EDITORIAL

(The editorials are written by the members of the Editorial Advisory Board or by guest writers. The editorials express the personal views of the writers.)

CUTANEOUS REACTIONS TO INSECT BITES — SARCOPTES SCABEI NOT EXCUSED

Insect bite reactions are a common cause of morbidity, but it is not known, how often they cause death under labels of acute systemic disease involving the Nervous, Cardiovascular and Haemopoietic systems. It should not be a surprise that cutaneous effects are a common cause of dermatologic consultation, as the association between the world's oldest and the most recently evolved inhabitants is often intimate. Seasonal variations are usual. They arise as much from breeding habits of insects as from changes in man's hobbies and occupation. The last named, results from agricultural requirement, leading to periodic movement of population from cities to villages and back. A shift in residence from cities to suburbs or vice versa, causes exposure of whole families to new vectors.

The dominant factor in the pathogenesis of insect bite reaction patterns is allergic transformation of the repeatedly assaulted individual. This holds pests true for the commonly encountered viz. mosiquitoes, fleas, bed bugs, ants and mites. Sensitivity to more than one species is not unusual. The initial reaction which is transient, consists of erythematous, edematous, and rarely bullous lesions. With repeated exposures, the classical picture of papular urticaria, characterised by erythematous edematous firm papules often capped by a vesicle, is seen to emerge (Fig 1). The most characteristic mode of healing of these lesions is central hypopigmentation surrounded by a zone of hypermelanosis; both together measuring 4-10 mms in diameter (Fig 2). Eventual disappearance of reactivity is expected to be replaced by a phase of tolerance. This stereotyped behaviour, however, is not invariable and there are individuals who never lose their sensitivity. This accounts for recurrent attacks of papular urticaria and recidivative phenomena. The latter though reported with certain bugs, are also commonly seen when other vectors are the cause. In the most classical form a single bite is followed by exacerbation of pre-existing lesions, recurrence at healed sites, and generalized pruritus. Sometimes fever with lymphadenopathy and rarely phlyctenular conjunctivitis or episcleritis accompany each attack.

Eczematous changes (Fig 3) around lesions, a nummular presentation, palmo-plantar ids and even a picture indistinguishable from the Mucha-Habermann disease may emerge in sensitized and usually young subjects. The intense pruritus generated by these is a common cause of pyoderma, lymphangitis, cellulitis and lymphadenitis.

As in any other allergic process, variations in response are to be expected. A heightened reactivity often follows a viral exanthem. Indeed, the first attack of papular urticaria may follow such an episode. As vaccination is

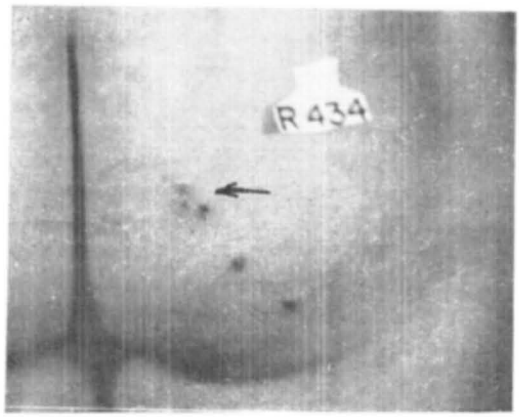


Fig. 1 The cluster of three (arrow) is typical of flea bites but the entire group could still be caused by bed bugs and Strongyloides stercoralis larvae.

being performed on neonates, a similar precipitation is being observed and seems to explain the occurrence of papular urticaria in infants below 4 months of age. A similar heightened reactivity is observed in the initial stages of a malignant lymphoma, including Hodgkin's disease. These papulovesicular lesions may be confused with the nonspecific lesions of these disorder, but a good history and protective measures clear the cobwebs. With relentless progress of the disease or the immunologic paralysis induced by drugs used for their treatment the lesions cease to arise. Similarly, in patients with organ transplants the picture is one of gross local edema, erythema, haemorrhages and necrosis rather than papular urticaria.

A more chronic form presents with pigmentation, papular and excoriated lesions, intermixed with small areas of hypopigmentation and scaling (Fig 4). The favoured sites are extensor aspects of the upper extremities extending to the scapular region, and the lower extremities upto the gluteal zone. This picture tends to be more common when man and animals live in close proximity, and suggests an easy, exchange of vectors like sarcoptic mites. In one such instance, (association with pigs) the histology revealed acanthosis, compact hyperkeratosis and tunneling of the epidermis. These observations are important because it is not unusual for papular urticaria and scabies to present simultaneously in the same patient. The overlap in symptomatology suggests that the nonhuman sarcoptic mites share a common antigen with the human variety. This explains why an occasional patient with animal scabies needs treatment with antiscabetic preparations, and why, after cure of human scabies vesicular lesions recur after bites by unidentified insects. A patient may receive treatment for both disorders alternately ; This may appear outre, yet it is thus.

Mistaking insect bite reactions for dermatitis herpetiformis will continue as long as eosinophils are regarded as diagnostic cells of the latter disease.

Bullous lesions may accompany other allergic manifestations, but in a most dramatic manner, they result from crushing of venomous insects on the skin

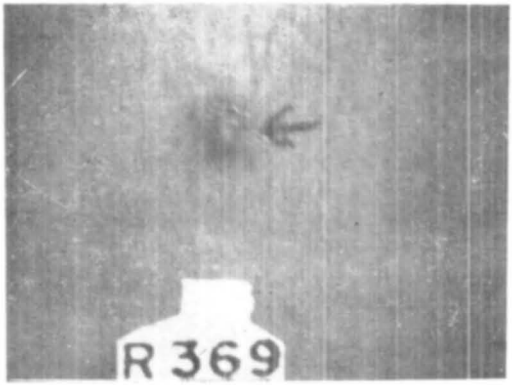


Fig. 2 Characteristic colour changes in a subsiding lesion. With complete healing the contract increases.

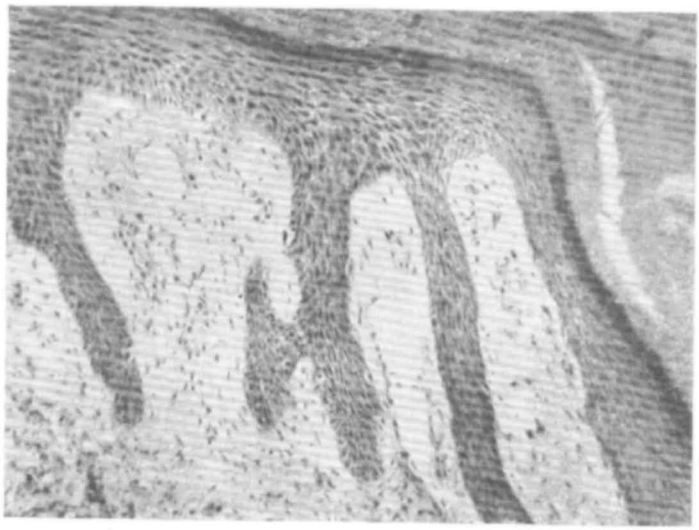


Fig. 2 A H.E. 100 X of figure 2 shows a subacute dermatitis. The lesion is in a healing phase, as the granular layer has reformed under a mass of hyperparakeratotic stratum corneum. Keratinocytic nuclear polychromasia indicates increased mitotic activity, a prerequisite for healing. Large numbers of fibroblasts in the dermis indicate dermal repair in progress.

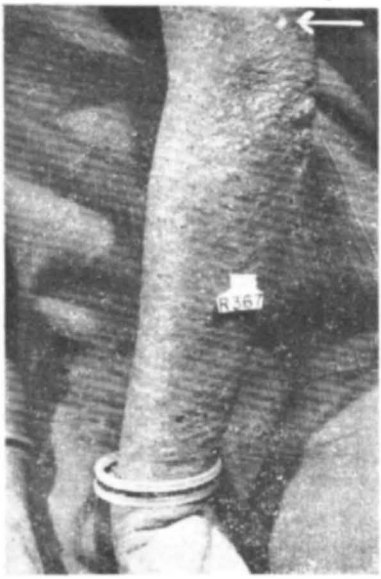


Fig 3 Mosquito bite followed by eczematization and secondary infection. The involved site had become intolerant to solar irradiation and remained so, for sometime after healing.

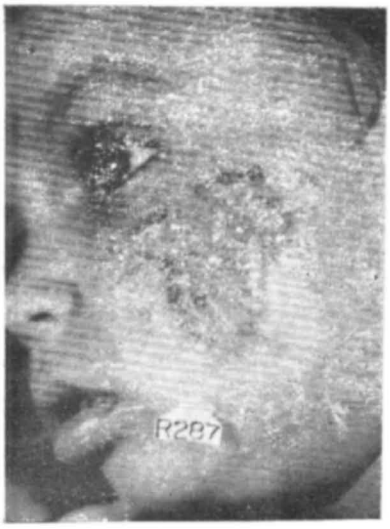
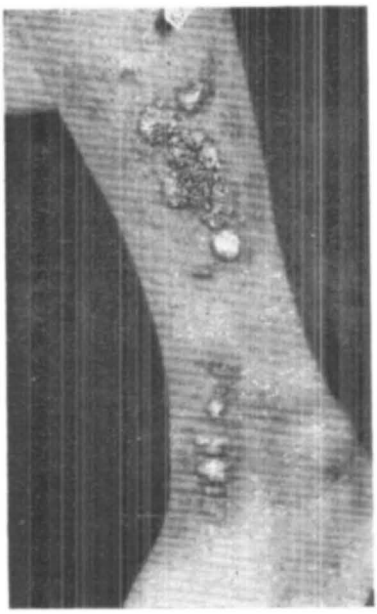


Fig. 4 Extensive eruption arising out of frequent and repeated assaults. Lesions were papular, papulovesicular, scabbed, pigmented, scaly and depigmented (arrow). Histopathology may simulate scabies.



◀ **Fig. 5** Prurigo nodularis resulting from persistent itching at sites bitten several years earlier. These lesions may participate in recidivating phenomena.

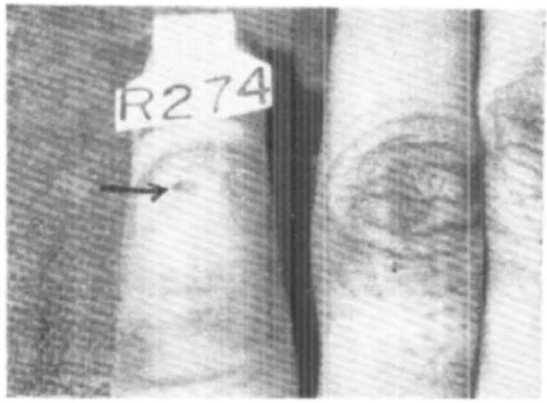


Fig 6 Arrow points to the site of a cockroach bite on the little finger. Pus formation and edema were subsequent complications.

Fig. 7 Lesions of lichen planus but smaller than usual; some showed a punctum caused by mosquito bites.

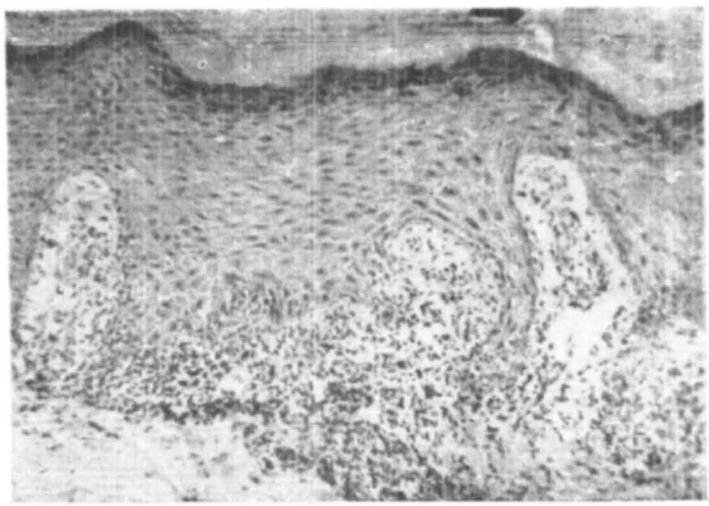


Fig 7A H.E 100 ×. Histology from one of the lesions from Fig. 7 Though typical of lichen planus, the stratum corneum (arrow) is speculative of a proboscis effect.

surface. Healed lesions of this type leave linear trails of a brownish colour, and when suitably located are indistinguishable from berloque melanosis.

Bullous as well as acute ulcerative lesions often develop because of powerful pharmacologic agents injected into the dermis during a bite. These do not represent allergic reactions, and clinically simulate anthrax and acute vasculitis. Chronic ulcers arising out of retention of parts of the insect, need differentiation from disease processes, resulting from deposition of organisms of which the insect acts as a vector. The bluish black periphery and central scab have inspired clinical diagnoses of pigmented basal epithelioma and Kaposi's haemorrhagic sarcoma. Histologically, they are more often mistaken for histiocytomas and melanomas than mycosis fungoides. Healing of dermal damage may not only cause keloids, but set up a more sinister process of pseudosarcomatous fasciitis.

Histological changes of papular urticaria are those of acute or subacute dermatitis (Fig 2A). Though eosinophils constitute a fair proportion of the dermal infiltrate, they are not a sine qua non for diagnosis. In immunosuppressed organ transplant patients the reaction pattern is characterised by a jelly like dermis with a mononuclear infiltrate and areas of haemorrhages. The hallmark of immunosuppression is reflected in the epidermis as stacking of epidermal cells. This signature is hardly capable of being forged by any epidermal disorder.

Sometimes a localized lesion remains pruritic for so long that it evolves into that of prurigo nodularis (Fig 5), with a corresponding histopathology.

Recurrent, superficial, jagged ulcers with scabs, 2 to 3 mms in diameter, located on the fingers are traceable to cockroach bites (Fig 6). They seem to be caused by food remnants or their odors attracting these pests.

Insect bites constitute a physical or chemical injury, hence all disorders associated with a Koebner phenomenon undergo exacerbation. This is most convincingly observed in psoriasis and lichen planus (Fig 7), with their diagnostic histology (Fig 7A). Rarely vitiligo may be so associated (Fig 8).

Fig. 8 The sequence of events was hyperthyroidism, vitiligo, bug bites with erythema and edema (but no scabbing) healing with irregular areas of vitiligo (arrows).

In conclusion insect bite reactions whether mediated through physical, chemical or immunologic mechanisms, lead to morbidity. The immunologically altered individual reacts differently from one not so involved. Several aspects of scabies become clearer if the Sarcoptes scabei var humanus is viewed as an insect sharing antigenic similarity with other mites. Finally, the interactions between insect bite effects and other dermatoses alter the latter's behaviour.

— **V. R. Metha**
Dept. of Dermatology, Leprology & Venereology,
LTMG Hospital and LTMM College,
Sion, Bombay-400022