Tungiasis

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Abstract
Tungiasis is a parasitic skin disease caused by the female sand flea Tunga penetrans, which burrows into the skin, usually on the feet. The ectoparasitosis is associated with poverty and occurs in many resource-poor communities in the Caribbean, South America and Africa. In some communities, prevalence may be as high as 50% in the general population. In the beginning the lesions are itching, later they become painful. Although tungiasis is a self-limited infection, superinfected lesions lead to formation of pustules, suppuration and ulcers. The diagnosis is made clinically taking into consideration the dynamic nature of the morphology of the lesion. Severe complications occur in heavily infested individuals such as loss of toenails, deformation and auto-amputation of digits. In non-vaccinated individuals the infestation may lead to tetanus. Surgical extraction of the flea under sterile conditions and treatment of secondary infection is the only appropriate treatment currently available.

Keywords
Tungiasis; ectoparasite; *Tunga penetrans*

Disease name/synonyms

Tungiasis; sand flea disease; jigger flea; chigoe; bicho de pé; pulga de bicho; pulga de porco; nigua; puce chique; ogri eye; pique; pico; kuti; suthi-pique; sikka; chica; piqui.

Definition/diagnostic criteria

Tungiasis is an ectoparasitic infestation by the female sand flea *Tunga penetrans*. The diagnosis is made clinically.

Differential diagnosis

Verruca vulgaris; pyogenic infection/abscess; myiasis; foreign body; mycosis; warts; acute paronychia; infected insect bite or sting; dracunculiasis; dermoid cysts; melanoma.

Etiology

Tungiasis is a parasitic skin disease due to the permanent penetration of the female sand flea *T. penetrans* into the skin of its host. The flea undergoes a peculiar hypertrophy, which begins during penetration, and after some days the abdominal segments have enlarged up to the size of a pea. Hundreds of eggs are expelled during a period of about three weeks (Eisele *et al*. 2003). After all eggs have been expelled, the involution of the lesion begins. Three to four

weeks after penetration, the parasite dies *in situ* and eventually is sloughed from the epidermis by tissue repair mechanisms.

Clinical description

Based on clinical and morphological criteria the natural history of tungiasis can be divided into five stages (Eisele *et al*. 2003). In stage I (flea in *statu penetrandi*, 30 min to several hours) a tiny reddish spot of about 1 mm appears. In stage II (beginning hypertrophy, one to two days after penetration) the parasite becomes more obvious as a growing whitish or mother-of-pearl-like nodule develops. In the protruding rear cone of the flea, the anal-genital opening appears as a central black dot, which is surrounded by an increasing erythema. In stage III (maximal hypertrophy, two days to three weeks after penetration) a watch glass-like white patch with well-defined borders and a central black dot appears, often in association with hyperkeratosis and desquamation of the surrounding skin. Expulsion of eggs and faeces are typical in this stage. The lesions are painful and produce the sensation of foreign bodies expanding under the skin. In stage IV (three to five weeks after penetration), a black crust covers an involuted lesion containing a dead parasite. A residual scar in the stratum corneum is characteristic for stage V (six weeks to several months after penetration).

Typically, *T. penetrans* affects the periungual area of the toes, the heels and the soles. However, embedded sand fleas can be found on almost every part of the body, e.g. hands, elbows, neck, buttocks and the genital region (Heukelbach *et al*. 2002b; Heukelbach *et al*. 2003). Severe infestations with more than one hundred embedded sand fleas are not rare.

Although tungiasis is a self-limited infection, severe complications are common in the endemic area (Feldmeier *et al*. 2003). Sequels include deformation and loss of toenails, as well as deformation and auto-amputation of digits. The sore in the skin caused by the protruding rear end of the flea is an entry point for pathogenic microorganisms (Feldmeier *et al*. 2002). Superinfected lesions lead to formation of pustules, suppuration and ulcers. In non-vaccinated individuals tungiasis may lead to tetanus (Obengui 1989; Tonge 1989).

Diagnostic methods

The diagnosis is made clinically taking into consideration the dynamic nature of the morphology of the lesion (Heukelbach *et al*. 2001). The observation of eggs being expelled or attached to the skin around the rear cone and the release of brownish threads of faeces are pathognomonic signs. Faeces threads are of a helical structure and often spread into the dermal papillae. Expulsion of eggs can be provoked by massaging the hypertrophy zone slightly.

Biopsy of the lesion for diagnosis or treatment is not indicated. Histological sections demonstrate the presence of the ectoparasite or chitinous fragments (Franck *et al*. 2003).

Even the untrained physician can diagnose the ectoparasitosis if the typical topographic localisations and the dynamic changes of the clinical picture are taken into account. In endemic areas locals diagnose tungiasis usually with a higher degree of certainty than the untrained physician.

Epidemiology

The sand flea (jigger) *T. penetrans* is one of the few parasites, which has spread from the Western to the Eastern hemisphere. Originally, the ectoparasite occurred only on the American continent. The flea came to Angola with ballast sand carried by the ship Thomas Mitchell that left from Brazil in 1872. Within a few decades, *T. penetrans* spread from Angola along trading routes and with advancing troops in the entire sub-Saharan Africa, including areas with tropical rainforest (Heukelbach *et al*. 2001). At the end of the 19th century the sand flea reached East-Africa and Madagascar. In 1899, returning British troops brought *T. penetrans* to the Indian Subcontinent; the parasite, though, never established there.

Today, tungiasis is found on the American continent from Mexico to northern Argentina, on several Caribbean islands, as well as in almost every country of sub–Saharan Africa (Heukelbach *et al*. 2001). A single case report indicates sporadic occurrence in India (Sane & Satoskar 1985). Recently tungiasis was diagnosed in an Italian lifeguard who worked on the beach of the Tyrrhenian Sea (Veraldi *et al*. 2000). As he never had stayed in an endemic area, tungiasis must have been imported and fleas completed their free-living life cycle on the beach. If global temperature increases further and immigration from endemic areas continues, tungiasis eventually may become endemic on the Mediterranean Coast.

Within the endemic areas the parasitosis has a patchy distribution: it occurs in underdeveloped communities in the rural hinterland, in secluded fishing villages along the coast and in the slums of urban centres. Tungiasis is associated with the presence of dry sandy soils, but may also be found in the rain forest as well as in banana plantations located on laterite soil.

Similar to other parasitic skin diseases, the occurrence of severe tungiasis is linked to poverty. In poor communities in Brazil, Trinidad and Nigeria, point prevalence rates ranged between 16% and 54% (Ade-Serrano & Ejezie 1981;Chadee 1998; Wilcke *et al*. 2002; Muehlen

et al. 2003). Prevalence and parasite burden are correlated, and commonly individuals harbour dozens of fleas. Tungiasis shows a characteristic seasonal variation with highest prevalence in the dry season (Heukelbach *et al*. 2004b).
Tungiasis has been observed in different animals such as elephants, monkeys, cattle, sheep, goats, sylvatic rodents, coatis and armadillos. Domestic animals such as dogs, cats and pigs, but also rats are important animal reservoirs. In a survey in a slum in Northeast Brazil, 67% of dogs and 50% of cats were found to be infested, many of the animals harbouring dozens of fleas (Heukelbach *et al*. 2004a). Rodents also seem to be an important reservoir. In 59% of *Rattus rattus* captured in a poor urban neighbourhood tungiasis was diagnosed. In the rural area, pigs and cattle are known reservoirs for *T. penetrans* (Verhulst 1976; Franco da Silva *et al*. 2001).

Management including treatment
Surgical extraction of the flea under sterile conditions is the appropriate treatment. Fleas should be extracted as early as possible to avoid secondary infections. The existing opening in the epidermis should be carefully widened e.g. with a sterile needle to enable the extraction of the entire flea. If the flea is torn during extraction or if parts are left in the skin, severe inflammatory ensues. After extraction the wound should be treated with a topical antibiotic. Tetanus prophylaxis is indicated.
Closed shoes and socks may prevent tungiasis to a certain degree although complete protection cannot be achieved by these means. Daily inspection of the feet and immediate extraction of embedded fleas protect against complications. Zanzarin®, a repellent based on coconut and jojoba oil has shown to be effective (Schwalfenberg *et al*. 2004).

Unresolved questions
As data on the importance of the different animal reservoirs on human infestation are missing and no efficacious chemotherapy is currently available to kill embedded fleas, the control of tungiasis is a difficult task. Eggs, larvae and pupae may persist in the environment for weeks - if not months - and the reduction of only one type of animal reservoir would result in rapid re-infection of man due to other persisting reservoirs (Heukelbach *et al*. 2002a). It is obvious that reliable and rapid means are needed to determine incidence of tungiasis and severity of disease before and after intervention.
Studies have to be performed to assess the efficacy of chemotherapy as well as to analyse health care seeking behaviour of the population in different settings. It is necessary to investigate the potential of modern insecticides to combat *T. penetrans* in the host and in the environment. It is also imperative to increase knowledge on the pathophysiology of the disease and on the biology and ecology of the parasite and to sensitise all, the affected individuals, the health care profession and the scientific community.

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