Paederus dermatitis

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INTRODUCTION

Paederus dermatitis, also known as dermatitis linearis[1] or blister beetle dermatitis[2] is a peculiar irritant contact dermatitis characterized by erythematous and bullous lesions of sudden onset on exposed areas of the body. The disease is provoked by an insect belonging to the genus *Paederus*. This beetle does not bite or sting, but accidental brushing against or crushing the beetle over the skin provokes the release of its coelomic fluid which contains paederin, a potent vesicant.[3]

ETIOPATHOGENESIS

The genus Paederus belongs to family *Staphyllinidae*, order *Coleoptae*, class *Insecta* and consists of over 622 species which are distributed worldwide.[2,4] Paederus beetles have been associated with outbreaks of dermatitis in various countries including Australia,[5] Malaysia,[6] Sri Lanka,[7] Nigeria,[8] Kenya, Iran,[2] Central Africa, Uganda, Okinawa, Sierra Leone,[9] Argentina, Brazil, France, Venezuela, Ecuador and India.[10-12]

Adults of these beetles are usually 7-10 mm long and 0.5 mm wide, having the size around one and half times that of a mosquito. They have a black head, lower abdomen and elytral (this structure covers the wings and first three abdominal segments) and a red thorax and upper abdomen.[8,9] Paederus beetles live in moist habitats[13] and feed on debris. Although these insects can fly, they prefer to run and are extremely agile. They have a characteristic habit of curling up their abdomen when they run or are disturbed and this habit allows an "on the spot" identification on many occasions.[4] Eggs are laid singly on a moist substance and typically develop in 3-19 days to larvae and adults. Paederus beetles are beneficial to agriculture because they eat crop pests.[9]

The species commonly causing Paederus dermatitis are *Paederus melampus* in India, *Paederus brasilensis* in South America, commonly known as podo, *Paederus colombius* in Venezuela, *Paederus fusipes* in Taiwan and *Paederus peregrinus* in Indonesia.[13]

Paederus are nocturnal and attracted by incandescent and fluorescent lights and as a result, inadvertently come into contact with humans.[9] Hemolymph of the beetle contains paederine (latigaza)[14] which is released on crushing of the insect onto the skin due to the reflex of brushing away the insect. Paederin ($C_{25}H_{45}O_9N$) is an amide with two tetrahydropyran rings and makes up approximately 0.025% of an insect's weight (for *P. fusipes*).

Recently, it has been demonstrated that the production of paederin relies on the activities of an endosymbiont (*Pseudomonas* species) within Paederus. The manufacture of paederin is largely confined to adult female beetles. Larvae and males only store paederin acquired maternally (i.e., through eggs) or by ingestion.[15] It is a vesicant and blocks mitosis at levels as low as 1 ng/ml apparently by inhibiting protein and DNA synthesis without affecting RNA synthesis.[9] Acantholysis is probably caused by the release of epidermal proteases.[2]

CLINICAL FEATURES

The dermatitis may affect persons of either sex, all ages, races or social conditions, since it depends on the patient's activities and insect habitat. The exposed areas are affected with a greater frequency. The incidence of cases is reported to

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be greater during the last quarter of the year, the period that immediately follows the rainy season.[4] The lesions are erythematous and edematous which may be linear, giving a whiplash appearance. The vesicles generally appear towards the center of the plaque. The vesicles turn into pustules quite frequently. The signs appear after 24 to 48h of contact and take a week or more to disappear.[4,8] A striking feature is the presence of "kissing lesions" that occur whenever apposition of damaged areas to previously intact skin is possible, e.g., flexure of the elbow, adjacent surfaces of the thighs.[4]

Diffuse erythematous and desquamative lesions which predominantly occur on the upper body and face have been reported and several possible causes for this atypical variant of paederus dermatitis[2] are:

1. Contact with a different species of Paederus.
2. Recurrent contact during a short period of time.
3. The existence of underlying disorders such as atopic dermatitis.
4. The use of heavily infested natural source of water for washing.
5. An immunologic phenomenon resulting in an eczematized reaction pattern.

Complications include postinflammatory hyperpigmentation, secondary infections, and extensive exfoliating and ulcerating dermatitis requiring hospitalization.[2,5,9]

Ocular and genital involvement is relatively common; it occurs secondary to transfer of the toxic chemical from elsewhere on the skin by fingers. However, ocular area may be the only site of involvement. Ocular involvement usually presents with unilateral periorbital dermatitis, or keratoconjunctivitis, which has been named the Nairobi eye.[2]

HISTOPATHOLOGY

Early lesions show neutrophilic spongiosis leading to vesiculation and eventual reticular degeneration of the epidermis. This is followed by confluent epidermal necrosis, usually with a surviving layer of suprabasal cells. Scattered acantholytic cells may be present. The large number of intraepidermal neutrophils, combined with areas of confluent necrosis and reticular degeneration, are characteristic. Older lesions show irregular acanthosis and pallor of superficial keratinocytes, with overlying parakeratotic scale containing a neutrophilic exudate.[16]

In the early vesicular stage, there are intraepidermal vesicles. The top of the vesicle is usually formed by the horny layer or by one or two rows of flattened cells. The floor consists of the basal cell layer and sometimes one or more strata of the malpighian layer. Inside the vesicle there is fluid and a weft, formed by degenerated epithelial cells ("ghosts"). There is always some admixture of polymorphonuclear cells. The basal cell layer may be intact or indistinct and there may even be destruction of the dermo-epidermal junction.

In the dermis there is a perivascular infiltrate, more marked in the papillae and the upper reticular dermis. It consists mainly of mononuclear cells, but some polymorphonuclear cells are also present. There may be edema, more marked at the papillae. In the full blown pustular stage, the histopathological characteristics mentioned above are exaggerated. The vesicles change into pustules, full of neutrophils, which are seen migrating through the neighboring areas of the epidermis as well as the papillae. The perivascular infiltrate and the edema are more intense. The papillary edema may give a false impression of a subepidermal vesicle.

During healing, the pustule is pushed upwards by maturing new keratinocytes and then may resemble the spongiform pustule of Kogoj. Finally, all that remain are crust, some acanthosis and mild perivascular infiltrate.[4]

Direct immunofluorescence is negative for deposition of immunoglobulins (IgG, IgM and IgA) and C3.[2]

DIFFERENTIAL DIAGNOSIS

Clinical appearance of paederus dermatitis may be confused with herpes simplex, herpes zoster, liquid burns, acute allergic or irritant contact dermatitis, millipede dermatitis and phytophotodermatitis.[2] The characteristic linear appearance of the lesions, their predilection for exposed areas, the presence of kissing lesions, the histopathology, appropriate patch test and finally epidemiological features (occurrence of similar cases in a given area, the seasonal incidence and identification of the insect) should enable the clinician to arrive at the right diagnosis.[4]

TREATMENT

The cases should be managed as irritant contact dermatitis—removal of irritant, initial washing with soap and water, application of cold wet compresses followed by topical steroid and antibiotic, if secondarily infected.[9]

PREVENTION

Preventing human-beetle contact is the primary method of

preventing paederin-based trauma. Tactics that can be employed to achieve this[2,7] include:

1. Learn to recognize paederus beetles and avoid handling or crushing these insects against exposed areas of skin.
2. Keep doors closed and put screens on doors. Both doors and windows should be kept in good repair to reduce the entry of beetles into buildings.
3. Sleeping under a bed net, preferably permethrin-treated, may also reduce the probability of a beetle falling on the skin during the night.
4. A net or mesh can be tied under the lights to prevent the beetle from dropping onto humans.
5. Patients should be advised to avoid crushing the beetle on the skin, manipulating primary lesions or rubbing the eyes following primary involvement.
6. If a beetle lands on the skin, try to remove it gently (example: blow it off, try to get the beetle to walk on to a piece of paper and then remove it). Wash contact areas of the skin.
7. Check areas for beetles (especially on walls and ceiling around the light) before going to bed. Beetle, if present, should be killed by using insecticide (pyrethroid) followed by sweeping up and removal of beetle carcasses. Remember that beetles can cause symptoms, alive or dead and hence avoid handling the beetle directly.
8. Clear excess vegetations from and around the residence, as beetles may rest in these areas.

CONCLUSION

Paederus dermatitis is a common condition. We believe that increased public awareness of this condition can decrease mucocutaneous exposure to paederin.

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Announcement