarring with perifollicular inflammation of plasma cells, lymphocytes and neutrophils around the upper portions of the follicles sparing at the hair root level. Multiple hairs are seen emerging from a single follicular opening (**Figure 2**) [13].



Figure 1. Cicatricial alopecia with tufted folliculitis.

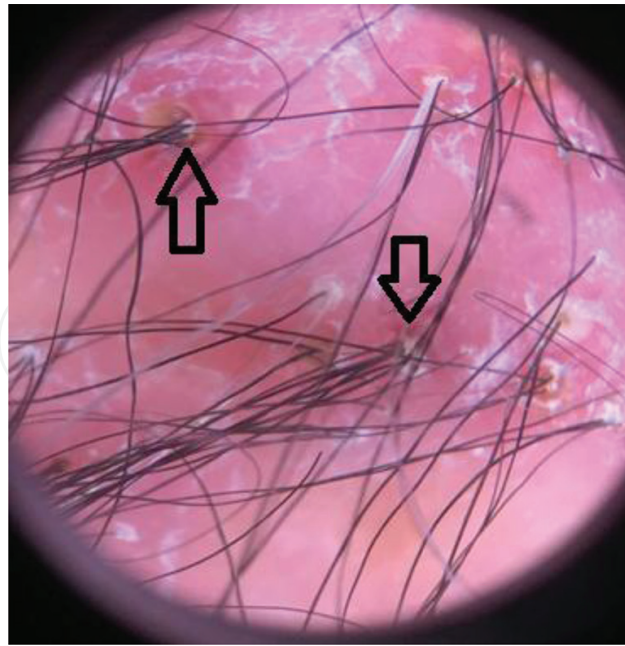


Figure 2. Dermoscopic image of the scalp with tufted folliculitis characterised by multiple hairs emerging from one single dilated follicular orifice.

THF may be seen with dissecting cellulitis of the scalp, folliculitis decalvans, acne keloidalis, Melkersson-Rosenthal syndrome and hidradenitis suppurativa [16]. In some case reports it has been described that tufted folliculitis in association with medication use, specifically with cyclosporine and lapatinib [17, 18]. Differential diagnosis consists of folliculitis decalvans, folliculitis keloidalis nuchae, kerion celsi, dissecting cellulitis of scalp, trichostasis spinulosa, follicular lichen planus and relapsing staphylococcal folliculitis [19].

The course of THF is chronic and the patient may experience intermittent flares and remissions. Treatment of this relapsing condition is notoriously difficult. As *S. aureus* is the initial causative agent, systemic antibiotics including ciprofloxacin, erythromycin, flucloxacillin and amoxicillin/clavulanic acid are regarded the standard therapy. Rifampicin and nadifloxacin have been proven as more effective than other therapeutic modality to control the pustular phase of the disease, of the best antibiotics active against *S. aureus*, as well as to prevent possible recurrences [4, 20]. Rifampicin can be used with a dose of 450 mg twice per day for 4 weeks or 600 mg daily for 10 weeks [20, 21]. Recently, a case of tufted hair folliculitis being treated with trastuzumab, a selective HER2 inhibitor, has been reported [22]. Good results with excision of the areas of scarring have also been described [13, 15].

2.4. Acne keloidalis nuchae

Acne keloidalis nuchae [AKN] is a chronic scarring folliculitis characterised by fibrotic, keloid-like papules and plaques on the occipital scalp and posterior neck. The term acne keloidalis was given to this condition in 1872 by Bazin [23].

Early AKN lesions are seen as mildly pruritic papules and pustules arranged in irregularly linear groups just below the hairline. With continued inflammation or infection the

papules tend to coalesce and form hypertrophic scars or keloids that may be painful and disfiguring (**Figures 3 and 4**). In advanced cases, abscesses and sinus tracts with purulent discharge may develop [24, 25]. Actually AKN is not a form of acne vulgaris and unlike true acne vulgaris, comedones are not a feature of AKN. It is very common in individuals of African descent. Its prevalence ranges from 1 to 16% and the male to female ratio is at least 20:1. The probable onset age of AKN is 15–25 years and reduces after 55 years of age [26].



Figure 3. Acne keloidalis nuchae. Papules are seen below the hairline.



Figure 4. Acne keloidalis nuchae. Papules and hypertrophic scars are noticed on the occipital region.

The exact aetiology of AKN is unclear but it is associated with several factors including androgen excess, chronic mechanical trauma such as close haircuts and chronic rubbing of

the area by clothing stimulating an inflammatory reaction, secondary bacterial infection, mast cell density and medications such as antiepileptic drugs or cyclosporine [24, 26–28].

Histological studies show evidence of follicular and perifollicular infiltrate at the upper one-third of the hair follicle in early lesions, whereas more advanced lesions reveal disrupted hair follicles, a foreign-body reaction with granulomatous inflammation and fibrotic dermis [25, 29]. Differential diagnosis of AKN consists of the other chronic scarring folliculocentric pustules localised to the scalp.

First step in treatment is patient education. It should be advised that the patient should avoid from mechanical irritation from clothing for prevention. Prognosis becomes good if the treatment begins at early stage. However, once major scarring develops, therapy is more difficult and morbidity is increased. If pathogenic microorganism with culture are identified, appropriate antibiotics should be prescribed. Conventional treatment modalities usually involve use of topical, intralesional or systemic steroids in combination with retinoids and/or oral antibiotics such as doxycycline or minocycline to decrease inflammation. Oral isotretinoin of 20 mg daily may be used alone or in combination with topical fusidic acid and oral cefadroxil [500 mg twice daily for 2 weeks] to treat the patient [30]. Other treatment options are cryotherapy and targeted ultraviolet B [290–320 nm] phototherapy. Combination of cryotherapy and intralesional steroid may help to reduce the size and firmness of papules and nodules [31]. Radiation therapy and intralesional 5-fluorouracil are alternative treatment strategies for refractory cases [32]. Recently, laser treatment such as CO₂ laser, 1064-nm Nd:YAG laser, 59-nm pulse dye laser [PDL] and 810-nm diode laser have been used which allow for 82–95% improvement in one to five sessions [33]. Patients who present with big fibrotic nodules would benefit most from surgical excision. Excision with primary closure may be used for excellent cosmetic results for the management of extensive cases of AKN [23, 28, 34].

3. Fungal infections of scalp

3.1. Tinea capitis

Tinea capitis (TC) is a disease caused by dermatophytes of the skin of the scalp with a propensity for attacking hair shafts and follicles. It occurs predominantly in pre-pubertal children aged between 3 and 7 years. It is reported more in boys than in girls within pre-pubertal age. Tinea capitis is the most seen pediatric dermatophyte infection worldwide [35]. All species of *Trichophyton* and *Microsporum* can cause TC. *Microsporum canis* is the cause of the most seen dermatophyte worldwide, whereas in the United States *Trichophyton tonsurans* is the most common organism. Transmission occurs by direct head-to-head contact through fomites, from animals to humans, and, least commonly, acquired from soil. Asymptomatic carriers of *Trichophyton tonsurans* are common, making it difficult to treat [36].

From the site of inoculation, the fungal hyphae grow centrifugally in the stratum corneum. According to the patterns of contamination, tinea of the scalp is classified into two types: ectothrix and endothrix. In an ectothrix infection, the fungi continue downward

growth into the follicle and they invade the keratin part of the hairs. Ectothrix invasion is usually associated with *Trichophyton verrucosum*, *Trichophyton mentagrophytes* and all *Microsporum* species. The endothrix hair invasion caused by *Trichophyton tonsurans* and *Trichophyton violaceum* is characterised by the development of the fungi within the hair shaft only. While the ectothrix-infected hairs can be of fluoresce bright green or yellow green caused by the destruction of the cuticle of the hair, the endothrix infection do not fluoresce, because the cuticle of the hair remains intact in endothrix infection. The hair is very fragile and breaks the surface of the scalp. Therefore, leaves the infected dark stubs visible in the follicular orifices. Thus, endothrix infection is often described as a 'black dot' appearance [37].

Among predisposing factors for dermatophyte infections are humid environment, atopic diathesis, such as cell-mediated immune deficiency, systemic immunocompromised states, prolonged immunosuppression with the use of topical glucocorticoids and broad-spectrum antibiotic use [38].

Typical clinical features are small areas of fine scale with minimal hair loss. 'Moth-eaten' alopecia is seen. When *Trichophyton tonsurans* is responsible, black dots are seen. Erythema is also a clue for *Microsporum canis*. Shedding of fungal spores may continue several months despite active treatment; therefore, children with tinea capitis may attend school. Also, short haircuts and wearing a cap during treatment are not necessary. Clinical manifestations of TC may resemble pityriasis amiantacea, seborrheic dermatitis, bacterial folliculitis, pediculosis capitis, trichotillomania, alopecia areata or pustular psoriasis, which are often treated incorrectly [39].

Definitive diagnosis of TC is made through 10–20% potassium hydroxide [KOH] examination and culture from lesions. The turn-around time for culture may take several weeks. Wood light examination is also helpful to demonstrate fungal fluoresce. Infected hair produces bright green or yellow green fluorescence. It should be kept in mind that endothrix organisms do not fluoresce. Trichoscopy is an additional tool for the diagnosis of tinea capitis. Comma shaped hairs, corkscrew hairs and zigzag shaped hairs are the diagnostic trichoscopic features of tinea capitis [40, 41]. Skin biopsy can be done for differential diagnosis. Fungal hyphae in the stratum corneum can be demonstrated histopathologically.

As the dermatophyte penetrate the hair follicle, oral antifungals is required for the treatment of TC. Basically, three different groups of systemic antifungals are used to eliminate TC: Griseofulvin, azoles [itraconazole, fluconazole, ketoconazole] and allylamines [terbinafine]. Of these agents, itraconazole and terbinafine are most commonly used. Griseofulvin [10–25 mg/kg/day for 6–8 weeks] is the most frequently used antifungal agent, but recently it has also been reported that newer agents such as terbinafine [10–20 kg: 62.5 mg/day; 20–40 kg: 125 mg/day; >40 kg: 250 mg/day for 2–4 weeks], itraconazole [5 mg/kg/day for 2–6 weeks] and fluconazole [first dose 6–12 mg/kg, then 3–6 mg/kg/day for 2–4 weeks] to be effective. Selenium sulphide, zinc pyrithione, povidone iodine or ketoconazole shampoos have been shown to help to decrease the shedding of fungal spores. Common recommendations are to use these shampoos two to four times weekly for 2–4 weeks [42, 43].

3.2. Favus

Favus or tinea favosa is the most severe form of tinea capitis. It is caused by *Tricophyton schoenleinii*. The disease frequently occurs in children and is seen rarely in adults. If untreated, the disease persists forever. Favus is seen almost exclusively in Africa, the Mediterranean and the Middle East and, rarely, in North America and South America [44].

The most common clinical manifestations on the scalp are yellowish cup-shaped crusts termed scutula, which surround the infected hair follicles. The scutula have an unpleasant mousy odour. Besides the scalp, it may involve glabrous skin, hairy regions and nails. If not treated properly, the lesion advances peripherally and it can leave scarring alopecia [45, 46].

The diagnosis is confirmed by direct mycological examination and culture. A greyish-green fluorescence may be observed with Wood's lamp examination. Optical microscopy with KOH preparation shows invasion by the fungus, hyphae parallelly arranged to the axis, air spaces and a few spores.

In presence of scaly patches without alopecia, favus is misdiagnosed as seborrheic dermatitis, psoriasis, tinea amiantacea or lichen planus [47, 48].

The treatment of tinea favosa lies on the combination of an oral and topical antifungal agents. The local treatment consists in cutting of hair around the alopecia patches and applying once or twice a day of antifungal imidazol [shampoo, foam gel, lotion and spray]. Griseofulvin, terbinafine and itraconazole could be used in systemic therapy [44, 45].

3.3. Kerion

Kerion celsi [KC] [so-called deep tinea capitis] is an uncommon inflammatory presentation of tinea capitis [TC], which appears as a boggy, large inflammatory painful mass studded with broken hairs, pustules and, often, purulent drainage from its surface (**Figure 5**). Hair loss is frequently seen in KC. It is usually solitary but multiple lesions may be found. Reactive lymphadenopathy, especially cervical or suboccipital, is a very common associated feature. KC often occurs in children but it has been described in elderly patients [49–51]. The higher

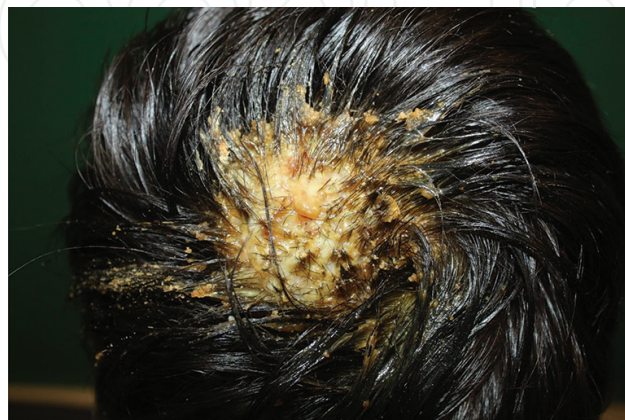


Figure 5. Boggy, large inflammatory painful plaque lesion is seen.

prevalence in females may be related to fact that they generally have longer hair [52]. KC is a markedly inflammatory type of TC secondary to a vigorous host immune response. It is generally caused by zoophilic dermatophytes [*Microsporum canis* and *Trichophyton mentagrophytes*], but also by anthropophilic [*Trichophyton rubrum*] and rarely by geophilic [*Microsporum gypseum*] species. KC is thought to be the result of a hypersensitivity reaction to dermatophytes [49].

The main source of the fungi responsible for KC is from humans or animals, though dermatophytes may spread via fomites [combs, hairbrushes, hats and contaminated wearing materials]. Although the gold standard diagnostic method is fungal culture, conventional sampling of a kerion can be difficult. Negative results are not uncommon in these cases. The diagnosis of kerion is usually made clinically. A moistened standard bacteriological swab taken from the pustular areas and inoculated onto the culture plate may yield a positive result [53].

Id reactions [so-called dermatophytid] are noted in patients with KC. This is an often pruritic dermatitis based on sensitisation to fungal antigens, reported in 4–5% of fungal infection. Although it may mimic an allergic reaction, it should not lead to discontinuation of antifungal treatment [54]. Acute vesicular dermatitis of the hands and feet is the most common type of id reaction. Other less common types of id reactions include annular erythema and erythema nodosum. These patients have a strong delayed-type hypersensitivity [DTH] reaction to intradermal trichophytin. EN is thought to be due either the deposition of immune complexes in capillaries and venules of the dermal and adipose plexus, or to a DTH reaction to an antigen [55, 56].

The principal differential diagnoses consist of impetigo and bacterial or sterile folliculitis or abscesses. KC is usually misdiagnosed as bacterial abscesses. However, bacterial infections do not cause alopecia, and hairs plucked from a kerion are painless. Diagnostic errors causes patients to undergo unnecessary surgery or antibiotic treatment. However, there is little evidence to support the use of antibiotics for severe KC [49]. Kerion celsi requires treatment with systemic antifungals to penetrate the affected hair shafts. Early short course of glucocorticosteroids with a dose of 1 mg/kg/day were often used in severe KC to reduce inflammation. Oral steroid is tapered to withdraw in 10 days. Manual pressure to remove pus from sinuses was an adjuvant therapy to systemic oral antifungal agents for severe KC.

4. Protozoal infection of scalp

4.1. Syphilitic alopecia

Syphilis is a sexually transmitted disease caused by *Treponema pallidum*. Syphilitic alopecia [SA] is an uncommon feature of secondary syphilis with an incidence of 2.9–11.2% [57]. The physical examination findings include numerous non-scarring, non-inflammatory, irregular in size without defined borders, 'moth-eaten' patches of alopecia of the scalp [58]. The eyebrows and beard may also be involved. SA can be seen with other mucocutaneous symptoms of secondary syphilis. Hair loss usually occurs late in the secondary syphilis, about 8–12 weeks after the first signs of secondary syphilis [59].

According to the patterns of McCarthy made in 1940, secondary SA is classified into two types: symptomatic SA and essential SA. The other cutaneous manifestations of syphilis is not seen in essential SA. Essential SA, characterised by alopecia without any other visible syphilitic lesions on the scalp, may appear as one of three different clinical patterns: 'moth-eaten' alopecia, diffuse alopecia and mixed pattern of alopecia [58, 60]. The 'moth-eaten' pattern alopecia [alopecia syphilitica] is considered the most common and characteristic form of secondary syphilis [61, 62].

The diagnosis of SA is confirmed by both patient's sexual history and positive serological tests for RPR and TPPA. The histopathology findings of SA usually include a normal epidermis with areas of follicular hyperkeratosis. While the number of anagen follicles are reduced markedly, the number of catagen and telogen follicles increase. A perivascular and perifollicular [especially in the peribulbar region] lymphocytic dermal infiltration with scattered plasma cells is observed in some cases [63].

The differential diagnosis of 'moth-eaten' alopecia includes alopecia areata, trichotillomania and tinea capitis except from syphilis [64]. Under trichoscopy black dots, focal atrichia, hypopigmentation of hair shaft and yellow dots are observed in the hair loss region. Alopecia areata, tinea capitis and trichotillomania differ from SA with absence of exclamation hair, coma hair, flame hairs or v-sign, respectively [57].

The antisyphilitic treatment with a weekly dose of 2.4 million units of benzathine penicillin for 3 weeks or procaine penicillin 600,000 units i.m. daily for 10 days leads to complete resolution of alopecia. Hair growth is observed about 6–12 weeks after the start of the treatment [57, 65, 66].

5. Infestations of scalp

5.1. Pediculosis capitis

Pediculosis capitis [PC] [head lice] is a major worldwide infestation caused by *Pediculus humanus capitis* seen in school-aged children of 3–12 years of age. The prevalence of PC is usually higher in girls and women and varies greatly from country to country. It is 0.7–59% in Turkey, 0.48–22.4% in Europe, 37.4% in England, 13% in Australia, up to 58.9% in Africa and 3.6–61.4% in the Americas [67]. There are estimates that 6–12 million children in the United States are infected with PC annually [68]. The prevalence in 5318 elementary school children, aged 8–16 years in Mersin, Turkey, was 6.8% [69]. In another study of 1569 school children, aged 7–14 years, the prevalence of head lice was 16.6% [70]. It tends to be more prevalent in children because they have a high incidence of head-to-head contact with other children. Girls were at a greater risk for head lice because of their tendency to have longer hair than boys and social behaviour [close contact].

The lice are spread by direct head-to-head contact as well as by the sharing of clothing, headgear, hats, combs, hairbrushes, hair barrettes and pillows. PC affects all socioeconomic classes. Although direct contact with an infested individual can cause PC, personal and environmental hygiene are not risk factors for PC [71].

The head louse [plural-lice] is an ectoparasite whose only host is humans for survival. There are three stages that comprise the life cycle of the louse: egg, nymph and adult. Adult female louse lays their eggs [nits] on hair shafts. The nits are usually laid close to scalp for warmth, because they must stay warm in order to hatch. These nits cannot be moved along the hair shaft in contrast to pseudonits. Only in warmer climates, nits can be found 15 cm or more from the scalp, especially favour the nape of the neck. They are of size 0.8 mm × 0.3 mm, oval and usually yellow to white and are located 6 mm from scalp. Nits take about 1 week to hatch [range 6–9 days]. The nit hatches and release a single nymph. Nymph becomes an adult after three molts, about 7–10 days after hatching. Each instar molts every 3–4 days, and after the third molt, it becomes adult louse (**Figure 6**). The first and second instar or nymph forms are relatively immobile. Mobile forms are the third instar forms and adults form. The mature adult louse is approximately the size of a sesame seed [2–3 mm] and is tan to greyish-white [68, 72, 73]. Female lice are larger than males and must take blood before copulation. After copulation she lays between 5 and 10 eggs a day. The adult males usually do not survive after copulation. The adult louse lives only up to 36 hours away from its host. Head lice can travel up to 23 cm/min by crawling. The head lice do not attach firmly to smooth surfaces [e.g., glass, metal, plastic, synthetic leathers] [72, 74].

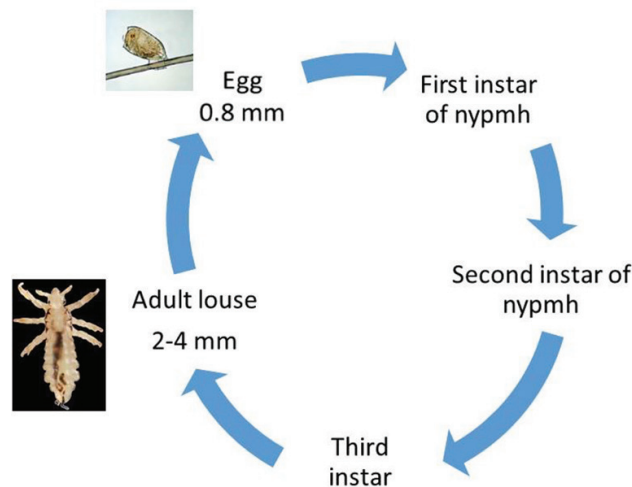


Figure 6. Schematic representation of life cycle of head louse.

The head louse takes a blood meal [hematophagia] usually 4–5 times per day. Chronic and heavy lice infestation can rarely lead to anaemia, especially in females. Pruritus is the most common complaint and is believed to be the result of a hypersensitivity reaction to the saliva of lice. On examination, nits are seen that firmly attached to hair shaft within 6 mm from the scalp skin especially in the occipital pit [louse pit] and retro-auricular areas. Scratches on the skin may lead to secondary bacterial infection and impetiginisation. Serous purulent discharge may result in the formation of a plica [plica polonica or plica neuropathica]. There are innumerable nits and live lice in patients with plica polonica. Cervical lymphadenopathy and conjunctivitis may also be seen. Lice infestation can cause allergic reactions within the nasal cavity manifested by nasal obstruction and rhinorrhea [72, 74, 75].

The diagnosis of head lice infestation is made through finding viable eggs [nits], nymphs and live adult lice. For the diagnosis, the use of a louse comb is more efficient than direct visual examination of the scalp [76]. Examining suggestive particles under the microscope may be helpful to confirm the diagnosis. Non-contact dermoscopy is also a useful instrument for differentiate nymph-containing eggs from empty cases or pseudonits [72]. Histology is rarely required for diagnosis. Examination of a louse bite reveals intradermal haemorrhage and a deep, wedge-shaped infiltrate with many eosinophils and lymphocytes.

Differential diagnosis of head lice includes seborrheic scales, hair casts [inner root sheath remnants], pityriasis amiantacea, white piedra, black piedra, impetigo, pili torti and monilethrix. It should be kept in mind that, in contrast to nits, hair casts are freely movable along hair shaft [72].

Basically, therapeutic wet combing, topical application of a pediculicide and oral treatment [Trimethoprim-sulfamethoxazole and ivermectin] are used to eliminate head lice. Wet combing is a mechanical removal of the lice. The patients must comb their wet hair with a fine-toothed comb every 3–4 days for a total of 2 weeks. If all young lice are combed out a few days after hatching, the infestation can be eradicated completely. Pediculicides are the most effective treatment for head lice. They can be divided into three types. Pediculicides with neurotoxic mode of action are permethrin [Nix®], pyrethrin [Rid®], malathion [Ovide®], carbaryl, lindan [Kwell®] and spinosad [Natroba®]. Dimeticones [Nyda®, Hedrin®], isopropyl myristate [Resultz®] and 1,2-octanediol are the pediculicides with physical mode of action. The other group of pediculicides is plant-based pediculicides. All topical preparations are used for two or three applications, 1 week apart [68, 71].

Because permethrin and pyrethrin are non-ovicidal, they should be reapplied on days 7 and 13–15. Permethrin 1% is a synthetic pyrethroid and is approved for use in children aged 2 months or older. It shows neurotoxic effect by inhibiting the sodium ion flux through nerve cell membrane channels [68]. It should be applied on damp unconditioned hair for 10 min and then rinsed off. Pruritus, erythema and edema are its usual side effects [77]. Pyrethrin is used for children aged 2 years and older. Malathion is a cholinesterase inhibitor. It is approved for use in individuals aged 6 years or older. Both malathion and lindan are ovicidal. Spinosad is ovicidal, killing both eggs and lice and also kills permethrin-resistant populations of lice. It is approved in patients aged 4 years and older [78]. Oral ivermectin is administered a single dose of 200 µg/kg and repeated in 10 days. It is restricted to children older than 5 years and weighing at least 15 kg [71].

Resistance of lice to the pediculocides is an important problem. Spinosad, benzyl alcohol 5% or malathion 0.5% may also be used, in case of resistance, for those older than 6 and 24 months, respectively. Another treatment option is manual removal of nits [especially the ones within 1 cm of the scalp]. It is recommended after treatment with any product. Besides these treatments, occlusive agents such as petroleum jelly, vinegar, isopropyl alcohol, olive oil, mayonnaise and melted butter can be used once per week for 3 weeks to suffocate the lice [68, 78].

Patients with head lice should have laundered potential fomites [e.g., towels, pillowcases, sheets, hats, toys] with hot water [at least 130°F/55°C] and then dried in a dryer using the

hottest cycle. For items that are not machine washable, dry-cleaning may be an effective alternative or storing for 2 weeks in a plastic bag. Children should also be educated not to share combs, brushes, hair accessories and towels and to avoid head-to-head contact [72, 73, 78].

6. Neoplasms of scalp

The neoplasms of the scalp consist of large group of different diseases due to arising different origin. They can be divided into two groups: benign and malignant. They can be categorised as shown in **Table 1**. The common benign swellings of the scalp are the epidermoid cysts, trichilemmal cysts, lipomas, dermoid cyst, pilomatrixoma, steatocystoma multiplex and cylindroma. In this section, neoplasms especially which is most seen and is arising from scalp are discussed in detail.

| Benign neoplasms | Malignant neoplasms |
|---|---------------------------------|
| Epidermoid cysts | Basal cell carcinoma |
| Trichilemmal cyst | Squamous cell carcinoma |
| Proliferating trichilemmal cyst | Melanoma |
| Seborrheic keratosis | Proliferating trichilemmal cyst |
| Lipoma | Angiosarcoma |
| Dermoid cyst | Cutaneous lymphoma |
| Pilomatrixoma | Cutaneous metastases |
| Steatocystoma multiplex | |
| Actinic keratosis | |
| Nevus sebaceous | |
| Melanocytic nevus | |
| Cylindroma | |
| Angiolymphoid hyperplasia with eosinophilia | |
| Neurothekeoma | |
| Infantile myofibromatosis | |
| Hemangioma | |

Table 1. The neoplasms of the scalp.

6.1. Trichilemmal cyst

Trichilemmal cyst [TC] also known as ‘pilar cyst’ is an adnexal tumour that arises from outer root sheath of the hair follicle. It is almost always benign but may be locally aggressive. Malignant transformation is very rare but may lead to distant metastases [77, 79].

It affects 5–10% of the population with a female preponderance [77, 80]. TC may be sporadic or they may be inherited as an autosomal dominant trait. It presents as an asymptomatic or mildly painful firm nodule but frequently they are multiple. The cysts are classically located in areas bearing hair follicle, mostly on the scalp and rarely the face, trunk and extremities may be involved. They are usually 1–2 cm in size but can be much larger.

Histopathologic evaluation can confirm the diagnosis. Histopathologically TCs are lined by stratified squamous epithelial cells and consist homogenous eosinophilic material that frequently show foci of calcification and abrupt keratinisation without an intervening granular layer.

TC may be misdiagnosed as epidermal cyst clinically but unlike epidermal cyst, no punctum is seen. Treatment is by complete excision. Trichilemmal cysts typically can be extracted more easily than epidermoid cysts [77, 79, 80].

6.2. Proliferating trichilemmal cyst

Proliferating trichilemmal cyst [PTC] also known as proliferating pilar tumour is a rare neoplasm arising from the isthmus region of the outer root sheath of the hair follicle. It can be inherited in an autosomal dominant pattern. It occurs most commonly on the scalp in women older than 50 years. Most tumours arise within a pre-existing trichilemmal cyst but some PTCs arise *de novo* [81]. They have different clinical and histologic features from trichilemmal cyst. PCTs may slowly or rapidly grow into a large, solitary, well-circumscribed painless mass overlying alopecia. The size may range from 1 to 10 cm, although there have been reports of lesions exceeding 25 cm in diameter. Inflammation, ulceration, bleeding and/or yellowish discharge may occur and may be clinically confused with squamous cell carcinoma. They usually behave in a benign nature but malignant transformation with local invasion and metastasis has also been described [82, 83].

Broad anastomosing bands and lobules of squamous epithelium in the cyst wall are the histologic features of PCT. PTC shows features of typical trichilemmal cyst, but additionally shows extensive epithelial proliferation, variable cytologic atypia and mitotic activity. Complete excision is recommended for the treatment of PCT owing to their potential for locally aggressive behaviour and malignant transformation [81, 83]. If malignant PTC with multi-nodal metastasis is identified, wide local excision with 1-cm margins should be performed, followed by adjuvant chemotherapy and radiation to prevent recurrence [84].

6.3. Nevus sebaceous

Nevus sebaceous [NS] is uncommon hamartomatous lesions that exhibits epidermal, follicular, sebaceous and apocrine malformation to varying degrees. It was first described by Jadassohn in 1895. It occurs most commonly on the head and neck while similar lesions elsewhere on the body are termed verrucous epidermal nevi. It often presents at birth with an incidence of 0.5–1% [85]. The characteristic clinical feature is a well-circumscribed, flesh-coloured, bald patch on the scalp at birth. At puberty, lesion grows proportionally with the patient and tends to be slightly yellow or orange, thick, velvety and verrucous surface. Familial cases have been reported [86].

Development of benign or malignant adnexal neoplasms may occur in NS, usually in adulthood. Syringocystadenoma papilliferum and trichoblastoma are the most common benign tumours arising in NS [87]. Other benign tumours include trichilemmoma, sebaceous adenoma, apocrine adenoma and poroma. Malignant transformation can develop in 10–15% of lesions in some series and basal cell carcinoma is the most seen malignant tumour. Actually, this rate is less than 1% of cases. Development of six different tumours has also been reported in one solitary lesion [88].

Histologically, early lesions show few abnormal features. A few misshapen hair follicles and small apocrine glands may be identifiable during childhood. At adolescence, as the lesions clinically thicken, NS looks like an epidermal nevus, with papillated epidermal hyperplasia, abortive hair follicles and enlarged apocrine glands.

The lesions must be distinguished from aplasia cutis congenita, congenital nevi, epidermal nevus syndrome and seborrheic keratosis. Although there is no consensus regarding the NS, increasing to the possibility of malignant tumour outgrowth from a NS with age, it is necessary to prophylactic excision especially in adulthood. Photodynamic therapy with topical aminolevulinic acid is another treatment option for non-surgical treatment [89].

6.4. Cylindroma

Cylindroma is a benign skin adnexal tumour differentiating towards either the eccrine or apocrine line. It has two different clinical presentations, solitary and multiple. Solitary cylindroma is a slow growing, benign tumour effecting the scalp especially the capillitium and neck region. Solitary cylindromas affect middle-aged and elderly persons and female-to-male ratios are 6:1 and 9:1 [90].

Multiple cylindromas are very rare and inherited in an autosomal dominant pattern by mutations in *CYLD1*, a tumour suppressor gene. They frequently occur on the scalp and rarely on the trunk. The term turban tumour is used to describe multiple cylindromas. Since the nodules enlarge and coalesce on the scalp, the patient looks as wearing a turban. Multiple cylindromas should suggest of Brook-Spiegler syndrome, which has been characterised by the development of multiple skin appendage tumours, such as cylindromas, trichoepitheliomas and spiradenomas [91, 92].

Histopathology is diagnostic with islands of basaloid cells in a jigsaw puzzle-like pattern separated by hyaline basement membrane material that is PAS positive. Eosinophilic PAS-positive hyaline material in globules is also seen within the lobules [93].

Differential diagnosis of cylindroma includes trichilemmal cysts, basal cell carcinoma, spiradenoma and dermatofibroma. Under dermatoscopy, cylindromas demonstrates arborising telangiectasia and scattered white globules on a background of white to salmon-pink. Vascular branches can be seen at the periphery which may extend into the centre of the lesion [94].

For solitary lesions, the treatment option is complete excision. Other treatments include electrodesiccation/curettage, cryotherapy and carbon dioxide laser. The patients with multiple cylindromas should follow up because of the tendency for new lesions to develop [95].

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