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Perspective

Life Cycle and Transmission of Tungiasis: A Parasitic Infection

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DESCRIPTION

Tungiasis, sometimes known as "sand flea illness," is a parasitic skin condition brought on by female sand fleas (*Tunga penetrans*), which can infest either people or animals. The *Tunga penetrans* also known as chigoe or jigger flea, is a parasite that attacks humans, pigs, and other animal hosts. A wide variety of domestic and peridomestic animals, including dogs, cats, pigs, and rats, are susceptible to tungiasis, a zoonosis. The risk of infection is high where people interact frequently with these animals and where environmental conditions and social behaviour encourage exposure.

LIFE CYCLE AND TRANSMISSION

The smallest known flea species, *Tunga penetrans*, is only 1 mm in length. Both males and females feed on blood, but only the female may permanently enter its host skin. There, it goes through a significant hypertrophy and releases hundreds of eggs over the course of two to three weeks.

Once all the eggs have been shed, the parasite perishes in its current location before being eventually removed from the epidermis by tissue healing processes. Eggs that land on the ground can become adult fleas in 3 to 4 weeks if they come into contact with a suitable environment. Sand fleas have excellent jumping and running speeds. When the feet or other body parts come into contact with polluted soil or a dirty floor, exposure occurs. Transmission happens both outside and inside of the houses.

The clinical features of tungiasis are covered in numerous case studies. However, they almost all just speak of tourists who get a minor illness after leaving the tropics. According to other investigations, the patients only had one or two lesions, and other than itchiness and local pain no clinical pathology was found. Older studies, however, reveal that native populations, recent immigrants, and deployed military personnel frequently experienced severe disease, which was characterised by deep ulcerations, tissue necrosis that resulted in bone denudation, and auto-amputation of digits, which led to physical disability, such as being unable to work or walk. Lethal tetanus has also been linked to tungiasis in unvaccinated individuals. In a study, 10% of tetanus cases were found to have tungiasis as their point of entry.

SYMPTOMS

The most frequent site of infection is the feet because the tunga flea has a limited ability to jump. The lesion often appears as a white patch with a black dot and develops into an ulceration or punctum. Through the lesion's opening, the flea breathes. The lesion's diameter can be between 4 mm and 10 mm. Even though there may be no symptoms in some cases, the lesions can be severe and extremely irritating. There may also occasionally be redness and swelling near the affected area.

When travelling through endemic areas, it is simple to avoid infestation by wearing shoes. Spraying the ground with an insecticide like malathion can considerably reduce the number of infestations in some flea-prone areas. The main cause of tungiasis in poor, rural populations is still walking barefoot, particularly in children.

Female *Tunga penetrans* fleas typically burrow painlessly at first. Symptoms such as itching and discomfort typically appear as the fleas mature fully and reach the engorged state. Multiple foot lesions can make walking difficult and cause significant inflammation and ulceration. Patients who are in pain avoid putting their entire foot on the ground when they walk, which results in a traditional gait that makes it easy to spot a tungiasis patient from a distance. With tungiasis, secondary bacterial infections such as tetanus and gangrene are not unusual.

The tungiasis lesions were counted and staged as mentioned below according to the Fortaleza classification. The first stage consists of a penetrating sand flea, the second of a brownish/black dot with a diameter of 1mm-2 mm, the third of a circular yellow-white watch glass-like patch with a diameter of 3 mm-10 mm and a central black dot, and the fourth of a brownish-black crust with or without surrounding necrosis. Whereas, the first and third stages are viable, the parasite is dying or has already died in stage four.

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TREATMENT

The primary therapy for burrowing sand fleas in endemic areas is surgical extraction, which is typically carried out by the patients themselves or a caretaker. In non-sterile circumstances, embedded parasites are extracted with implements like sticks, hairpins, sewing needles, or scissors. Children have a difficult time handling the painful treatment. If the flea bursts and releases pathogenic bacteria, the removal of the fleas may result in local inflammation and reinfection of the sore. The instrument is frequently used on multiple people later, increasing the risk of transmission of diseases like HIV or the Hepatitis B Virus (HBV), Hepatitis C Virus (HCV).

Only a skilled community health worker or a clinic with the necessary equipment should do surgical extractions using sterile tools. Sand fleas must be removed, and then the wound needs to be properly bandaged. The patient's tetanus vaccination status must be checked, and if necessary, a booster shot should be administered. In regions where tungiasis is prevalent, increasing the tetanus immunisation rate might offer long-lasting protection. Topical applications of metrifonate, thiabendazole, and ivermectin have been tried, but none have been shown to be adequately efficient. A two-component dimeticone with a specified viscosity, used to cure headlice, is very effective when applied relevantly.