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Painful, slow developing abscesses. Furuncular miyasis due to double skin infestation by Dermatobia hominis

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Abstract

Background: Myiasis is defined as invasion of tissues by Diptera flies. The condition is endemic in the forested areas of Mexico, Central and South America.

Main Observations: A 61-year old woman presented with two boil-like inflammatory and painful lesions on her back. She had been travelling in Central America. Biopsies revealed a myiasis with mature third instar larvae of Dermatobia hominis, a diptera fly endemic in this region. Complete surgical excision and systemic antibiosis led to a delayed but complete healing.

Conclusion: We presented a patient with a double infestation by Dermatobia hominis. Dermatologists should be aware of this disease, which has become increasingly common in travellers and is seen now also in unusual regions, other than Central and South America.

Introduction

Myiasis is defined as invasion of tissues by stages of Diptera flies. In Central and Northern Europe myiasis is uncommon among healthy people. The usual presentation is a myiasis on pre-existing chronic wounds such as leg ulcers. In contrast, myiasis without a wound is a rare occasion. Myiasis has become a common travel-associated dermatosis. Travellers to Central and South America are susceptible to infestation by Dermatobia hominis or human botfly. The condition is endemic to the forested areas of Mexico, Central and South America.[1] However, because of widespread travel, furuncular myiasis has become more common in Europe as well.[2,3] We report a case of furuncular myiasis in a female patient after return from a holiday trip to Guatemala and Belize.

Case Report

A 61-year-old woman presented with two stinging, painful and highly inflammatory boils on her back. She had just returned from a holiday trip to Guatemala and Belize where she stayed for a couple of weeks. The patient was of good general health and had no fever.

Laboratory diagnostics including serology for leishmaniasis, Dengue fever virus, amoebes, rickettsia and typhus were all completely negative. Liver enzymes (GOT and GPT) were slightly elevated, C-reactive protein and blood sedimentation rate were in the upper normal range.

On examination we observed two persistent lesions resembling a boil but having a dark central punctum with serous drainage on her back at a distance of about 1.5cm. Under local anaesthesia we performed a punch biopsy and found deeply located in the dermis on both sites a larva. The specimens were identified as Dermatobia hominis, mature third instar larva. The third-stage larva is elongate ovate, with narrow belts of sparsely set spines, and prominent mouth-hooks. The posterior spiracles have three slits, no button and are sunk in a pit (Fig. 1).

Histopathology showed a cavity containing the larva extending from the epidermis to the mid/lower dermis. The adjacent dermal tissue shows a mixed inflammatory infiltrate that consists of lymphocytes, neutrophils, and eosinophils. There are minor aggregates of plasma cells, some-

times with Russel's bodies. Due to larval enzymatic destruction collagen degradation is notable. In the neighbourhood of the larval cavity one can find a necrotic zone and collagen necrobiosis (Fig. 2). A complete surgical excision of the lesions was performed together with oral antibiotic (doxycyclin 100 mg bid) therapy. Wound healing was delayed.

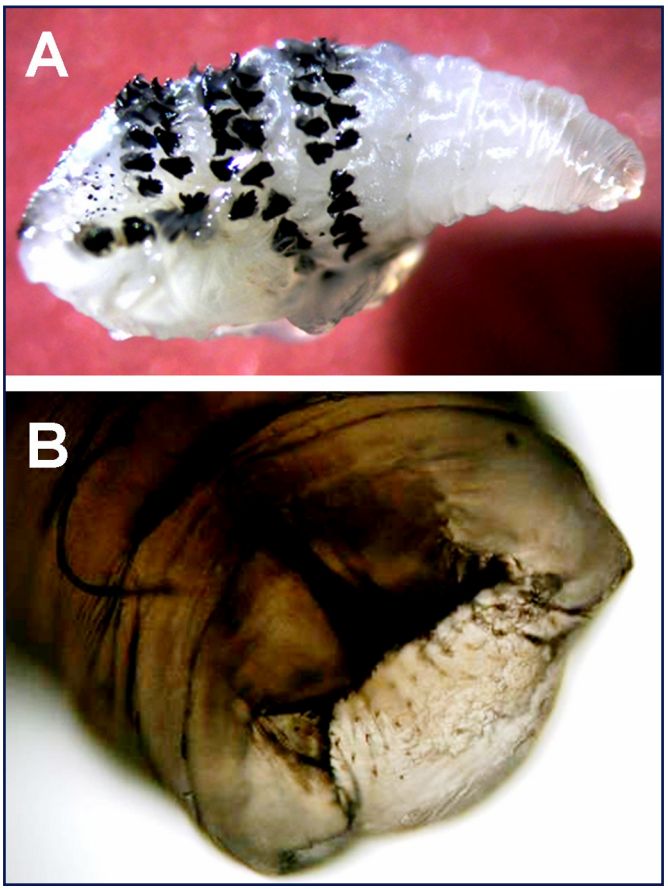

Figure 1
Dermatobia hominis: (A) overview shows an ovoid larva (third stage), (B) detail of the mouth opening.

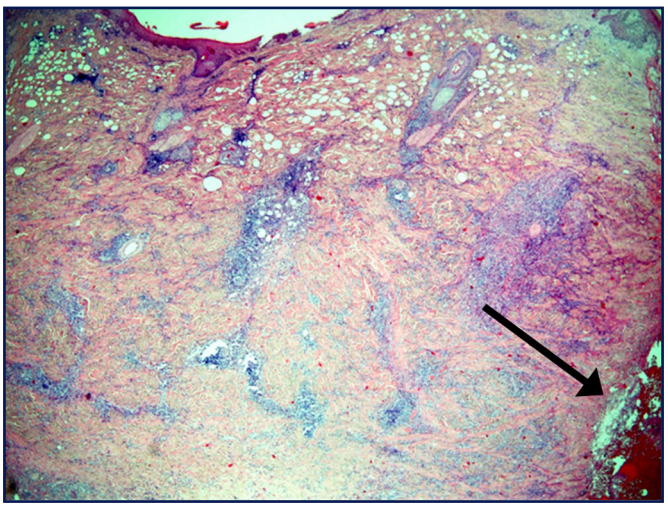

Figure 2
Histology of the adjacent skin. In the upper and mid dermis there is some vacuolization suggesting lipid droplets. Dermal is a granulating and fibrosing inflammation with a mixed inflammatory infiltrate that contains numerous (eosinophilic) granulocytes. The arrow shows the border to the larval cavity (Haematoxylin-eosin, x2).

Discussion

Myiasis due to Dermatobia hominis is known as "ver macaque" in French Guyana, "berne" in Brazil, "torsalo" in Colombia, or "human botfly" in English-language literature.[1] Dermatobia hominis is endemic in Central and South America but only seldom reported from the Caribbean Islands.[4] It causes skin furuncular myiasis at the site of "egg" penetration. The eggs are hatched on to a mosquito vector and injected into the human host as the mosquito feeds. The larvae rapidly burrow into the skin and develop for 50 to 60 days, when the adult larva drops to the ground and pupates. Lesions are painful, with associated inflammation (Fig. 3) and regional adenitis.[1] Ultrasound evaluation has been used in endemic areas, demonstrating sometimes more than one larva in a single boiled lesion.[5] In primary infested hosts, an inflammatory reaction begins slowly, the neutrophils being the main inflammatory cells, eosinophils being scarce. The reaction progresses with time, developing a necrotic halo around the larvae containing inflammatory cells surrounded by fibroblasts. The inflammation invades the adjacent tissue. In repeatedly infested hosts the inflammatory reaction is more rapid and gets stronger with an early involvement of eosinophils.[6] Collagen degradation is seen secondary to larval enzymatic destruction. The cavity around the larva may be epithelialised, and rupture may be associated with a foreign body reaction.[7]

Figure 3
Furuncular miyasis due to double skin infestation by Dermatobia hominis.

In experimentally infested rats (Rattus norvegicus) a variable inflammatory reaction depending on the larval stage of Dermatobia hominis was evident. Two days after infestation, the first-stage larvae were located deep in the dermis, surrounded by an inflamed area infiltrated predo-

minantly by neutrophils. A small warble was formed after the fourth day, increasing in size until the seventh day, when the larvae moulted to the second-stage. The inflammatory process continued with increasing numbers of neutrophils, macrophages, lymphocytes, eosinophils and mast cells invading the area, as well as the proliferation of fibroblasts and endothelial cells and the appearance of a few localized haemorrhages. After 18-20 days, the second-stage moulted to the third-stage larva, when a few plasma cells could be seen in the inflamed area. At 25-30 days there was a reduction in the necrotic layer, as well as in the number of neutrophils and lymphocytes, although large amounts of eosinophils, plasma cells, and collagen fibres were seen. A scar formed after 10 days.[8]

The most frequent differential diagnoses are infected sebaceous cyst, or a furuncle with associated lymphadenopathy. Since other species than Dermatobia hominis may cause infestations the animal should be identified. Almost all cases are present on scalp, limbs and back although infestations of the eye, the breast and the genitalia have been observed.[3,7,9-19]

A case of an erysipelas-like lesion resulting from an unusual infestation with nine larvae of Dermatobia hominis was described. In this case local application of petroleum jelly allowed the exit all larvae.[20]

Topical 1%-ivermectin solution has been also suggested treat myiasis by Dermatobia hominis[1,21], but removal of the parasite is essential to cure the inflammatory process and prevent secondary infections. Therefore, the most effective treatment is complete surgical excision.[2,7]

In conclusion, we presented a patient with a double infestation by Dermatobia hominis. The travel schedule and the typical live cycle of the larva in the host suggest that the miyasis had been acquired in Central America. Surgical treatment is the standard. Dermatologists outside the Americas should be a ware of this disease.

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