Unusual tungiasis: bullous lesions

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ABSTRACT

Tungiasis is a cutaneous ectoparasitosis caused by the gravid female sand flea Tunga penetrans, whose higher prevalence occurs in Sub-Saharan Africa, South America and the Caribbean. We report two males who presented bullous lesions suggestive of tungiasis on their return from Ferrugem (Brazil) and Misiones (Argentina). The diagnosis of tungiasis was confirmed in both cases. The history, epidemiology and biology are reviewed. Some hypothesis about the pathogenesis of bullous tungiasis are suggested (Dermatol Argent 2010;16(5):344-348).

Keywords:

tungiasis, Tunga penetrans, bullous tungiasis.

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Introduction

Tungiasis is a cutaneous ectoparasitosis caused by skin penetration of a gravid female sand flea, *Tunga penetrans* (Phylum *Arthropoda*, Class *Insecta*, Order *Siphonaptera*, Family *Tungidae*). It is also commonly known as *pulga de areia, nigua, pique, bicho dopé, bicho porco or jatecuba*, and in the United States and English-speaking American countries, as *jigger, sand flea, chigoe or burrowing flea*.[1]

Clinical cases

Case 1

A 58-year-old male patient without relevant past medical history who returned from a business trip in the province of Misiones. Consulted for two tense bullous lesions, of serohematic content, located in the foot sole. The blisters had centered black spot and surrounded by an erythematous halo **(Photo 1)**. The patient had local pain, with inability to walk. He reported no history of local trauma, but said that the hotel where he was staying was in reconstruction and walked barefoot on a carpet that had sand on the surface. Biopsy of the central area of the blister and material were sent for histopathology analysis. **Histopathology:** hyperplastic epidermis surrounding a cystic intraepidermal cavity. The dermis presents a mixed infiltrate of lymphocytes, plasma cells and eosinophils. In addition, histological sectioning shows the presence of internal structures belonging to the parasite.

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Case 2

51-year-old male patient, who traveled as a tourist to the south of Brazil (Ferrugem) 1 month prior to the consultation. Presented on the inner side of right foot, a large blister, tense, white, of 4x5 cm in diameter, with a central hole, surrounded by an erythematous halo. It was also observed a residual brownish rounded macule **(Photo 2)**. The patient complained of itching and local pain. The blister was opened and some material of it was sent for histopathology analysis.

Histopathology: epidermis fragment corresponding to the blister surface, presented hyperkeratosis, neutrophils accumulation and areas of epidermal necrosis. It is also observed eosinophilic material of fibrinoid aspect. No microorganisms with PAS are seen **(Photo 3)**.

Treatment

In both cases fleas were extracted, tetanus vaccination, local antiseptic and healing with mupirocin 2%, 2 times per day for 7 days, and 12 mg oral ivermectin in single doses. The clinical outcome was favorable.

Discussion

Tungiasis is a cutaneous ectoparasitosis caused by *Tunga penetrans*, originary from Central America, Caribbean and South America. Subsequently extended to Madagascar, tropical Africa, Pakistan and India's western coast.[2,3] It has high prevalence in Brazil.[4]

This biting flea has little host specificity, besides man, can affect poultry, dogs and pigs. The natural habitat consists of dry soil, sand, shaded and warm, as well as floors of sheds and stables for animals.[3]

In the anatomy of the parasite three parts are distinguished: head, thorax and abdomen. As hematophagous, in the head mouth parts or proboscis are located, composed of rigid jaws and long spikes sharp and protruding. The thorax has three segments that are shorter in front and the abdomen is subdivided into seven well-defined segments, which take a pointed shape in the male and in the female.[5]

The life cycle of *Tunga penetrans* is similar to that of other parasites. Eggs are deposited in the soil and hatch after three or four days. After two weeks, the larva forms a cocoon, where the pupa (or nymph) undergoes a metamorphosis for a period of one week or two until it breaks releasing an adult flea. Copulation involves the death of the male flea and the female pregnant flea survives to penetrate the skin of host. Once inside, it carvs a groove or fibrous sac until his head reaches the dermis and is in contact with vessels of the superficial vascular plexus and the abdominal segment positions parallel to the skin surface. Being a bloodsucking parasite feeds on host blood and increases in size up to 0.6 to 1 cm, at the expense of an abdomen full of eggs. For seven to ten days, the female expels 150-200 eggs per day through its caudal abdominal hole, but after the deposition the female dies, thus completing the cycle.[2,3]

PHOTO 1. Case 1. Blistering Tungiasis.

PHOTO 2. Case 2. Blistering Tungiasis.

Fortaleza classification proposed in 2003,[6] describes the natural history of human infection by *T. penetrans*, differentiating it into five stages:

Stage 1. Penetration phase. Females who have reached the skin begin the host penetration five minutes afterwards. The total phase lasts between three to seven hours, depending on the thickness of the skin. It is usually asymptomatic but it may cause mild erythema.

Stage 2. Hypertrophy of the abdominal segment. It starts one or two days after penetration. An erythematous and pruritic halo is formed with a diameter up to 30 mm

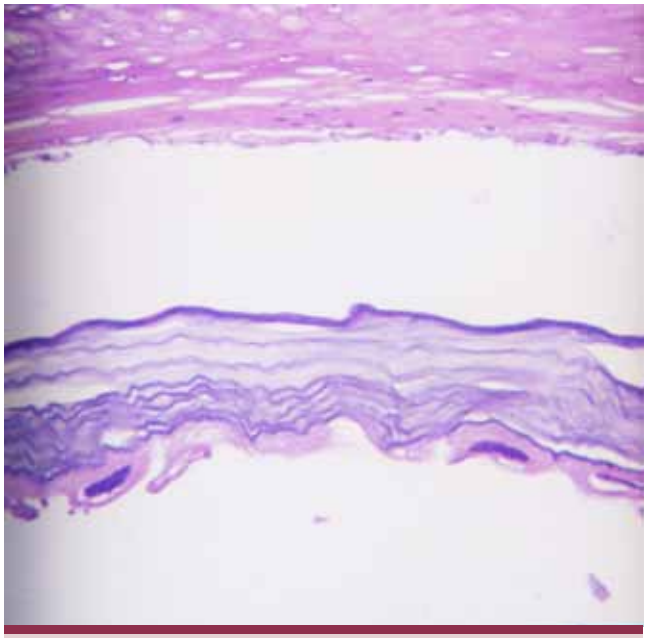

PHOTO 3. Case 2. Histopathology.

around the lesion with a central brown or blackish dot consisting the anogenital area of the flea.

Stage 3. White halo. It starts after 72 hours post-penetration. Hypertrophy is maximal and the abdomen becomes spherical. A white halo appears around the central point, of firm consistence, shaped like a watch glass, associated with brown and yellowish discharge and elimination of fecal material.

Patients complain of a foreign body sensation, throbbing pain, erythema and local warmth. At the stage of greatest development of this stage, eggs are released observing after six days, drying and decreased of consistency, keeping the central blackish brown dot.

Stage 4. Involution phase. The flea dies and stops to release eggs. It begins in the third week and ends on the fifth week, until all traces of the arthropod are deleted. The skin is wrinkled and considerate blackish brown color.

Stage 5. Scar formation in the stratum corneum. The whole process lasts four to six weeks.

Infestation by *T. penetrans* is characterized by the formation of papules with initial erythematous halo and then turning inot a whitish one, with a central black dot **(Photo 4)**.

The clinical appearance of lesions varies with the stage of life cycle of the flea in the human host.

Less common clinical variants have been described: crusted lesions, pustular, bullous, ulcerative and imitation of warts.[7]

A single case was published of the clinical bullous form presented in our patients. The clinical pathogenesis of this tungiasis variety is unknown. Two hypotheses have been raised: the blister is caused by the expulsion of flea lytic enzymes, or it is caused by a reaction to unknown antigens contained in flea saliva.[8]

In our patients eczema was ruled out by contact and history of allergic disease.

The lesions were located preferentially in feet, because the flea jumps are small. Preferentially located on interdigital spaces, sub and periungual regions, soles, back of foot and ankles. Ectopic locations have been described in crouches, testicles, penis, trunk, abdomen, hands, elbows, thighs, buttocks and upper eyelids.[4,9] Our patients had lesions in soles and inside faces of feet. In most cases the injury is unique, but it may be multiple. This is particularly important in patients with leprosy or diabetes due to the lack of sensitivity in the acral area.[10]

The histopathological study shows a hyperplastic epidermis surrounding a cystic cavity with intraepidermal eosinophilic cuticle. The dermis presents a mixed infiltrate of lymphocytes, plasma cells and eosinophils. Moreover, according to the histological cuts, some internal structures of the parasite can be revealed, such as the tracheal rings, sections of the digestive tract, etc. Electron microscopy shows that the surface of eggs has several openings of 1.25 to 2.95 microns.[11]

Tungiasis provokes Th 1 and Th 2 response characterized by an increased concentrations of interferon γ, tumor necrosis factor α and interleukin 4.[12]

Dermoscopy has been used as an effective diagnostic method for this infection, which describes a central brown ring and an area of bluish-black veil arranged in a radial pattern, corresponding to the eggs.[13]

Usually, this disease has a self-limited course, and complications are rare. However, if lesions are multiple, there may be cases of erysipelas, tetanus, cellulitis, gas gangrene, necrosis, sepsis and even death. Infection is the main complication, since the lesion acts as an open gateway.

Bacteria have recently been identified endosymbionts of the genus *Wolbachia* in the ovaries of the flea, whose antigens have been associated with a pathological immune response in diseases such as onchocerciasis, therefore, part of the immune response in tungiasis could be attributed to *Wolbachia* antigens released after the death of the parasite.[14]

Differential diagnosis will be made with pathologies such as acute paronychia, cutaneous larva migrans, foreign body granuloma, biting of *Pulex irritans, Cercaria* dermatitis, plantar wart, acral melanoma and in severe cases with tropical ulcers.[15] In our cases the differential diagnosis could consider blistering of *Dyshidrotic eczema*.

The treatment of choice is the removal of lesions and flea under sterile conditions as soon as possible to avoid infection. One can use a 6 mm punch to remove the flea. After extraction topical antimicrobial are indicated such as 2% mupirocin, applied twice per day for 7 to 10 days.

Tetanus toxoid should be administered to all patients not previously vaccinated or who received their last dose of tetanus vaccine more than five years earlier. When lesions are numerous or pustules are present there may be necessary to use systemic antibiotics.

Ivermectin is indicated orally with single doses of 200 µg/kg.[16]

With regard to prevention, during anti-malaria campaigns, fumigation of soil was carried out with malathion at 1%, resulting in the decrease of tungiasis.[17]
Travelers are advised to use enclosed shoes and avoid sitting down in places that this flea inhabits. It is also recommended the use of repellents in adults and children older than 2 months.[18] Repellents that contain DEET (N, N-diethyl-meta-toluamide) in concentrations of 30 to 50% are effective for several hours.[19]

Conclusions

We believe that although it constitutes an imported disease in our environment, and therefore unusual, it should be think about the possibility of a tungiasis when blackish keratotic lesions at acral areas are presented, and even more if the patient has done some recent travel to endemic areas of this condition. In non-endemic areas, this ectoparasite presents a diagnostic problem. Due to increased international travels to tropical destinations, we consider necessary to inform travelers on both measures for the prevention of this illness as well as doctors on clinical manifestations, and diagnostic and therapeutic behavior.

PHOTO 4. Classic Tungiasis.

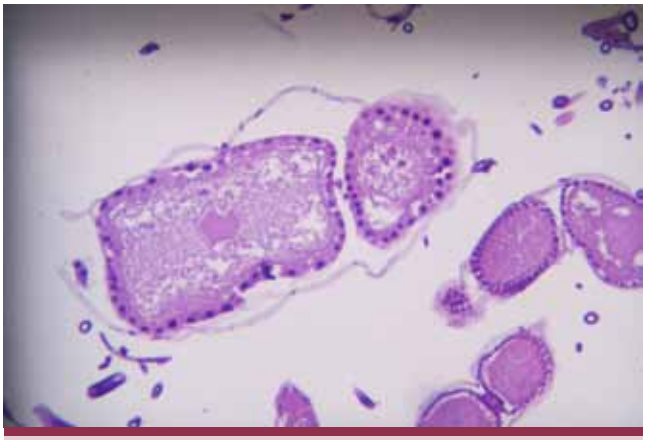

PHOTO 5. Histopathology structures inside the parasite.

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