SKIN LESIONS PRODUCED BY ARTHROPODS

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Abstract - Skin lesions resulting from arthropod exposure may arise via various pathologic pathways. There may be direct damage to human skin from mouthparts, fangs, stingers, etc., or indirect damage such as immune reactions to arthropod saliva or venom injected upon biting. Hypersensitivity may develop against venoms (stinging arthropods) or salivary proteins (biting arthropods). Infectious disease agents transmitted by arthropods may also be responsible for skin lesions. Rocky Mountain spotted fever, Lyme disease, and leishmaniasis are notable examples of arthropod-borne diseases with skin manifestations. Finally, secondary infection may result from arthropod bites or stings (especially as a result of scratching), leading to impetiginous lesions.
Key words - Skin lesions, stings, arthropods

INTRODUCTION

Arthropods including insects, ticks, mites, spiders, and scorpions are a significant cause of human skin lesions (Alexander, 1984; Goddard, 1996; Frazier, 1968; Allington and Allington, 1954) because people are inevitably exposed to biting and stinging organisms in the urban and suburban environment. Skin lesions resulting from arthropod exposure may arise via various pathologic pathways such as direct damage to tissue, hypersensitivity reactions to venom or saliva, or infectious disease (Table1). Even in the absence of allergic reactions to venom or saliva, much human morbidity is due to direct effects (injury) of arthropod biting/stinging. Direct injury can occur from mouthparts or stingers piercing human skin (Goddard, 1994). In addition, secondary infections may result from bacteria entering the skin via the bite/sting punctum. This is especially likely if the bite/sting site is scratched extensively. Vector-borne infectious diseases can produce skin lesions such as rash, ulcers, or eschar.

DISCUSSION

Direct damage to tissue

Some skin lesions are due to direct tissue damage from stings or bites. Arthropod mouthparts puncture the skin by various mechanisms (siphoning tube, scissor-like blades, etc.) leading to skin damage. In this case, damage may be a small punctum, dual puncta (from fangs), or lacerations. Stingers are needle-like structures that may puncture and damage human skin as well. Venom from certain spiders may directly affect human skin, causing tissue death (necrosis). In the U.S. violin spiders are primarily responsible for necrotic skin lesions, although the hobo spider has recently been recognized as a cause of necrotic arachnidism (CDC, 1996). Brown recluse spider venom contains a lipase enzyme, sphingomyelinase D, which is significantly different from phospholipase A in bee and wasp venoms. This specific lipase is the primary necrotic agent involved in the formation of the typical lesions. It is possible that neutrophil chemotaxis is induced by sphingomyelinase D. The subsequent influx of neutrophils into the area is critical in the formation of the necrotic lesion.

Mouthparts

Insect mouthparts can be generally divided into three broad categories: biting and chewing, sponging, and piercing-sucking. Within these categories there are numerous adaptations and/or specializations among the various insect orders. Biting and chewing mouthpart types, such as those in food pest insects, and sponging mouthpart types, found in the filth fly groups, are of little significance regarding human bites, but piercing-

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Table 1. Some skin lesions caused by arthropods.

Lesion Origin	Characteristics	Arthropod(s) Involved	Remarks
Direct Injury			
- Skin puncture	- Punctum, with or without edema and erythema	- Mosquitoes - Ticks - Fleas - Bed bugs - Biting flies - Others	- Sensitized individuals may develop large wheals and surrounding edema
- Necrotic venom	- Local tissue death	- Brown recluse spiders - Hobo spiders	- Hobo spider formerly called aggressive house spider
Hypersensitivity Reaction	- Large local rxn with extensive swelling or systemic rxn with urticaria	- Mostly stinging arthropods - Sometimes biting flies or kissing bugs	- Can be fatal; patient needs evaluation by a physician
Infectious Disease	- Rash - Ulcers - Eschar - Sandflies	- Mosquitoes - Ticks - Fleas	- Indirect effect of arthropod bite

sucking mouthparts, and especially the bloodsucking types, are of considerable importance. Insect piercing-sucking mouthparts vary in the number and arrangement of the stylets, which are needle-like blades, and the shape and position of the lower lip of insect mouthparts, the labium. Often, what is termed the proboscis of an insect with piercing-sucking mouthparts is an ensheathment of the labrum, stylets, and labium. These mouthparts are arranged in such a way that they form two tubes. One tube is usually narrow, being a hollow pathway along the hypopharynx, and the other is wider, formed from the relative positions of the mandibles or maxillae. Upon biting, saliva enters the wound via the narrow tube, and blood returns through the wider tube by action of the cibarial or pharyngeal pump.

Sting apparatus

In all stinging wasps, bees, and ants the stinger is a modified ovipositor, or egg-laying device, that may no longer function in egg laying. Accordingly, in the highly social Hymenoptera only a queen or other reproductive caste member lays eggs; the workers gather food, conduct other tasks, and can sting intruders. A typical ovipositor (nonstinging) consists of three pairs of elongate structures, called valves, which can insert the eggs into plant tissues, soil, etc. One pair of the valves makes up a sheath and is not a piercing structure, whereas the other two pairs form a hollow shaft that can pierce substrate in order for the eggs to pass down through. Two accessory glands within the body of the female inject secretions through the ovipositor to coat the eggs with a gluelike substance.

For the stinging configuration, the ovipositor is modified to enable a stinging function. The genital opening from which the eggs pass is anterior to the sting apparatus, which is flexed up out of the way during egg laying. Also, the accessory glands have been modified. One now functions as a venom gland and the other, called the Dufour's gland, functions in a yet unknown way. The venom gland is connected to a venom reservoir or poison sac, which may contain up to 0.1 ml of venom in some of the larger hymenopterans.

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The stinger itself is well adapted for piercing the skin of vertebrates. In the case of yellowjackets there are two lancets and a median stylet that can be extended and thrust into a victim's skin. Penetration is not a matter of a single stroke, but instead by alternate forward strokes of the lancets, sliding along the shaft of the stylet. The tips of the lancets are slightly barbed (and actually recurved like a fishhook in the case of honeybees) so that they are essentially sawing their way through the victim's skin. Contraction of venom sac muscles injects venom through the channel formed by the lancets and shaft. The greatly barbed tip of the lancets in honeybees prevents the stinger from being withdrawn from vertebrate skin, thus the sting apparatus is torn out as the bee flies away. Other hymenopterans, on the other hand, can sting repeatedly.

Reactions to venom/saliva

Very little lesion development is actually due to physical puncture of human skin. Most lesion development is due to immune reactions to venom or salivary components. Arthropod venoms are highly complex mixtures of pharmacologically and/or biologically active agents. Since some venoms are similar, there may be cross-reactivity reactions in humans, but not always. Histamine is the most predominant low molecular weight component. Serotonin, dopamine, noradrenalin, and acetylcholine are also usually present in venom. The amount of serotonin seems to be directly related to the painfulness of the sting. Melittin is a protein polypeptide toxin that is a primary constituent of honeybee venom. It is a direct agent of hemolysis. Apamin is one of the smallest polypeptides known (molecular weight 2038). It is a neurotoxin and its interaction with the spinal cord is well established. MCD (mast-cell degranulating) peptide, as a mastocytolytic agent, is very effective in releasing histamine. MCD peptide only comprises approximately 2% of bee venom but can produce effects equal to that of melittin, comprising as much as 50% of bee venom. Kinins take an intermediary position between biogenic amines and high molecular weight compounds. The relative importance of kinins in envenomization is yet to be clarified. Phospholipase A is an enzyme that can attack structural phospholipids resulting in damage to biological membranes, mitochondria, and other cellular constituents. There are at least two types of phospholipase: A_1 and A_2. Phospholipase A_2 is present in honeybee venom, whereas vespid (wasps, yellowjackets, and hornets) venoms contain phospholipase A_1. Hyaluronidase is a spreading factor that helps open the way for other venom components to move through host tissues. It works by hydrolyzing hyaluronic acid, which resists the spread of harmful substances through epithelial and connective tissue. Honeybee allergens are phospholipase A_2, hyaluronidase, acid phosphatase, and melittin. Allergens contained in vespid venoms are phospholipase A_1, hyaluronidase, and a protein called antigen 5.

An allergic dermatitis, characterized by eczema-like eruptions on the skin, may develop later in response to insect or mite body parts, saliva, or feces secondary to the immediate reaction. Delayed-type hypersensitivity reactions typically appear over a period of several days, perhaps not maximal until 48 or 72 hours after antigen exposure. This is cell-mediated immunity wherein CD4-positive T lymphocytes react with antigen and release lymphokines into tissues. These lymphokines may serve as attractants for monocytes. A late phase response in allergic individuals may appear 2 to 8 hours after challenge and is characterized by a dramatic influx of immune and inflammatory cells to the site of antigen exposure. Along with this influx of cells is a second wave of inflammatory mediators.

People may develop hypersensitivity to salivary secretions as well, resulting in allergic reaction to bites. However, systemic hypersensitivity reactions to arthropod bites are much less common (almost rare) than those resulting from stings. The groups most often involved in producing systemic effects by their bites are the kissing bugs (genus *Triatoma*), blackflies, horseflies, and deerflies. Even tick bites may sometimes cause extensive swelling and rash. Some examples are the hard ticks, *Ixodes holocyclus* and *Amblyomma triguttatum*, and the soft tick, *Ornithodoros gurneyi*. Arthropod saliva from biting insects contains anticoagulants, enzymes, agglutinins, and mucopolysaccharides. Presumably, these components of saliva serve as sensitizing allergens.

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Lesions from infectious diseases

Some arthropod-caused human skin lesions develop neither from direct injury nor allergic reactions, but instead, from an infectious disease process. Many vector-borne diseases are characterized by rashes. Rocky Mountain spotted fever causes a maculopapular rash that often begins on the palms of the hands and subsequently moves to the trunk. Murine typhus produces a macular rash on the trunk. The Lyme disease A rash or lesion, erythema migrans, typically begins as a red macule or papule, expanding over a period of days or weeks to form a large round lesion, often with central clearing. A few tick-borne diseases such as Boutonnneuse fever and African tick bite fever produce an eschar (tache noire) at the site of tick bite (CDC, 1998). The black, button-like lesion develops with a central dark necrotic area which may be accompanied by local swelling of the lymph glands. Leishmaniasis, a sandfly-transmitted parasitic disease, produces skin lesions ranging from small, round ulcers that are slow to heal, to huge eroded areas of nose/mouth tissue. Cutaneous leishmaniasis begins as a papule which gradually increases in size, becomes crusted, and ulcerates. The ulcer is often circular and shallow with raised, well-defined erythematous borders. There may or may not be a serous discharge. Mucocutaneous lesions develop in less than 5% of patients, typically after months or years, and usually follows cases of cutaneous leishmaniasis caused by *Leishmania braziliensis* or *L. panamensis*. It is believed that localization in the nasal mucosa occurs during parasitemia associated with the initial infection. The disease may be severely disfiguring, eroding the cartilaginous tissues of the nose and palate.

CONCLUSIONS

A human's first line of defense against invasion or external stimuli is the skin (Alexander, 1984). It may react in a variety of ways against all kinds of stimuli, physical or chemical, including arthropods and their emanations. Skin lesions may result from arthropod exposure, although not all lesions have the same pathological origin, some are due to mechanical trauma, some due to infectious disease processes, and some result from sensitization processes. Physicians and other health care providers are frequently confronted with patients having skin lesions attributed to a mysterious arthropod bite. Diagnosis is difficult, but may be aided by asking the patient numerous questions about the event and any recent activity which might have led to arthropod exposure. Questions like, Did you see the offending arthropod?; Was it worm-like?; Did it fly?; Where were you when these lesions occurred?; might provide useful information. Most treatments (except in cases of infectious diseases) involve counteracting immune responses to venoms, salivary secretions, or body parts using various combinations of antihistamines and corticosteroids. Infectious diseases may require aggressive antibiotic/supportive care.

REFERENCES CITED

Alexander, J. O. 1984. Arthropods and human skin. Berlin: Springer-Verlag, 422 pp.
Allington, H. V. and R. R. Allington. 1954. Insect bites. J. Amer. Med. Assoc. 155: 240-247.
CDC. 1996. Necrotic arachnidism B Pacific Northwest. CDC, MMWR. 45: 433-436.
CDC. 1998. African tick bite fever among international travelers B Oregon, 1998. CDC, MMWR. 47: 950-952.
Frazier, C. A. 1968. Diagnosis of bites and stings. Cutis. 4: 845-850.
Goddard, J. 1994. Direct injury from arthropods. Lab. Med. 25: 365-371.
Goddard, J. 1996. Physician's guide to arthropods of medical importance, 2nd Ed. Boca Raton: CRC Press, 374 pp.

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