Parasitic Diseases and Infestations of the Hair and Scalp

8

Ralph M. Trüeb, Maria Fernanda Reis Gavazzoni Dias, and Hudson Dutra Rezende

Contents

First used in the English language in 1539, the word parasite comes from the Medieval French parasite, from the Latin parasitus, the latinization of the Greek παράσιτος, meaning "one who eats at the table of another," i.e., from παρά (para), "beside, by" + σῖτος (sitos), and "wheat," hence "food."

In contemporary popular language, the word parasitism has a derogatory sense. In everyday speech, a parasite is a sponger, a lazy profiteer, and a drain on society. However, in the Classical era, the concept of the parasite was not strictly pejorative: the parasitus was an accepted role in Roman society, in which a person could live

R. M. Trüeb
Dermatologische Praxis und Haarcenter Professor Trüeb, Wallisellen, Switzerland
e-mail: r.trueeb@derma-haarcenter.ch

M. F. R. Gavazzoni Dias
Dermatology, Universidade Federal Fluminense, Niterói, Rio de Janeiro, Brazil

H. Dutra Rezende (✉)
Centro Universitário Lusíada, São Paulo, São Paulo, Brazil
e-mail: contato@hudsondutra.com.br

R. M. Trüeb et al. (eds.), *Hair in Infectious Disease*,
https://doi.org/10.1007/978-3-031-30754-6_8

off the hospitality of others, in return for flattery, simple services, and a willingness to endure humiliation. In English writer and physician John William Polidori's (1795–1821) original Gothic horror novel *The Vampyre* (1816), Lord Ruthven is the prototype and antecedent to Bram Stoker's Dracula (1897). As a thief, seducer, creator, and mimic, the eponymous Count is the ultimate parasite. The whole point of vampirism is sucking other people's blood, living at other people's expense [1]. Finally, revolting and horrifying parasitic alien species have become widespread in modern science fiction, with Swiss surrealist painter H.R. Giger (1940–2014) winning an Academy Award for Best Achievement for Visual Effects for his respective design work on the movie *Alien* (1979).

Parasitism represents a close and persistent long-term biological interaction between the parasite and its host. Unlike saprophytes, parasites feed on living hosts, and unlike commensalism, the parasitic relationship harms the host, either feeding on it or consuming some of its food.

Because parasites interact with other species, they can also act as vectors of pathogens, transmitting specific infectious diseases.

Parasitism has an extremely wide taxonomic range, including protozoans, animals, fungi, and plants. Many bacteria are parasitic, though they are more generally thought of as pathogens causing disease [2]. Viruses are obligate intracellular parasites, characterized by extremely limited biological function, lacking all the usual machinery of the cell such as enzymes, relying entirely on the host cell's ability to replicate DNA and synthesize proteins.

Parasitism is a major aspect of evolutionary ecology. As hosts and parasites evolve together, their relationships often change. When a parasite is in a sole relationship with a host, selection drives the relationship to become more benign, even mutualistic, as the parasite can reproduce for longer if its host lives longer. But where parasites are competing, selection favors the parasite that reproduces fastest, leading to increased virulence. There are thus varied possibilities in host-parasite coevolution.

Parasites can exploit their hosts to carry out a number of functions that they would otherwise have to carry out for themselves. Parasites which lose those functions then have a selective advantage, as they can divert resources to reproduction. Many insect ectoparasites including bedbugs, lice, and fleas have lost their ability to fly, relying instead on their hosts for transport.

The sensory inputs that a parasite employs to identify and approach a potential host are known as host cues. Such cues can include, for example, exhaled carbon dioxide, skin odors, visual and heat signatures, and moisture [3].

Hosts have evolved a variety of defensive measures against their parasites, including physical barriers like the skin and the reaction of the immune system. Once inside the body, parasites must overcome the immune system's serum proteins and pattern recognition receptors, intracellular and cellular, that trigger the adaptive immune system's lymphocytes such as T cells and antibody-producing B cells. The skin reactions to parasites can be either due to irritant effects of the contents of the parasite's saliva or the results of development of an immunologically mediated response to parasitic antigens. Secondary bacterial infections of scratch

excoriations on the skin are common, especially in warm humid climates, and depending on hygiene.

Entomology from Ancient Greek ἔντομον "insect" and -λογία "study of" is the scientific study of insects. Like other fields that are categorized within zoology, entomology is a taxon-based category. Any form of scientific study in which there is a focus on insect-related inquiries is, by definition, entomology.

The discipline of medical entomology, or public health entomology, and also veterinary entomology is focused upon insects and arthropods that impact human health. Veterinary entomology is included in this category, because many animal diseases can jump species and become a human health hazard. Medical entomology also includes scientific research on the behavior, ecology, and epidemiology of arthropod disease vectors and involves a tremendous outreach to the public, including local and state officials and other stake holders in the interest of public safety. There are many insects that affect human health. They can parasitize, bite, sting, and cause allergic reactions and/or vector disease to humans. Medical entomologists worldwide are working to combat the known effects in order to improve public health.

Dermatological entomology represents the branch of medical entomology that applies to the skin and its appendages [4]. Entodermoscopy is the term that has been proposed for the use of the dermatoscope in the diagnosis of skin infestations [5] although as yet dermoscopy is of minor importance in the diagnosis and management of infections and infestations of the skin and hair. Examples have been scabies, tungiasis, larva migrans, ticks, lice, myiasis, and urticating hairs of the tarantula. With some sarcasm indeed, trichological entomology could be proposed to include the science of insects as they relate to diseases of the hair and scalp and entodermoscopy, underlining the problem of extreme specialization in medicine with its propensity for neologisms of little practical value, while as dermatologists and physicians, we should rather maintain a broad view on health and disease.

Personal pests such as lice, fleas, bedbugs, ticks, and scabies mites may vector pathogens. They are hematophagous, meaning they feed on the blood of their host. Nearly all personal pests can be transmitted to an uninfected host with prolonged exposure to an infected host. Lice, fleas, bedbugs, and ticks are known as ectoparasites. Ectoparasites live on the skin of their host. They have adaptations that allow them to access the nutrients inside of the host, such as methods to penetrate skin, insert digestive enzymes, and a gut microbiome that can digest the nutrients received from the host. While these ectoparasites feed, the transfer of fluids may transmit diseases such as typhus, plague, and Lyme disease.

8.1 Lice

Lice are hematophagous, wingless insects. Pediculosis, the infestation of the human with lice, has been documented for thousands of years. They are very host-specific; therefore human lice are not transmitted to or from pets or other animals. There are three species of lice that infest humans with particularities in body size, form, and

preferred regions of infestation: *Pediculus humanus humanus* the body or clothing louse; *Pediculus humanus capitis*, the head louse; and *Pthirus pubis*, the pubic or crab louse.

The anticoagulant together with a vasodilator in lice saliva produces irritation and pruritus in most individuals. Lice, like most insects, harbor symbiotic bacteria. In the body lice, these produce necessary B vitamins specifically nicotinic acid, pantothenic acid, and biotin to meet the louse's nutritional requirements. Therefore, body lice are capable of inhabiting a host that is nutritionally deficient. However, the symbiotes of head lice do not possess these nutritional-enhancing capabilities; therefore head lice seek out healthy, well-nourished children as host.

Unfortunately, body lice have erroneously given all human lice a connotation of filth and poor hygiene. In fact, head lice prefer clean and healthy heads.

Finally, body lice are vectors of diseases such as epidemic typhus (*Rickettsia prowazekii*). Epidemics occurred routinely throughout Europe from the 16th to the 19th centuries, particularly during the English Civil War, the Thirty Years' War, and the Napoleonic Wars, and accounted for a significant proportion of casualties among the combatants. In fact, during Napoleon's retreat from Moscow in 1812, more French soldiers died of typhus than were killed by the Russians [6]. Also, trench fever caused by *Bartonella (rochalimaea) quintana* is transmitted by the body louse, and louse-borne relapsing fever (LBRF) is caused by *Borrelia recurrentis*. Lice prefer a narrow range of temperature and will leave a febrile person in search of another individual with a more suitable microclimate. Thus, there is an additional risk of migration of lice from the sick to the healthy. In the case of death of the host, lice quickly leave the cooling body in search of a new host.

The role of head lice and pubic lice as transmitters of disease has not been scientifically confirmed. However, head lice have been shown to transmit *Staphylococcus aureus* and group A *Streptococcus pyogenes* resulting in pyoderma of the scalp. In these cases, treatments with antibiotics are only temporary cures unless the causative agent, the louse, is effectively eradicated.

8.1.1 Head Lice

Head lice infest all levels of society and most ethnic groups. The mechanisms of transmission and the habits of head lice differ between cool and warm climates. Maximum egg production occurs at optimum temperatures of 29–30 °C with an ample supply of food. For these reasons it is commonly held that most eggs are laid close to the scalp.

Robert Hooke's 1667 book, *Micrographia: Or Some Physiological Descriptions of Minute Bodies Made by Magnifying Glasses with Observations and Inquiries Thereupon*, illustrated a human louse (Observation 54), drawn as seen down an early microscope (Fig. 8.1a). Lice, like all other insects, have six legs. These project from the fused segments of the thorax, are short and terminate with a single claw and opposing "thumb". Between its claw and thumb, the louse grasps the hair of its

host. With their short legs and large claws, lice are well adapted to clinging to the hair of their host.

Body lice and head lice (Fig. 8.1b, c) are almost identical in appearance except for size. Head lice are shorter and narrower across the abdomen than body lice. Adult head lice are small (2.5–3 mm long), dorsoventrally flattened (see anatomical terms of location), and wingless. The thoracic segments are fused but otherwise distinct from the head and abdomen, the latter being composed of seven visible segments. One pair of antennae, each with five segments, protrudes from the insect's head. Head lice also have one pair of eyes. Head louse mouthparts are highly adapted for piercing the skin and sucking blood. These mouth parts are retracted into the insect's head except during feeding.

Like most insects, head lice are oviparous. Females lay about three or four eggs per day. Louse eggs, also known as nits, are attached near the base of a host hair shaft. Eggs are usually laid on the base of the hair, 3–5 mm off the scalp surface.

The nits (Fig. 8.1d, e) are oval-shaped and about 0.8 mm in length. They are bright, transparent, and tan to coffee-colored so long as they contain an embryo but appear white after hatching. Head lice hatch typically 6–9 days after oviposition. After hatching, the louse nymph leaves behind its egg shell, still attached to the hair shaft. The empty egg shell remains in place until physically removed by abrasion or the host, or until it slowly disintegrates, which may take 6 or more months.

The nape of the neck and behind the ears are their favorite places because these areas are more protected from extreme temperatures. Pruritus is the primary complaint. The intense itching forces the host to scratch, often excoriating the skin (Fig. 8.1f). Secondary bacterial infections are common.

Lice have no wings or powerful legs for jumping, so they move using the claws on their legs to move from hair to hair. Normally, head lice infest a new host only by close contact between individuals, making social contacts among children and parent-child interactions more likely routes of infestation than shared combs, hats, brushes, towels, clothing, beds, or closets. Head-to-head contact is by far the most common route of lice transmission. The number of children per family; the sharing of beds and closets; hair washing habits; local customs and social contacts; healthcare in a particular area, such as school; and socioeconomic status were found to be significant factors in head louse infestation. Girls are two to four times more frequently infested than boys. Children between 4 and 14 years of age are the most frequently infested group. Children with long- and medium-length hair are more often infested than children with short hair [7]. Finally, there appears to be an unknown element that causes some children to be more prone to repeated infestations than others. Whether blood type [8] or scalp microbiomata [9] plays a role is as yet to be further elucidated.

Many options for the treatment of head lice infestation exist. Nonprescription topical permethrin and pyrethrin are available. Prescription topical products include lindane, malathion, benzyl alcohol, carbaryl, and ivermectin.

Although treatment options are available, many have become less efficacious due to emergence of resistance with regional variety of prevalence and types of resistance.

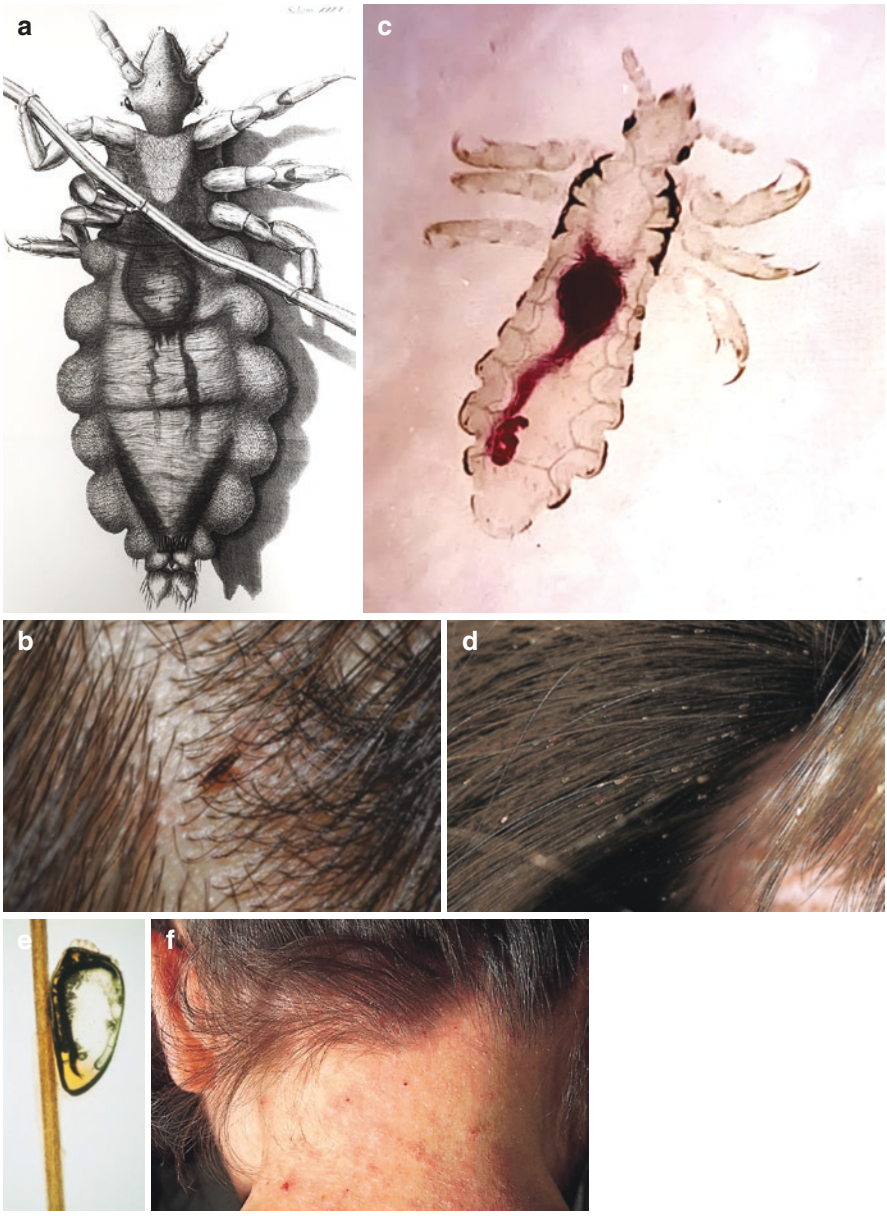

Fig. 8.1 (**a**–**c**) Pediculosis capitis: (**a**) the head louse, as originally drawn by Robert Hooke (from facsimile of the 1667 book *Micrographia* in the author's personal library), (**b**) head louse (courtesy Prof. Fábio Francesconi, Federal University of Amazonas, Brazil), (**c**) magnified, (**d**) nits attached to the hair, (**e**) magnified, and (**f**) excoriations of the skin in the nape of the neck

Two treatments 1 week apart are necessary for all topical pediculides, given their poor ovicidal activity. Because no product is 100% ovicidal, it is important to concentrate on grooming and nit removal in the period between treatments to enhance efficacy.

Many parents are reluctant to apply pesticides to their children's heads and are turning to alternative therapies. Some have claimed to have successfully cured their infestation by using inexpensive, non-pesticide products, including Vaseline petrolatum jelly, hair pomade, olive oil, mayonnaise, vinegar, mineral oil, or essential oils from the health food store. However, these require repeated overnight treatments because they are not as effective as the currently available products, and they usually require many hours of painstaking combing. Noninsecticidal agents, including dimethicone and isopropyl myristate, show promise in the treatment of pediculosis [10].

Extensive environmental decontamination is not necessary after pediculosis is diagnosed. However, some school authorities may insist on a "no nit" policy to ensure freedom from infestation and proof of adequate treatment. This requires physically removing the nits form the hair. Although time-consuming, it relieves school authorities, nurses, and physicians of the difficulty of deciding whether eggs are viable, but it puts a burden on the parents, especially if they have a few children. Nit combs are constantly being improved on. The task of removing nits is made easier when the hair is wet, and the additional use of a detangler, crème rinse, light oil, or condition facilitates combing.

Cotrimoxazole (CTM) has been observed to be an effective treatment for pediculosis given orally [11]. So far, the use of antibiotics for the treatment of pediculosis capitis has been regarded appropriate only for infested children who have secondary bacterial infections of the scalp. The rationale behind this treatment is that, because lice are obligate blood feeders and are dependent on their symbiotic bacteria for survival, a blood meal containing antibiotic will kill their symbiotes. The minimal effective dose of CTM in pediculosis was found to be one tablet containing 80 mg of trimethoprim and 400 mg of sulfamethoxazole taken twice daily for 3 days. Patients are retreated with CTM twice daily for 3 days, 10 days after completion of the initial CTM therapy, by which time nits had hatched.

Oral ivermectin is an option for the treatment of head lice infestation, especially in individuals who have experienced a treatment failure. Published evidence from clinical trials indicates that oral ivermectin at a 2 × 200 μg/kg dosage given at day 1 and 7 is as effective as currently available topical treatments [12].

8.1.2 Crab Lice

The crab louse usually is found in a person's pubic hair. Although the louse cannot jump, it can also live in other areas of the body that are covered with coarse hair, such as the perianal area, the entire body in men, and the eyelashes (phthiriasis palpebrarum). Pubic lice have wide, short bodies resembling a tiny crab (Fig. 8.2). An adult crab louse is about 1.3–2 mm long and slightly smaller than the body louse

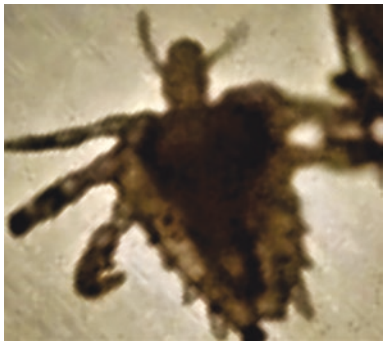

Fig. 8.2 Crab louse (pediculosis pubis)

and head louse and can be distinguished from those other species by its almost round body. Another distinguishing feature is that the second and third pairs of legs of a crab louse are much thicker than the front legs and have large claws.

The eggs of the crab louse are laid usually on the coarse hairs of the genital and perianal regions of the human body. The female lays about three eggs a day. The eggs take 6–8 days to hatch, and there are three nymphal stages which together take 10–17 days before the adult develops, making a total life cycle from egg to adult of 16–25 days. Adults live for up to 30 days. Crab lice feed exclusively on blood and take a blood meal four to five times daily. Outside the host they can survive for 24–48 h. Crab lice are transmitted from person to person most commonly via sexual contact.

The main symptom of infestation with crab lice is itching, usually in the pubic hair area, resulting from hypersensitivity to louse saliva, which can become stronger over 2 or more weeks following initial infestation. In some infestations, a characteristic gray-blue or slate coloration appears (maculae ceruleae) at the feeding site, which may last for several days.

Crab louse infestation can be suspected through the presence of the maculae ceruleae and diagnosed by identifying the presence of active stages of the louse, as well as of nits on the pubic hair or other hairs of the body.

Crab lice prefer hair that is widely spaced because the usually grasp one hair with the claws on one side of their body and another hair with the claws on the other side. Crab lice are relatively rare in Asians, perhaps because of sparser genital hairs. Infestation with pubic lice is relatively common among homosexual man. Crab lice found on the head or eyelashes of children may be an indication of sexual exposure or abuse [13]. And yet, although pediculosis pubis is considered a sexually transmitted disease, fomite transmission (bedding, clothing) may play a larger role than originally supposed. It has been suggested that an increasing percentage of humans removing their pubic hair, especially in women, has led to reduced crab louse populations in some parts of the world [14].

The role of pubic lice as transmitter of infectious disease has not been scientifically confirmed. And yet, the possibility of co-infection with another sexually

transmitted disease must be taken into consideration in the particular individual depending on behavioral risk factors. Crab lice collected from human immunodeficiency virus (HIV)-positive and HIV-negative volunteers were tested by stands for polymerase chain reaction (PCR) for the human immunodeficiency virus (HIV). All specimens obtained from HIV-1-positive individuals were found positive by ethidium bromide gel and confirmed on Southern blot [15]. As yet, HIV transmission by crab lice has not been demonstrated.

First-line pharmacologic treatment for pediculosis pubis is permethrin 1% lotion or shampoo. In adults, the presence of pubic lice should prompt an evaluation for sexually transmitted infections [16]. Pubic lice have always appeared to be among the hardiest of human lice when it comes to treatment. It is important to treat all hairy areas of the body since it is not uncommon to have other body areas infested without the patient being aware of it, and lice will wander away from a treated area in search of a more suitable environment.

Products for head and pubic lice are too irritating to use in the sensitive eye region. Although a safe and effective treatment for eyelash involvement with crab lice (phthiriasis palpebrarum) has been petrolatum jelly (Vaseline), it is slow and needs to be applied at least five times a day for 10 days. A course of oral tetracycline can be useful in killing the lice sooner. Finally, physostigmine 0.2% eyedrop, a pupillary dilator, has also been used successfully when applied to eyelashes; however, again the application must be repeated several times over a 3-day period.

8.2 Scabies

Scabies is one of the three most common skin disorders in children, along with ringworm and bacterial skin infections.

The Italian physician Giovanni Cosimo Bonomo (16666–1696) and biologist and entomologist Diacinto Cestoni (1637–1718) showed in the seventeenth century that scabies is caused by *Sarcoptes scabiei*, this discovery of the itch mite in 1687 marking scabies as the first disease of humans with a known microscopic causative agent.

Human scabies is caused by the female itch mite *Sarcoptes scabies* var. *hominis*, family Sarcoptidae, class Arachnida, and as such has four pairs (eight) of legs (two pairs in front and two pairs behind). Adult scabies mites are spherical, eyeless mites that are recognizable by their oval, ventrally flattened and dorsally convex tortoise-like bodies and multiple cuticular spines. No demarcation into cephalothorax or abdomen occurs, and the mite's surface has folds covered with short bristles. The front legs end in long, tubular processes known as suckers, and the hind legs end in long bristles (Fig. 8.3a). When placed on the skin, the female mite exudes a fluid that dissolves the skin surface, forming a well into which she sinks. In cool climates,

the female scabies mite forms a tunnel or burrow that may extend a distance of 0.5–5 mm per day. Gravid females lay the first egg within hours of burrowing and 2–3 eggs a day. The eggs hatch within 2.5–4 days. The six-legged larvae, which hatch in 3 to 10 days, move about on the skin, molt into a nymphal stage, and then mature into the eight-legged adult mites. The adult mites live 3–4 weeks in the host's skin. The male mites which are only half the size of females do not burrow, however do enter the females' residence shortly to copulate, leaving irritant antigenic matter. In fact, the pruritic eruption of the skin is caused by both the burrowing and release of irritant and antigenic secretions and excretions of the mite.

Scabies represents a contagious skin infestation, the most common skin symptoms being severe itchiness often worse at night, causing papular pustular-vesicular eruptions of the skin. Although the life cycle of the scabies mite is only about 2 weeks, individual patients are seldom found to have more than about a dozen mites on them. Even so, this number is sufficient to cause agonizing itching and severe damage to the skin as a result of scratching, in particular by the introduction of infective bacteria, which may lead to impetigo or eczema. A delayed type IV hypersensitivity reaction to the mites, their eggs, or packets of feces (skybala) occurs approximately 30 days after infestation. The presence of the eggs produces a massive allergic response that, in turn, produces more itching. Individuals who already are sensitized from a prior infestation can develop symptoms within hours.

Crusted scabies (Fig. 8.3b–e), also called Norwegian scabies, is an infestation characterized by thick crusts of skin that contain large numbers of scabies mites and eggs. It represents a severe form of the disease that occurs most often elderly, disabled, and people with impaired immune systems, such as those with HIV/AIDS or cancer or those on immunosuppressive medications. On those with weaker immune systems, the host becomes a more fertile breeding ground for the mites, which spread over the host's body, including the scalp [17–23]. The mites in crusted scabies are not more virulent than in non-crusted scabies; however, they are much more numerous (up to two million). People with crusted scabies exhibit scaly rashes, slight itching, and thick crusts of skin that contain large numbers of scabies mites. For this reason, persons with crusted scabies are more contagious to others than those with typical scabies. Such areas make eradication of mites particularly difficult, as the crusts protect the mites from topical scabicides, necessitating prolonged treatment of these areas and/or oral treatment.

The mite is found in all parts of the world. Scabies may be diagnosed clinically in geographical areas where it is common when diffuse itching presents along with either lesions in two typical spots or itchiness is present in another household member.

The classical sign of scabies is the burrow made by a mite within the skin. To detect the burrow (Fig. 8.3f), the suspected area is rubbed with ink from a fountain pen or a topical tetracycline solution, which glows under a special light. The skin is then wiped with an alcohol pad. If the person is infected with scabies, the characteristic zigzag or S pattern of the burrow will appear across the skin; however, interpreting this test may be difficult, as the burrows are scarce and may be obscured by scratch marks.

A definitive diagnosis is made by finding either the scabies mites or their eggs and fecal pellets (Fig. 8.3g). Searches for these signs involve either scraping a suspected area, mounting the sample in xyline, and examining it under a microscope.

On histopathology, scanning power view of scabies shows a pattern of an epidermal and wedge-shaped dermal inflammatory process (Fig. 8.3h). The epidermal histological findings in the primary scabetic lesion are hyperkeratosis, acanthosis, and spongiotic edema and vesiculation. The dermal changes consist of perivascular and diffuse cell infiltrates, mainly mononuclear cells, and sometimes eosinophilic

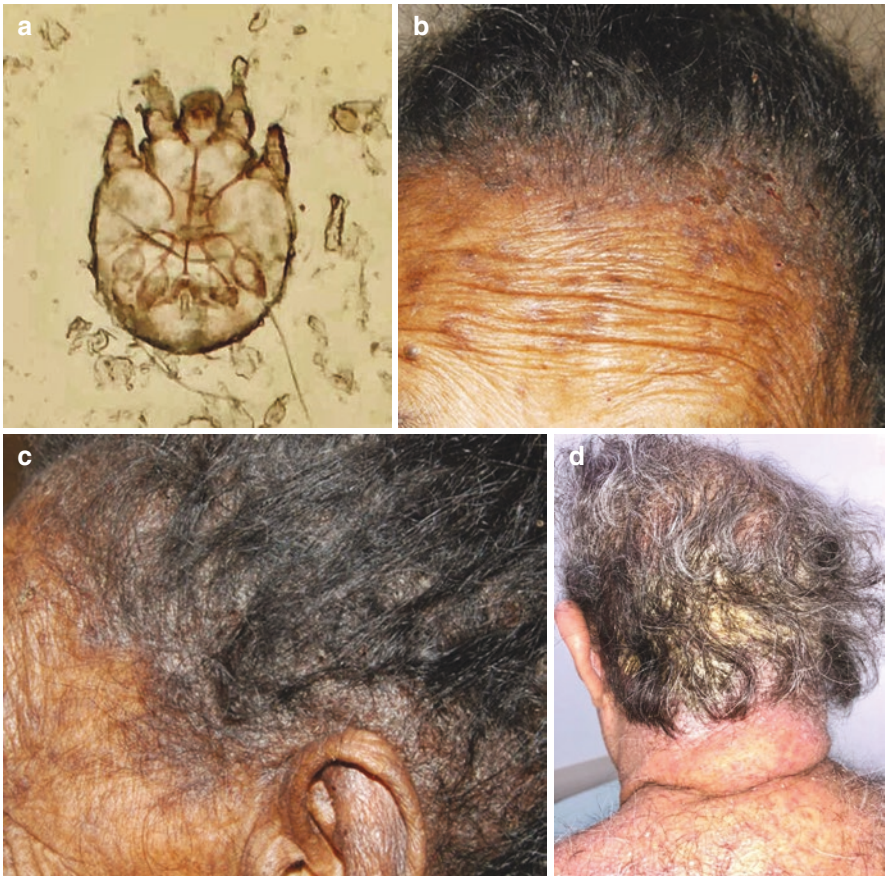

Fig. 8.3 (**a**–**i**) Scabies: (**a**) Scabies mite (*Sarcoptes scabies*), courtesy Dr. Marcelo Teixeira, Dermatologist, Brazil, (**b**–**e**) crusted scabies of the scalp (courtesy Dr. Marcelo Teixeira, Dermatologist, Federal Fluminense University, Brazil, and Prof. Fábio Francesconi, Federal University of Amazonas, Brazil), (**f**) dermoscopy of scabies burrow, (**g**) scabies fecal pellets (skybala) (arrows), (**h**–**j**) histopathology: (**h**) pattern of an epidermal and wedge-shaped dermal inflammatory process, (**i**) perivascular and diffuse mainly mononuclear cell infiltrates with eosinophilic granulocytes, (**j**) presence of scabies parts evident as solid fragments representing the chitinous exoskeleton of the *Sarcoptes scabiei* within the epidermis (courtesy Prof. Werner Kempf, kempf und pfaltz histologische diagnostik, Zurich, Switzerland)

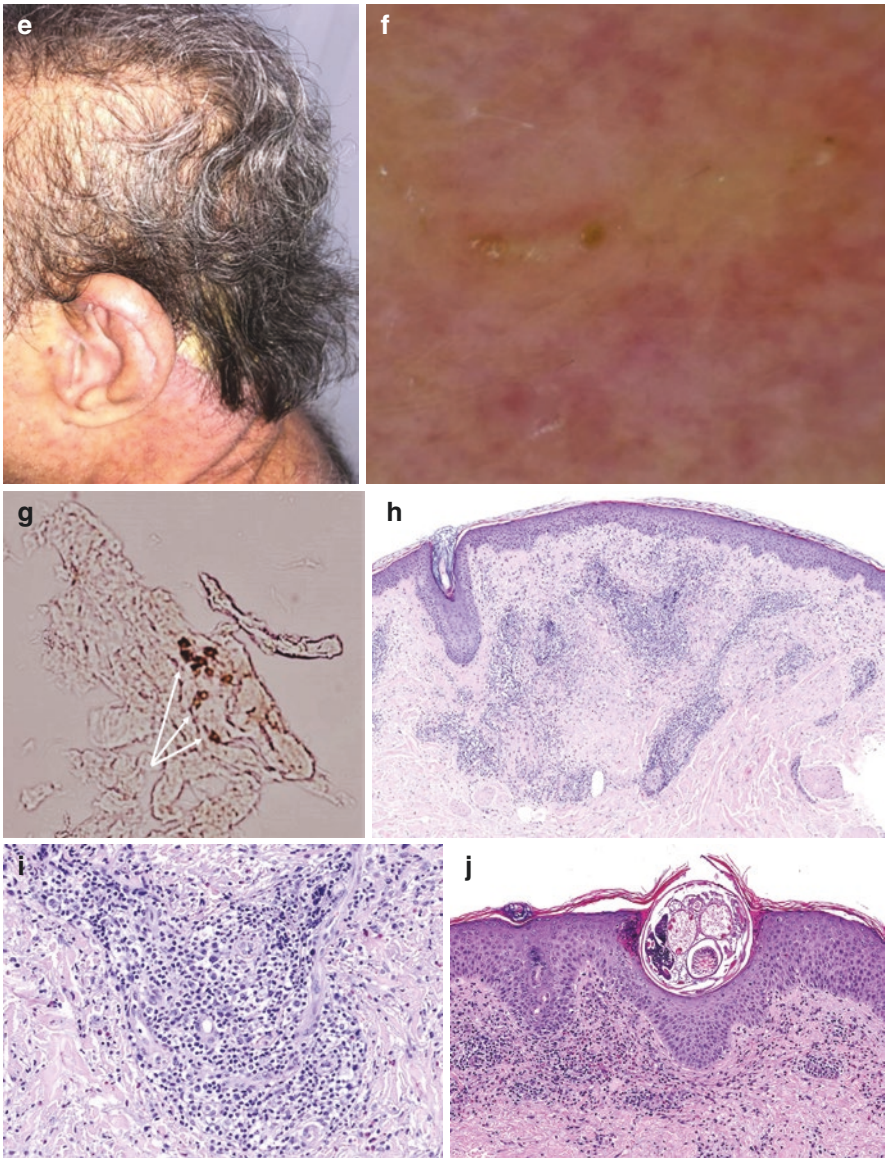

Fig. 8.3 (continued)

granulocytes (Fig. 8.3i). Deep interstitial eosinophils are an important clue to an arthropod bite reaction. Ultimately, the presence of scabies parts evident as solid pink eosinophilic fragments representing the chitinous exoskeleton of the *Sarcoptes scabiei* mite is diagnostic (Fig. 8.3j).

Several medications are effective in treating scabies. The simultaneous treatment of all close contacts is recommended, even if they are asymptomatic, to reduce rates of recurrence. Bedding, clothing, and towels used during the previous 3 days should be washed in hot water and dried in a hot dryer. Treatment protocols for crusted scabies are significantly more intense than for common scabies.

Permethrin 5% dermal cream is the most effective treatment for scabies and remains the treatment of choice. It is applied from the neck down, usually before sleep, and left on for about 8 to 14 h, then washed off in the morning. Care should be taken to coat the entire skin surface, not just symptomatic areas. Any patch of skin left untreated can provide a safe haven for one or more mites to survive, particularly the nails. These are important as the principal tools for scratching and may also act as a reservoir for mites and their eggs. One application is normally sufficient, as permethrin kills eggs and hatchlings, as well as adult mites, though many physicians recommend a second application 3–7 days later as a precaution.

Oral ivermectin at a single 200 μg/kg dose and at two doses in patients with crusted scabies, respectively, is effective in eradicating scabies. It is the treatment of choice for crusted scabies and is sometimes prescribed in combination with a topical agent. One review found that the efficacy of permethrin is similar to that of systemic or topical ivermectin. A separate review found that although oral ivermectin is usually effective for treatment of scabies, it does have a higher treatment failure rate than topical permethrin. Another review found that oral ivermectin provided a reasonable balance between efficacy and safety. A study has demonstrated that scabies is markedly reduced in populations taking ivermectin regularly. The drug is widely used for treating scabies and other parasitic diseases particularly among the poor and disadvantaged in the tropics [24–32].

Other treatments of the past have included lindane (1% lotion), benzyl benzoate (25% to 30% to be applied for 3 alternate or consecutive days), crotamiton (10% cream), and sulfur preparations (at concentrations of 6% or less in several applications over 2 or 3 days). Today, lindane is not recommended for its neurotoxicity and emergence of lindane-resistant scabies and crotamiton for lack of efficacy and lack of toxicity data. Because permethrin cream is approved for use on infants aged 2 months or older, it has largely replaced sulfur in pediatric practice.

Mass treatment programs that use topical permethrin or oral ivermectin have been effective in reducing the prevalence of scabies in a number of populations. Since mites can survive for only 2–3 days without a host, other objects in the environment pose little risk of transmission except in the case of crusted scabies. Therefore, cleaning is of little importance. However, rooms used by those with crusted scabies require thorough cleaning. Particularly nursing homes and hospital staff have notably become infested by changing bedding, even with no patient contact.

Among the famous people who have allegedly suffered of scabies, Napoleon (1769–1821) has been the most notorious. And yet there are no authentic records to prove that he ever had this disease. Considering the emperor's habits of hygiene; the

Fig. 8.4 (**a**–**c**) Solar-powered scratching Napoleon by Kikkerland. Solar cell is at the top of his hat. Napoleon's characteristic pose with his hand beneath his shirt was rather a mannerism than due to itching form scabies

long duration of his skin condition, which began with the siege of Toulon (1793) and lasted for 9 or 10 years; and the numerous sulfur baths which he took, it seems improbable that he should have suffered of scabies. Moreover, the remedy consisting of a concoction of ointments and salves containing olive oil, alcohol, and powdered "cevilla," which his personal physician Jean-Nicolas Corvisart (1755–1821) used successfully, would have been practically useless for the treatment of scabies. In fact, Friedman in his book, *The Emperor's Itch: The Legend Concerning Napoleon's Affliction with Scabie*s, suggests that Napoleon may have actually suffered from dermatitis herpetiformis. The author also thinks that Napoleon's characteristic pose with his hand beneath his shirt was rather a mannerism possibly cultivated in his youth (Fig. 8.4a–c) than due to itching from cutaneous disease [33].

8.3 Demodex

Demodex is a genus of microscopic mites that live in or near hair follicles of mammals. Around 65 species of Demodex are known. Two species live on humans: *Demodex folliculorum* and *Demodex brevis*, both frequently referred to as eyelash mites, alternatively face mites or skin mites. *D. folliculorum* is found in hair follicles, while *D. brevis* lives in sebaceous glands connected to hair follicles. Both

species are primarily found in the face near the nose, the eyelashes, and eyebrows but also occur elsewhere on the body, including the scalp.

The adult mites are only 0.3–0.4 mm long, with *D. brevis* slightly shorter than *D. folliculorum* [34]. Each has a semitransparent, elongated body that consists of two fused segments. Eight short, segmented legs are attached to the first body segment (Fig. 8.5). The body is covered with scales for anchoring itself in the hair follicle, and the mite has pin-like mouthparts for eating skin cells and oils that accumulate in the hair follicles. The mites can leave the hair follicles and slowly walk around on the skin, at a speed of 8–16 mm per hour, especially at night, as they try to avoid light. The mites are transferred between hosts through contact with hair, eyebrows, and the sebaceous glands of the face.

Older people are much more likely to carry the mites; about a third of children and young adults, half of adults, and two-thirds of elderly people carry them [35]. The lower rate in children may be because children produce less sebum or simply have had less time to acquire the mite.

29% of unselected pathological and forensic autopsy cases revealed *Demodex folliculorum* and *brevis* in hair follicles and sebaceous glands of the scalp. The

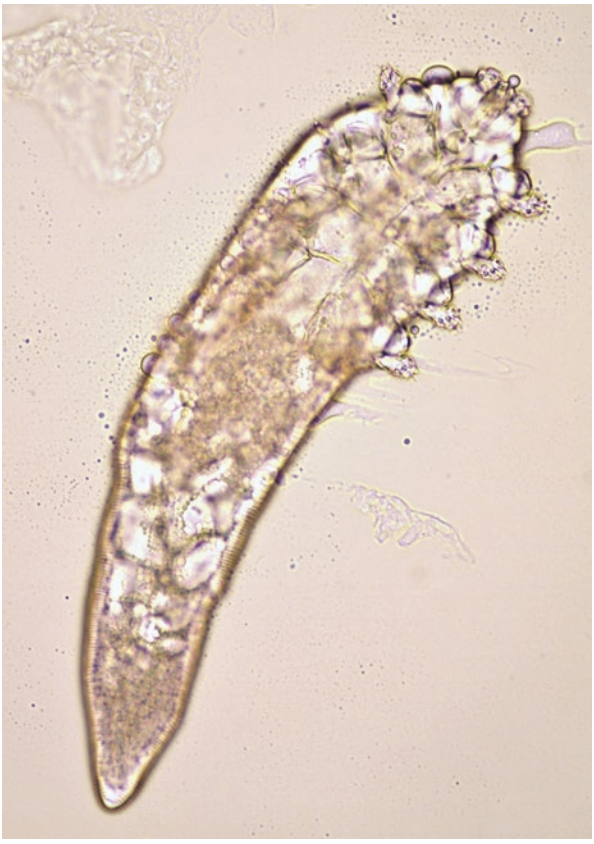

Fig. 8.5 *Demodex folliculorum*

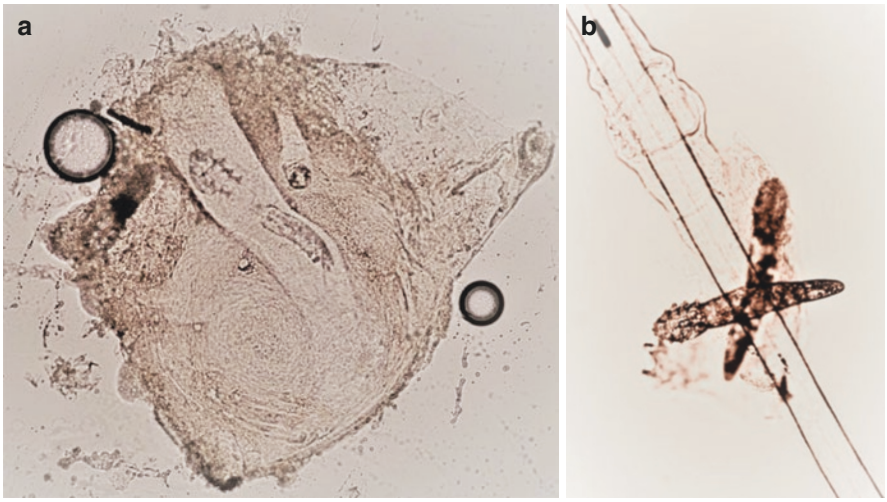

Fig. 8.6 (**a**, **b**) Demodex mites are common (**a**) commensals of the pilosebaceous unit, and often (**b**) found in plucked hair mounted for the trichogram examination

frequency was equal in male and female subjects and correlated to the number of sebaceous glands, and not to the density of hair follicles. However, there was a tendency to an increased number of parasites inhabiting the scalp of people of advanced age, or with a bald head. A chronic lymphocytic infiltration of the skin was conspicuous in more than 70% of cases [36].

A 2014 study of $n = 29$ people in North Carolina, USA, found that all the adults ($n = 19$, over 18 years of age) carried mites and that 70% of those under 18 years of age carried mites [37]. This study using a DNA detection method, more sensitive than traditional sampling and observation by microscope, along with several studies of cadavers, suggests that previous work might have underestimated the mites' prevalence. However, the small sample size and small geographical area involved prevent drawing broad conclusions from these data.

Demodex infestation of the scalp has previously been put forward as a possible etiological factor in some cases of scalp rosacea [38]. However, demodex mites are common commensals of the pilosebaceous unit (Fig. 8.6a), often found in plucked hair mounted for the trichogram examination (Fig. 8.6b) However, there has been a controversy to what degree demodex mites are causative of skin pathology and how they might contribute to disease in humans until topical ivermectin has successfully been introduced for treatment of rosacea [39].

Demodex folliculorum and *D. brevis* have been identified and implied to play a role in pustular folliculitis of the face (Fig. 8.7a, b) [40], papulopustular scalp eruptions (Fig. 8.7c, d) [41], eosinophilic follicular reaction [42], and blepharitis (Fig. 8.7e) [43]. In general, the clinical presentations of demodicidosis have been classified into three main groups:

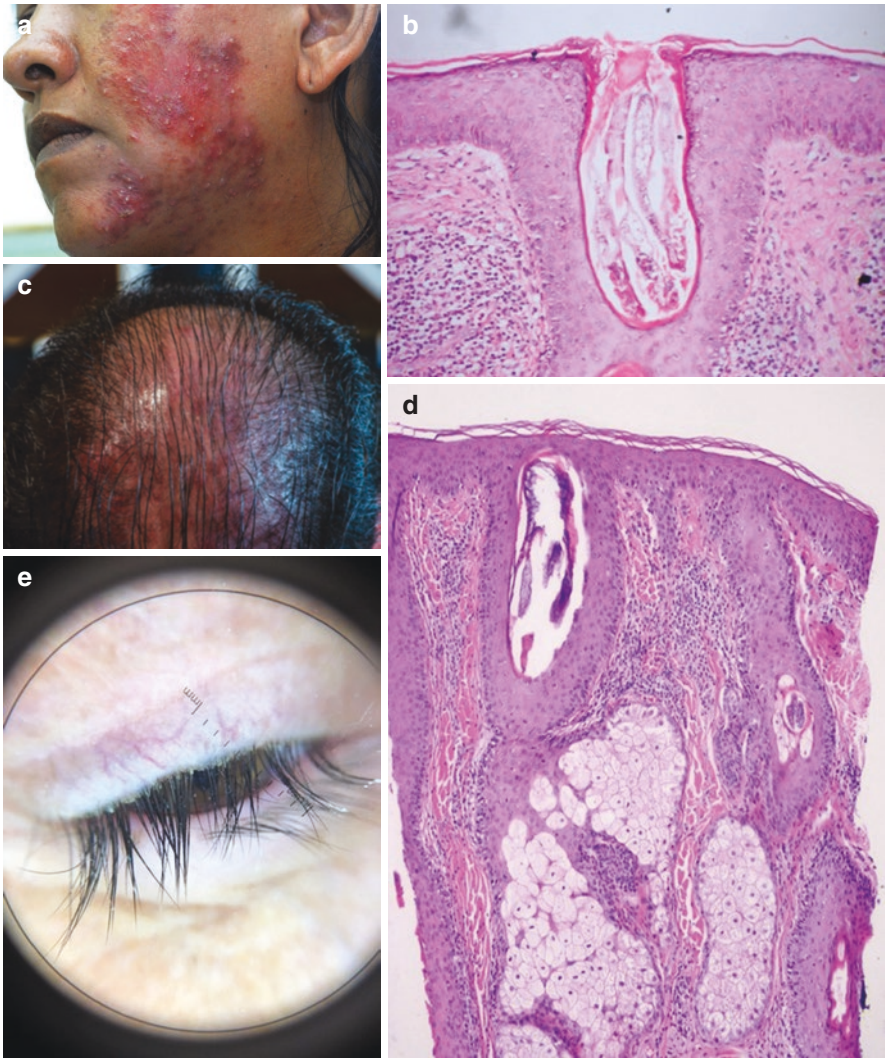

Fig. 8.7 (**a**–**e**) Demodicidosis: (**a**) pustular folliculitis of the face, (**b**) histopathology (courtesy Prof. Fábio Francesconi, Federal University of Amazonas, Brazil), (**c**) papulopustular scalp eruptions, (**d**) histopathology, (**e**) blepharitis

- Pityriasis folliculorum
- Rosacea-like demodicidosis
- Granulomatous rosacea-like demodicidosis gravis

A case of scalp demodicosis in a patient with frontal fibrosing alopecia dermoscopically mimicking active disease was reported. A 78-year-old Caucasian male with a previous history of a well-controlled frontal fibrosing alopecia with high-potency topical corticosteroids presented with scalp itching, follicular hyperkeratosis, and erythema. A scalp biopsy of this area revealed numerous mites in the follicular openings. Based on these findings and the history of chronic use of topical corticosteroids, a diagnosis of secondary scalp demodicosis was made. Discontinuation of topical corticosteroid treatment was recommended, and four doses of oral ivermectin 200 mcg/kg on a weekly basis were prescribed. One month later, dermoscopic signs suggestive of demodicosis as well as pruritus resolved [44]. It is also our observation that sometimes persistent follicular inflammation, scaling, and pruritus in patients with frontal fibrosing alopecia despite topical corticosteroid treatment may sometimes resolve upon a trial with topical 5% permethrin cream [unpublished data], while we alternatively interpreted this observation as an abnormal reaction of the hair follicle on commensal demodex mites.

Methods used for diagnostic purposes in demodicosis include cyanoacrylic adhesives, comedone extractor, cellophane tape preparations, skin scraping, punch biopsy, and standardized surface skin biopsy [45].

Therapy includes oral metronidazole (250 mg bid for 30 days), 5% topical permethrin cream once daily (Fig. 8.8a, b), or oral permethrin (200 micrg/kg) followed by maintenance therapy with 5% topical permethrin cream weekly [46].

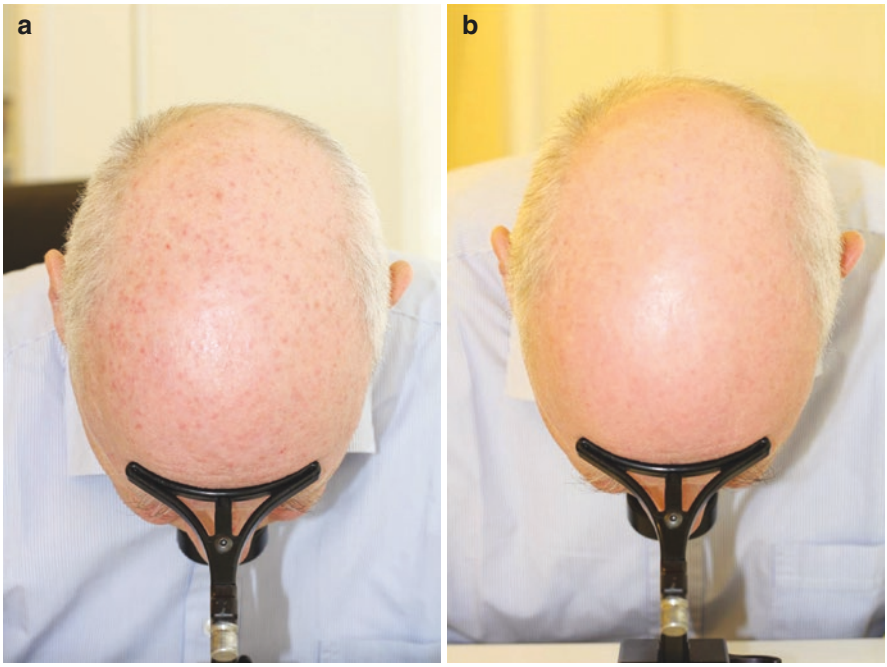

Fig. 8.8 (**a**, **b**) Scalp demodicidosis (**a**) before, and after (**b**) successful treatment with 5% topical permethrin cream daily

8.4 Tick

Ticks (order Ixodida) are parasitic arachnids that are part of the mite superorder *Parasitiformes.* Ticks are ectoparasites and consume blood to satisfy all of their nutritional requirements. They are obligate hematophages and require blood to survive. On locating a suitable feeding spot, the tick grasps the host's skin and cuts into the surface. It extracts blood by cutting a hole in the host's epidermis, into which it inserts its hypostome and prevents the blood from clotting by excreting an anticoagulant or platelet aggregation inhibitor [47]. Ixodidae remain in place until they are completely engorged. Tick saliva contains about 1500 to 3000 proteins, depending on the tick species. The proteins with anti-inflammatory properties, called evasins, allow ticks to feed for 8–10 days without being perceived by the host animal.

Ticks are implicated in the transmission of a number of infections caused by pathogens such as bacteria, viruses, and protozoa. Species of the bacterial genus *Rickettsia* are responsible for typhus, rickettsial pox, boutonneuse fever, African tick bite fever, Rocky Mountain spotted fever, Flinders Island spotted fever, and Queensland tick typhus (Australian tick typhus). Other tick-borne diseases include Lyme disease and Q fever, Colorado tick fever, Crimean-Congo hemorrhagic fever, tularemia, tick-borne relapsing fever, babesiosis, ehrlichiosis, Bourbon virus, and tick-borne meningoencephalitis, as well as bovine anaplasmosis and the Heartland virus. In the United States, Lyme disease is the most commonly reported vector-borne disease in the country. Finally, a tick can harbor more than one type of pathogen, making diagnosis more difficult.

Not all ticks in an infective area are infected with transmittable pathogens, and both attachment of the tick and a long feeding session are necessary for diseases to be transmitted. Consequently, tick bites often do not lead to infection, especially if the ticks are removed within 36 h. While adult ticks can be removed with fine-tipped tweezers or proprietary tick removal tools then disinfecting the wound, there is growing consensus that ticks should be killed in situ and frozen with either a custom spray or medical wart remover and left to fall out to avoid anaphylactic/allergic reactions.

Tick bite alopecia represents a distinctive tick bite-related condition characterized by a solitary oval zone of alopecia with a central eschar. Histologic findings are not well described but generally indicate dense perifollicular lymphocytic inflammation. The mechanism for hair loss is poorly understood, but the prognosis for hair regrowth appears to be favorable [48]. As anticoagulants may cause alopecia, it has been speculated on the possibility that their presence may be responsible for the depilating effect of tick saliva [49]. In view of the histological findings, however, it is more likely that alopecia is the result of the joint effect of tick saliva and host reaction. Tissue necrosis in the immediate vicinity of the attachment site of the tick probably accounts for the central eschar and initial central hair loss. Dilution of the salivary juice as is spreads through the host tissue would result in progressive loss of activity with diminishing destructive power [50].

The scarring form of tick bite alopecia is characterized by the formation of an eschar that progresses to patches of cicatricial alopecia. This scarring form of tick

bite alopecia histologically is characterized by a loss of hair follicles with fibrosis, periadnexal lymphocytic inflammation, and an interstitial eosinophilic or granulomatous infiltrate. The etiology of the hair loss may be related to the host response to tick saliva, external forces, and, in some scarring cases, rickettsial infection [51]. Tick-borne lymphadenopathy syndrome, classically transmitted by ticks of the genus *Dermacentor* and caused by *Rickettsia slovaca* infection, is an entity typically seen in Europe and highly associated with alopecic eschar, fever, malaise, and painful nuchal lymphadenopathy [52, 53]. Doxycycline is the treatment of choice.

Both, pseudopelade Brocq [54] and acute diffuse and total alopecia of the female scalp [55] have been reported as possible sequelae of stage III Borrelia infection. In both cases, treatment with intravenous cefotaxime and ceftriaxone, respectively, were successful in arresting progression of scarring alopecia and recovering hair in the diffuse and total alopecia.

Besides several reports of localized alopecia after tick bites, there have also been few reports of ant-induced alopecia in the literature [56–58]. Ant-induced alopecia should be considered in the differential diagnosis of localized sudden onset alopecia, at least in some geographic areas of the world.

8.5 Furunculoid Myiasis

Cutaneous myiasis is a parasitic infestation that is caused by developing larvae (maggots) of a variety of fly species which are classified within the arthropod order Diptera [59–61]. Although flies are most commonly attracted to open wounds and urine- or feces-soaked fur, some species can create an infestation even on unbroken skin. The larvae feed on the host while they slowly grow until they achieve mature stages and finally complete the larvae cycle [59, 60, 62]. The most common flies that cause human disease are *Dermatobia hominis* (also called human botfly) and *Cordylobia anthropophaga* (tumbu fly) [62, 63].

The type of presentation that is seen in patients with myiasis depends both on the fly species involved and where their eggs are laid [60, 64]. Most authors classify human myiasis based on the body area that the parasites end up settling in order to grow, e.g., skin, oral cavity, nose cavity, and eyes [63].

The cutaneous presentation depends both on the type of larvae that is involved and on the anatomical site that has been targeted by the vector:

- Furuncular cutaneous myiasis
- Wound myiasis
- Creeping/migratory cutaneous myiasis

Wound myiasis is most commonly observed in patients with chronic and open wounds from different etiologies, such as skin cancer and diabetic ulcers, but larvae can also penetrate the unbroken skin [65, 66]. Since flies usually thrive in warm

environment and under poor sanitary conditions, people from rural areas are at special risk for the infestation [65, 67]. It also seems that elderly patients, especially those who are mentally disabled, are more affected than young ones, which is probably due to a lower level of attention when flies approach.

Migratory cutaneous myiasis is usually observed in patients who deal with cattle which can be eventually infested by *Hypoderma bovis* and in people who work with horses; in this case, *Gasterophilus intestinalis* is usually involved [60, 61]. This clinical presentation is not frequently seen in clinical practice, but some cases may well be misdiagnosed as cutaneous larva migrans.

The ample variation of prevalence that is noted from country to country is due to differences in latitude that ultimately interferes in the life cycle of the several species of flies [60, 61]. Typically, case reports of myiasis report on patients who live or have recently travelled to tropical global areas, such as Africa and South America [63, 68].

For myiasis to happen, it is essential that a fly exists in the disease chain so that the parasite eggs can ultimately reach human soft tissues [65, 67]. Different species of flies can carry the eggs that will finally evolve into larvae. The type of fly that acts as vector varies considerably from region to region owing to the large variability of environmental factors that can ultimately impact the life cycle of the insects, such as humidity and temperature [60–62].

A case series published by Lachish and colleagues showed that *Dermatobia hominis* was the vector responsible for approximately 80% of cases of cutaneous myiasis [63]; *Cordylobia anthropophaga* was involved in 17% of the cases and caused disease especially in patients who have recently visited Africa [63]. The literature is inconsistent though when it comes to determining the most commonly involved species in cutaneous myiasis, but it seems that *Dermatobia hominis* is the most common causative agent of the furunculoid presentation in Central and South America [63, 64, 69, 70].

The cutaneous infestation usually occurs after a direct contact of the fly with the affected person; nonetheless, fly eggs can also be deposited in fabric and only then reach the human body when they turn into a larval stage [61]. *Dermatobia hominis,* for instance, first lays its eggs on ordinary mosquitoes, and these will then take the eggs to the skin surface of a given person [61]. As opposed, *C. anthropophaga* usually deposits its eggs on fabric and clothes, as well as in soiled blankets and in sand. The larva is capable of living up to 15 days without feeding, until a host is ultimately found [61, 62].

In wound myiasis, eggs are laid in an open cutaneous orifice, such as chronic wounds, necrosis, or ulcers [65]. Larvae can be deposited in any body area and may give rise to complications, especially when the nasal cavity, sinuses, and scalp are affected.

The most common clinical presentation of cutaneous myiasis is furuncular or furunculoid myiasis, named after its similar clinical appearance as a boil (furuncle), caused by *Dermatobia hominis* [62, 68]. Each larva produces a separate lesion, which is clinically recognized as a nonhealing or boil- or furuncle-like lesion, even though multiple larvae can be simultaneously observed at the same body site [62,

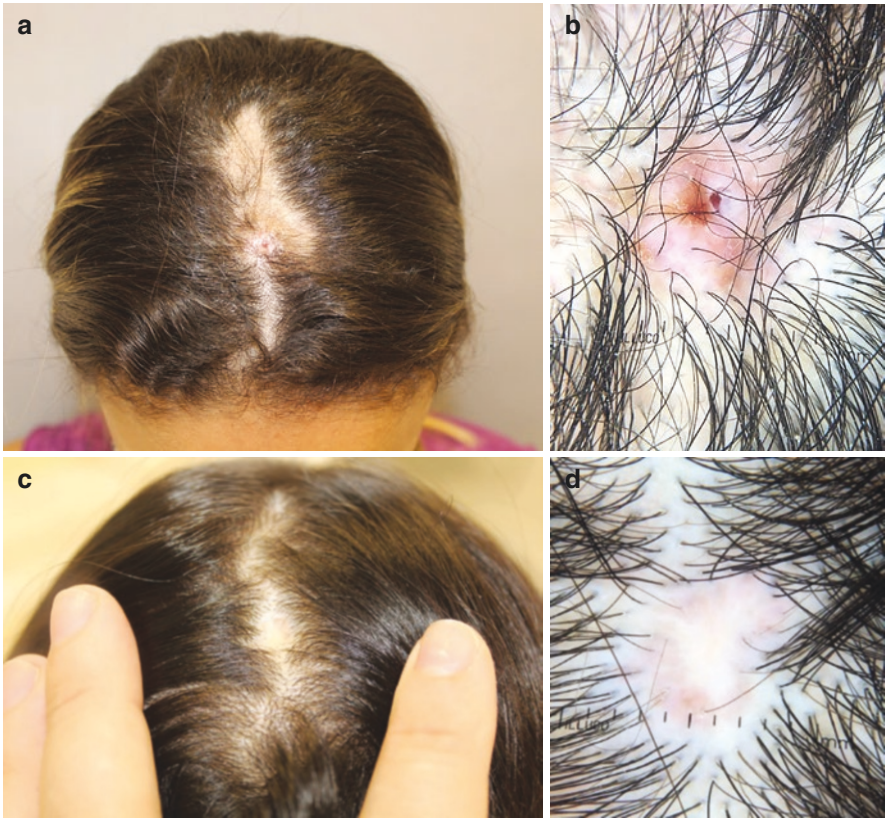

Fig. 8.9 (**a–d**) Furunculoid myiasis: (**a**, **b**) boil-like lesion with a central punctum, (**c**, **d**) residual scar

63, 66]. The lesion is recognizable as such by discharge from a central punctum (tiny hole) or a small, white structure protruding from the lesion. Healing usually occurs with a scar (Fig. 8.9a–d). Up to 60% of lesions occur on exposed body areas, but it is not mandatory [62]. These lesions oftentimes cause itching sensation that comes along with tenderness on palpation, emission of blood, and purulent discharge, which makes it difficult to be differentiated from simple cutaneous furunculosis and infected epidermal inclusion cysts.

In many cases, myiasis is an accidental diagnosis, being rather a surprise for both the patient and the attending physician.

Table 8.1 lists some symptoms reported by affected individuals on the occasion of their first consultations and the signs observed by the medical team.

The patients' previous history of trips to endemic areas, such as Brazil and other counties of South America, may be of great help to the attending physician when it comes to suspecting of the diagnosis.

Table 8.1 Clinical signs and symptoms of furuncular scalp myiasis

Authors	Signs and symptoms
Biswas and Mcnerney [60]	Scalp bleeding
Kondoh et al. [61]	Bleeding and pain Painful boils, sensation of "something moving inside"
Dunphy and Sood [62]	Pruritus, drainage of serosanguinous fluid, intermittent sharp, stabbing episodes of pain
Calderaro et al. [64]	Pain, itching, and sensation of movement under the skin

In 2008, Calderaro and colleagues [64] reported the case of a male Italian patient who had presented for dermatological evaluation owing to the presence of a furunculoid scalp lesion after coming back from a trip to Brazil. The patient reported being stung by insects on the scalp and noticing something mobile protruding from the lesions, resembling larvae. The lesion was surgically accessed, and the larvae were removed.

Since myiasis is not a primary disease of the hair follicles, it is not expected from infected patients to present alopecia. Nevertheless, scalp may well be involved, as reported by several authors [60–62, 66, 69, 70]. It is interesting that hairy areas can be targeted by flies, since dense hair coverage would naturally work as a physical barrier, even though the majority of the authors who have published on scalp myiasis have not mentioned in their papers if the patients were bald or not.

Myiasis has been reported in patients with the diagnosis of scalp malignancies, such as basal cell carcinoma and squamous cell carcinoma [61, 66, 69]. Other scalp conditions have also been associated with local myiasis (Fig. 8.10), as reported by Pereyra-Rodríguez et al. in 2010 [70], when the authors reported a case of a 12-year-old girl with severe scalp larval infestation in lesions of psoriasis.

A dramatic case of scalp myiasis was published by Wollina in 2010 [66]. The author reported on an 89-year-old male patient who was taken to the emergency department with a bleeding large ulcer on his scalp. At the time point, the patient had already had the diagnosis of a squamous cell carcinoma of the scalp for which he had refused surgical treatment. He was mentally healthy and had no social or financial issues, and he was being supported by an ambulatory nursing service. The removal of the wound dressing revealed the presence of more than a hundred maggots of *Lucilia sericata*. This case reinforces the fact that the elderly are at a special risk for cutaneous myiasis but also calls attention to the fact that myiasis can complicate any type of scalp ulcer, even if good healthcare is being provided.

Even though clinical inspection is sufficient to diagnosis wound myiasis, there are several differential diagnoses to be considered in the case of furunculoid myiasis. In regions where myiasis in endemic, in any nonhealing cutaneous furuncular lesion, furuncular myiasis must be considered in the differential diagnosis.

Bacterial furunculosis presents with a prominent surrounding erythema and is centered by a hair follicle orifice, which can be recognized both by the naked eyes and dermoscopy [71]. In case of doubt, a Doppler ultrasound scan can visualize

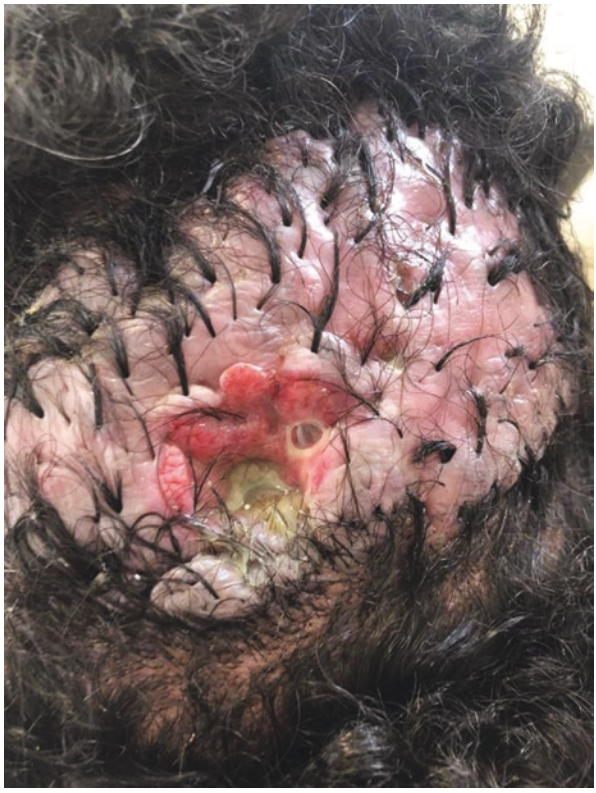

Fig. 8.10 Furunculoid myiasis on the background of folliculitis decalvans

larval movements within lesions initially thought to be ordinary furuncles [62, 63, 71].

Clinical history is usually enough when it comes to distinguish furuncular myiasis from epidermal inclusion cysts, an exaggerated arthropod bite reaction, and abscesses. In areas where cutaneous leishmaniasis is endemic, it is of ultimate importance to evaluate the patient with extra caution, since leishmaniasis may mimic several cutaneous disorders, including furunculosis [72]. For such cases, complementary diagnostic tools, such as dermoscopy, cutaneous ultrasound, and skin biopsy, may be of help either by showing the presence of the larvae or by demonstrating the presence of *Leishmania* spp. [72, 73].

It is noteworthy that physicians from endemic countries, such as Brazil, usually have the necessary expertise for an immediate clinical diagnosis. Practically speaking, despite the value of several diagnostic tools, the diagnosis of furuncular myiasis is usually solely based on clinical grounds.

Although cutaneous myiasis is considered a self-limiting condition with minimal morbidity in most of patients, treatment is recommended so that relief of pain can be achieved. The great impact on the patients' cosmetic and psychological aspects should also be considered on the decision to treat.

Patients with wound myiasis must have their larvae mechanically removed. Since not all larvae are visible and there is a risk of them to tunnel away, a mixture of 15% chloroform with olive oil has been used in the wound; this mixture is believed to immobilize the larvae and facilitate maggot removal [60, 61].

The literature shows different approaches that have already been proposed to treat furuncular myiasis. After noticing that a breathing hole has been formed in the skin surface, covering it is usually a strategy of success, since it asphyxiates the parasite [68]. This method can be performed with application of petrolatum jelly, which will ultimately oblige the larva to come to the skin surface to breath and then the condition can be more easily managed [68]. Other occluding strategies, such as adhesive tapes and bacon, are also described as effective methods to create localized hypoxia [62, 68] (Fig. 8.11a).

For some patients, removing the larva surgically will be necessary. It must be done under local anesthesia, and even though a late foreign body reaction can occur if parts of the larva remain, surgery is usually curative. For this purpose, the physician must perform a minor incision right across the parasite orifice, and with a slight pressure around the lesion, the larva can be removed with a forceps [68] (Fig. 8.11b–d). Local care after surgery includes the prescription of antiseptic dressings and antibiotics in case of secondary infection.

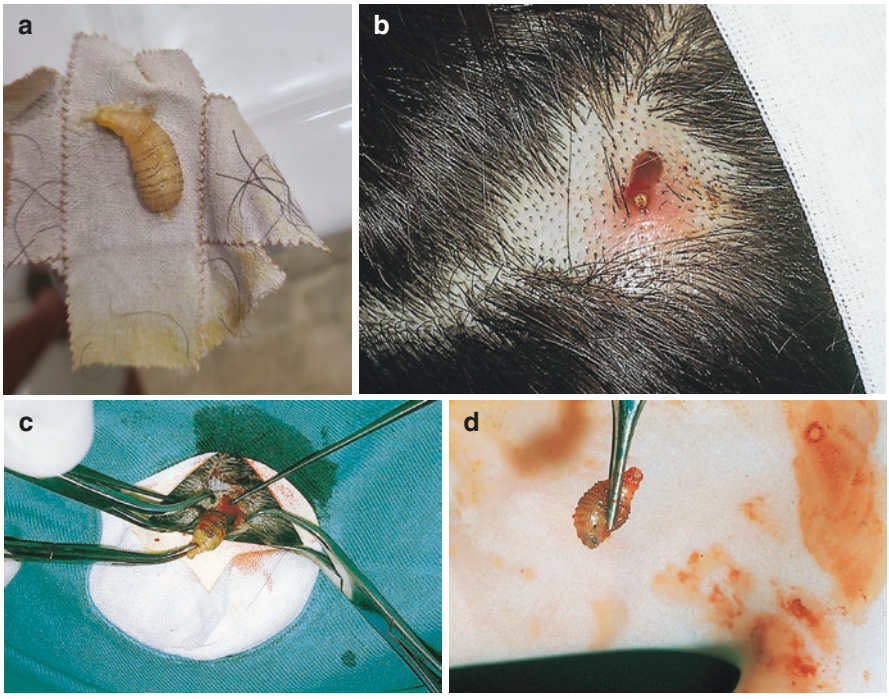

Fig. 8.11 (**a**–**d**) Treatment of furunculoid myiasis. (**a**) adhesive tape, (**b**–**d**) surgical extraction

Fig. 8.12 Extracted *Dermatobia hominis*. Notice rows of spines that work as hooks

Finally, patients should be discouraged from managing the lesions on their own, since the larva in furuncular myiasis have several rows of spines that work as hooks (Fig. 8.12) and usually prevent the patient from a successful home extrusion [62, 64].

8.6 Cutaneous Larva Migrans

Cutaneous larva migrans, also known as creeping eruption, is a pruritic dermatitis due to the inoculation of helminths larvae in the skin often occurring in children in tropical and subtropical areas. It represents a zoodermatosis caused by the cutaneous penetration usually of parasites from the small intestines of cats and dogs in people who visit beaches or sandy terrains which are polluted with the feces of dogs and cats. Clinically it is characterized by the presence of intensely pruritic erythematous tunnels of linear and serpiginous character. The lesion topography

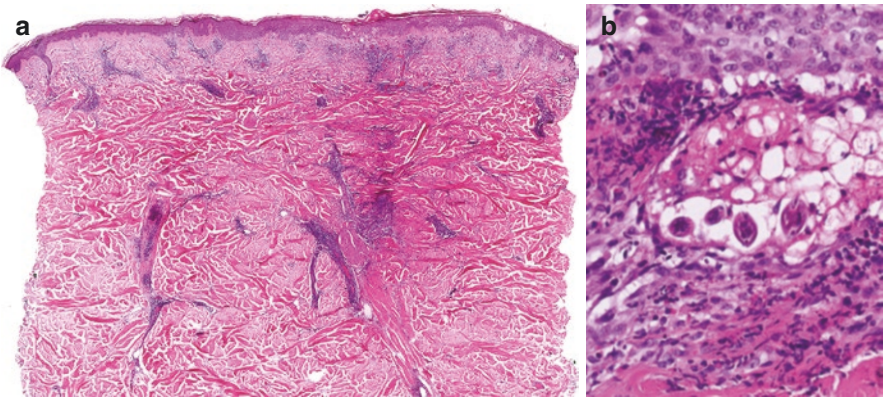

Fig. 8.13 (**a**, **b**) Cutaneous larva migrans. Histopathology (courtesy Prof. Werner Kempf, kempf und pfaltz histologische diagnostik, Zurich, Switzerland)

usually depends on the area which is in wider contact with the ground, like feet, legs, or gluteal regions. Skin biopsy from advancing point of the lesion can show parts of parasite(s), where they typically present as curvilinear eosinophilic larvae within the epidermis (Fig. 8.13a, b).

The main species responsible for cutaneous larva migrans are *Ancylostoma caninum* and *Ancylostoma braziliense*. Among the agents that can also cause the disease are other parasitic larvae of dogs and cats, such as *Uncinaria stenocephala*, *Ancylostoma tubaeforme*, *Gnathostoma spinigerum*, and some strains of *Strongyloides stercoralis*; bovine parasites, *Bunostomum phlebotomum*; rodent parasites, *Strongyloides myopotami*; and wild dogs, *Strongyloides procyonis.* Larvae of *Gasterophilus* and *Hypoderma* flies and ants of *Solenopsis geminata* species may also cause the same clinical manifestations. Another parasitic larva of dogs that deserves emphasis is the species *Toxocara canis*, which on men can cause visceral and ocular *larva migrans*.

Involvement of the scalp with cutaneous larva migrans is an uncommon even but has been reported with the sebaceous gland representing a facilitated route of entry for the parasite [74, 75]. Concomitant typical lesions on other sites may facilitate the diagnosis.

Depending on the number of lesions and their localization, the treatment can be topical or systemic. The drugs of choice are albendazole 400 mg/day for 3 days, ivermectin 200 mcg/kg in a single dose or tiabendazole 25 mg/kg/day, divided into two doses for 5 days. If there are few lesions, the tiabendazole ointment or cream 10% may be used.

References

1. Otis L. Networking: communicating with bodies and Machines in the nineteenth century. Ann Arbor: University of Michigan Press; 2001. p. 216.

2. McFall-Ngai M. Adaptive immunity: care for the community. Nature. 2007;445(7124):153.
3. Cardé RT. Multi-cue integration: how female mosquitoes locate a human host. Curr Biol. 2015;25(18):R793–5.
4. Mumcuoglu Y, Rufli T. Dermatologische Entomologie. Humanmedizinisch bedeutsame Milben und Insekten in Mitteleuropa. Erlangen: Perimed Fachbuchgesellschat mbH; 1983.
5. Tschandl P, Argenziano G, Bakos R, Gourhant J-Y, Hofmann-Wellenhof R, Kittler H, Rosendahl C, Minas S, Zalaudek I. Dermoscopy and entomology (entomodermoscopy) [Article in English, German]. J Dtsch Dermatol Ges. 2009;7(7):589–96.
6. Conlon JM. The historical impact of epidemic typhus. Archived 2009-11-06 at the Wayback Machine.
7. Mumcuoglu KY, Miller J, Gofin R, Adler B, Ben-Ishai F, Almog R, Kafka D, Klaus S. Epidemiological studies on head lice infestation in Israel. I. Parasitological examination of children. Int J Dermatol. 1990;29(7):502–6.
8. Mumcuoglu KY, Danilevich M, Zelig O, Grinbaum H, Friger M, Meinking TL. Effects of blood type and blood handling on feeding success, longevity and egg production in the body louse, Pediculus humanus humanus. Med Vet Entomol. 2011;25(1):12–6.
9. Haidamak J, Davila Dos Santos G, Lima BJFS, Soares VM, de Menezes RV, Bisson AA, Talevi AS, Gomes RR, Vicente VA, Valero MA, Klisiowicz DDR. Scalp microbiota alterations in children with pediculosis. Infect Genet Evol. 2019;73:322–31.
10. Cummings C, Finlay JC, MacDonald NE. Head lice infestations: a clinical update. Paediatr Child Health. 2018;23(1):e18–24.
11. Shashindran CH, Gandhi IS, Krishnasamy S, Ghosh MN. Oral therapy of pediculosis capitis with cotrimoxazole. Br J Dermatol. 1978;98(6):699–700.
12. Sanchezruiz WL, Nuzum DS, Kouzi SA. Oral ivermectin for the treatment of head lice infestation. Am J Health Syst Pharm. 2018;75(13):937–43.
13. Klaus S, Shvil Y, Mumcuoglu KY. Generalized infestation of a 3 1/2-year-old girl with the pubic louse. Pediatr Dermatol. 1994;11(1):26–8.
14. Armstrong NR, Wilson JD. Did the "Brazilian" kill the pubic louse? Sex Transm Infect. 2006;82(3):265–6.
15. Meinking TL. Infestations. Curr Probl Dermatol. 1999;11:90.
16. Gunning K, Kiraly B, Pippitt K. Lice and scabies: treatment update. Am Fam Physician. 2019;99(10):635–42.
17. Farrell AM, Ross JS, Bunker CB, Staughton RC. Crusted scabies with scalp involvement in HIV-1 infection. Br J Dermatol. 1998;138(1):192–3.
18. Lewis EJ, Connolly SB, Crutchfield CE 3rd, Rest EB. Localized crusted scabies of the scalp and feet. Cutis. 1998;61(2):87–8.
19. Anbar TS, El-Domyati MB, Mansour HA, Ahmad HM. Scaly scalp associated with crusted scabies: case series. Dermatol Online J. 2007;13(3):18.
20. Lai YC, Teng CJ, Chen PC, Chiou TJ, Liu CY. Unusual scalp crusted scabies in an adult T-cell leukemia/lymphoma patient. Ups J Med Sci. 2011;116(1):77–8.
21. Yee BE, Carlos CA, Hata T. Crusted scabies of the scalp in a patient with systemic lupus erythematosus. Dermatol Online J. 2014;20(10):13030/qt9dm891gd.
22. Cohen PR. Concurrent scabies incognito and crusted scabies with scalp lesions masquerading as Erythrodermic dermatitis: scabies surrepticius in an immunosuppressed nonagenarian with hyperkeratotic plaques on her head. J Drugs Dermatol. 2019;18(1):105.
23. Chiu HH, Lan CE. Crusted scabies with scalp involvement in an institutionalized elderly. Indian J Sex Transm Dis AIDS. 2020;41(1):137–8.
24. Meinking TL, Taplin D, Hermida JL, Pardo R, Kerdel FA. The treatment of scabies with ivermectin. N Engl J Med. 1995;333(1):26–30.
25. Jaramillo-Ayerbe F, Berrío-Muñoz J. Ivermectin for crusted Norwegian scabies induced by use of topical steroids. Arch Dermatol. 1998;134(2):143–5.
26. Dourmishev AL, Serafimova DK, Dourmishev LA, Mualla MA, Papaharalambous V, Malchevsky T. Crusted scabies of the scalp in dermatomyositis patients: three cases treated with oral ivermectin. Int J Dermatol. 1998;37(3):231–4.

27. Patel A, Hogan P, Walder B. Crusted scabies in two immunocompromised children: successful treatment with oral ivermectin. Australas J Dermatol. 1999;40(1):37–40.
28. Cestari SC, Petri V, Rotta O, Alchorne MM. Oral treatment of crusted scabies with ivermectin: report of two cases. Pediatr Dermatol. 2000;17(5):410–4.
29. Haas N, Henz BM, Ohlendorf D. Is a single oral dose of ivermectin sufficient in crusted scabies? Int J Dermatol. 2001;40(9):599–600.
30. Yonekura K, Kanekura T, Kanzaki T, Utsunomiya A. Crusted scabies in an adult T-cell leukemia/lymphoma patient successfully treated with oral ivermectin. J Dermatol. 2006;33(2):139–41.
31. Ortega-Loayza AG, McCall CO, Nunley JR. Crusted scabies and multiple dosages of ivermectin. J Drugs Dermatol. 2013;12(5):584–5.
32. Fujimoto K, Kawasaki Y, Morimoto K, Kikuchi I, Kawana S. Treatment for crusted scabies: limitations and side effects of treatment with ivermectin. J Nippon Med Sch. 2014;81(3):157–63.
33. Friedman R. The emperor's itch: the legend concerning Napoleon's affliction with scabies. JAMA. 1940;115(23):2020.
34. Rufli T, Mumcuoglu Y. The hair follicle mites Demodex folliculorum and Demodex brevis: biology and medical importance. A review. Dermatologica. 1981;162(1):1–11.
35. Sengbusch HG, Hauswirth JW. Prevalence of hair follicle mites, Demodex folliculorum and D. brevis (Acari: Demodicidae), in a selected human population in western New York, USA. J Med Entomol. 1986;23(4):384–8.
36. Hellerich U, Metzelder M. Incidence of scalp involvement by Demodex folliculorum Simon ectoparasites in a pathologic-anatomic and forensic medicine autopsy sample. Arch Kriminol. 1994;194(3–4):111–8.
37. Thoemmes MS, Fergus DJ, Urban J, Trautwein M, Dunn RR, Kolokotronis S-O. Ubiquity and diversity of human-associated Demodex mites. PLoS One. 2014;9(8):e106265.
38. Wong CS, Kirby B. Demodicidosis in scalp rosacea? Clin Exp Dermatol. 2004;29:318–9.
39. Signore RJ. A pilot study of 5 percent permethrin cream versus 0.75 percent metronidazole gel in acne rosacea. Cutis. 1995;56(3):177–9.
40. Baima B, Sticherling M. Demodicidosis revisited. Acta Derm Venereol. 2002;82:3–6.
41. Miskjian HG. Demodicidosis (demodex infestation of the scalp). AMA Arch Derm Syphilol. 1951;63:282–3.
42. Sabater-Marco V, Escutia-Muñoz B, Botella-Estrada R. Eosinophilic follicular reaction induced by Demodex folliculorum mite: a different disease from eosinophilic folliculitis. Clin Exp Dermatol. 2015;40:413. https://doi.org/10.1111/ced.12566. [Epub ahead of print].
43. Shah PP, Stein RL, Perry HD. Update on the management and treatment of Demodex blepharitis. Cornea. 2022;41:934. https://doi.org/10.1097/ICO.0000000000002911.
44. Altemir A, Lobato-Berezo A, Pujol RM. Scalp demodicosis developing in a patient with frontal fibrosing alopecia: a clinical and trichoscopic mimicker of active disease. Int J Dermatol. 2021;61:e134–6. https://doi.org/10.1111/ijd.15775.
45. Karaman U, Celik T, Calik S, Sener S, Aydin NE, Daldal UN. Demodex spp. in hairy skin biopsy specimens. Turkiye Parazitol Derg. 2008;32:343–5.
46. Forstinger C, Kittler H, Binder M. Treatment of rosacea-like demodicidosis with oral ivermectin and topical permethrin cream. J Am Acad Dermatol. 1999;41(5 Pt 1):775–7.
47. Mans BJ, Louw AI, Neitz AW. Evolution of hematophagy in ticks: common origins for blood coagulation and platelet aggregation inhibitors from soft ticks of the genus Ornithodoros. Mol Biol Evol. 2002;19(10):1695–705.
48. Sperling LC, Sutton E, Wilke MS. What's eating you? Tick bite alopecia. Cutis. 2016;98(2):88–90.
49. Marshall JJ. Alopecia after tick bite. S Afr Med J. 1966;40:555–6.
50. Heyl T. Tick bite alopecia. Clin Exp Dermatol. 1982;7(5):537–42.
51. Lynch MC, Milchak MA, Parnes H, Ioffreda MD. Tick bite alopecia: a report and review. J Dermatopathol. 2016;38(11):e150–3.
52. Cascio A, Torina A, Valenzise M, et al. Scalp eschar caused by tick bite with rickettsia massiliae. Emerg Infect Dis. 2013;19:836–7.

53. Lipsker D, Boeckler P, Cribiert B. Tick-borne lymphadenopathy/dermacentor-borne necrosis erythema lymphadenopathy: an infectious cause of cicatricial alopecia. Clin Exp Dermatol. 2008;33:518–9.
54. Schwarzenbach R, Djawari D. Pseudopelade Brocq--possible sequela of stage III borrelia infection? [Article in German]. Hautarzt. 1998;49(11):835–7.
55. Bhardwaj EK, Trüeb RM. Acute diffuse and total alopecia of the female scalp associated with borrelia-infection. Int J Trichology. 2015;7(1):26–8.
56. Shamsadini S. Localized scalp hair shedding caused by Pheidole ants and overwiew of similar case reports. Dermatol Online J. 2003;9(3):12.
57. Mortazavi M, Mansouri P. Ant-induced alopecia: report of 2 cases and review of the literature. Dermatol Online J. 2004;10(1):19.
58. Ganjei Z, Reisi-Vanani V, Razavi SZ, Alizadeh M. Ant-induced alopecia: a new differential diagnosis for nonscarring alopecia. Clin Case Rep. 2021;9(8):e04648.
59. Wollina U. Myiasis auf Plattenepithelkarzinomen der Haut. Wien Med Wochenschr. 2015;165(3–4):79–82.
60. Biswas S, Mcnerney P. Interesting case series Furuncular myiasis of the scalp. Eplasty. 2016;16:ic21.
61. Kondoh A, Ota M, Tokuyama M, Makiuchi T, Tachibana H, Mabuchi T. Case of wound myiasis in a squamous cell carcinoma lesion of the scalp. Tokai J Exp Clin Med. 2022;47(2):44–6.
62. Dunphy L, Sood V. Dermatobia hominis "the human botfly" presenting as a scalp lesion. BMJ Case Rep. 2019;12(3):2018–20.
63. Lachish T, Marhoom E, Mumcuoglu KY, Tandlich M, Schwartz E. Myiasis in travelers. J Travel Med. 2015;22(4):232–6.
64. Calderaro A, Peruzzi S, Gorrini C, Piccolo G, Rossi S, Grignaffini E, et al. Myiasis of the scalp due to Dermatobia hominis in a traveler returning from Brazil. Diagn Microbiol Infect Dis. 2008;60(4):417–8.
65. Tileklioğlu E, Yildiz İ, Kozan FB, Malatyali E, Ertuğrul MB, Ertabaklar H. Wound Myiasis in diabetic foot ulcer: Calliphoridae and Sarcophagidae Family. Iran J Parasitol. 2021;16(4):678–85.
66. Wollina U. Massive scalp myiasis with bleeding in a patient with multiple malignancies. Int Wound J. 2010;7(4):297–9.
67. Ben Ghezala H, Feriani N, Snouda S. Myiase du cuir chevelu: une observation exceptionnelle aux urgences. Presse Med. 2016;45(4):469–70. https://doi.org/10.1016/j.lpm.2016.01.027.
68. Brewer TF, Wilson ME, Gonzalez E. Bacon therapy and Furuncular myiasis. JAMA. 1993;270(17):2087–8.
69. Demaj D, Jorgaqi E, Zikaj G, Jafferany M. Furuncular myiasis of the scalp associated with pigmented basal cell carcinoma: first case diagnosed in Albania. Dermatol Ther. 2020;33(1):e13198.
70. Pereyra-Rodríguez JJ, Bernabeu-Wittel J, Conejo-Mir MD, Ruiz-Pérez De Pipaón M, Conejo-Mir J. Treatment of cutaneous myiasis associated with scalp psoriasis in a 13-year-old girl with oral ivermectin. J Am Acad Dermatol. 2010;63(5):908–9.
71. Filho FB, Martins G, Barbará EF, de Paiva MLB, Filho RLPC, da Costa Nery JA. Dermoscopy as an auxiliary tool for the diagnosis of furuncular myiasis. An Bras Dermatol. 2014;89(4):663–5.
72. Gurel MS, Tekin B, Uzun S. Cutaneous leishmaniasis: a great imitator. Clin Dermatol. 2020;38(2):140–51. https://doi.org/10.1016/j.clindermatol.2019.10.008.
73. Mendonça MG, De Brito AEF, Rodrigues EHG, Bandeira V, Jardim ML, Abath FGC. Persistence of Leishmania parasites in scars after clinical cure of american cutaneous leishmaniasis: is there a sterile cure? J Infect Dis. 2004;189(6):1018–23.
74. Guimarães LC, Silva JH, Saad K, Lopes ER, Meneses AC. Larva migrans within scalp sebaceous gland. Rev Soc Bras Med Trop. 1999;32(2):187–9.
75. Meotti CD, Plates G, Nogueira LL, Silva RA, Paolini KS, Nunes EM, Bernardes FF. Cutaneous larva migrans on the scalp: atypical presentation of a common disease. An Bras Dermatol. 2014;89(2):332–3.