PERSISTENT "INSECT BITES" (DERMAL EOSINOPHILIC GRANULOMAS) SIMULATING LYMPHOBLASTOMAS, HISTIOCYTOSES, AND SQUAMOUS CELL CARCINOMAS *

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In 1942, opportunity was afforded at the Army Institute of Pathology to review the histologic slides of a lesion said to have been produced by a tick bite. The microscopic sections seemed at the time indistinguishable from mycosis fungoides or Hodgkin's disease, especially in view of the presence of multiple lesions in the patient. However, following the study of the cutaneous reactions to arthropods (ticks, mosquitoes, and chiggers), it was quickly appreciated that not only were these diagnoses of neoplasia wrong but that the misinterpretation of these reactions was a common and serious error.[1] The errors involved the misconstruction not only of the dermal reaction but also of the epidermal changes. The latter response was confused with squamous cell carcinoma; the dermal infiltrate was mistaken for mycosis fungoides, Hodgkin's disease, lymphosarcoma, giant follicular lymphoblastoma, and Spiegler-Fendt sarcoid. Undoubtedly the principal reason for the failure to attribute these reactions properly to bites of arachnida and insects was referable to the general impression, despite clear-cut clinical histories, that such reactions last only for days, whereas, in truth, they may persist for as long as 2 years. More recently, the problem has been further complicated by introduction into the literature of a lesion called "eosinophilic granuloma of skin," an entity of questionable nosologic justification.[2-4] Therefore, because of the major importance of establishing a definitive diagnosis and because of the interest in the pathogenesis of a much mimicked histologic pattern, it was felt of use to record the experience in this matter.

MATERIAL

There were available for study the histologic preparations of reactions to "bites" of 20 arthropods, including 9 ticks, 4 mosquitoes, 3 chiggers ‡ ("red bugs"), and 4 insects not otherwise identified. For comparison there were available the sections of 30 eschars of scrub typhus (tsutsugamushi disease) and 2 sections of skin infected with the ovum or larva of worms. The eschars produced by mites of the

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‡ The "chiggers" are the larval mites of *Trombicula irritans* and are to be distinguished from "jiggers," or adult sand fleas (*Pulex penetrans*).

species Trombicula offered no diagnostic problems inasmuch as they were all from active cases of scrub typhus. No examples of the late residue of such eschars were studied.

The lesions occurred on the extremities in 8 cases, on the abdomen in 5, on the back in 3, on the penis in 2, and in the axilla in 2. Their duration varied from 3 weeks to approximately 2 years; 6 were present longer than 1 year. All but one of the patients were white; in the one Negro, the lesion, following a tick bite, lasted 15 months before it was excised. Two of the group were females. The ages ranged from 21 to 49 years. In 3 instances, the ages were not recorded. Twelve of the 20 bites were acquired in California, Georgia, Texas, and Louisiana. The clinical appearance of the lesions varied from a patch of eczema such as might be caused by an irritant of dermatitis venenata, to a smooth, firm, erythematous, persistent, pruritic, slowly growing or stationary papule. The latter form of lesion simulated a fibroma. Some of the lesions were excoriated or ulcerated; some oozed, formed crusts, and desquamated. One of the lesions on the penis suggested a syphilitic chancre. There was a tendency for the ulcerations to heal, to recur subsequently, and to heal again. In such lesions a portion of the tick was left embedded in the skin. Marked pruritis was a particularly prominent and constant symptom. The lesions varied from 1 to 7.5 cm. in maximum diameter. It was generally impossible to estimate the duration from their clinical appearance.

Findings

The histologic picture as stated had two distinct components which might or might not be combined: (1) changes in the epidermis and (2) the dermal infiltrate.

Epidermal Reaction

The most striking feature of the epidermal reaction was the marked pseudo-epitheliomatous hyperplasia which occurred in 7 of the 20 instances (2 chiggers, 2 ticks, and 3 unidentified). In 6 of the 7 cases, lesions with the epidermal hyperplasia were submitted to the Institute with the incorrect diagnosis of squamous cell carcinoma (Figs. 1 to 4). The pseudo-epitheliomatous hyperplasia was quite like that often seen at the margins of cutaneous ulcers. The differentiation of pseudo-epitheliomatous hyperplasia from carcinoma was by no means always a simple matter inasmuch as a disruption of the epidermal basement membrane and an excessive number of mitotic figures in the advanced portions of the rete pegs were occasionally present (Fig. 4). The diagnosis of hyperplasia rested on the conclusion that the isolated epi-

dermal nests were not truly invasive but were obliquely cut pegs actually continuous with the epidermis. Moreover, if the cells in mitosis were excepted, the other cells in these deep nests were essentially as differentiated as those of the neighboring uninvolved epidermis. The disruption of the basement membrane was regarded as the effect of the adjacent extensive, compact, inflammatory reaction. In this connection it was noted that pseudo-epitheliomatous hyperplasia was in each instance accompanied by marked dermal inflammation and was most pronounced over the densest portion of the dermal reaction.

Other changes in the epidermis included varying degrees of spongiosis (Fig. 6) which in 4 lesions (2 ticks, 1 chigger, 1 unidentified) reached the degree of vesiculation (Figs. 3 and 5). The vesicles were multiloculated, eczematous collections of serum containing purulent exudate or, as occurred in one instance, masses of eosinophilic leukocytes. The roof of the vesicles and the adjacent epidermis tended to be parakeratotic. Often loosely scattered neutrophilic and a few eosinophilic leukocytes were located in the epidermis, particularly in the spongiotic areas (Fig. 6). In the absence of pseudo-epitheliomatous hyperplasia a relatively uniform acanthosis, usually associated with hyperkeratosis and focal parakeratosis, was present. Occasionally the hyperkeratosis took the form of prominent keratinous plugs of follicles. The acanthosis was of a nonspecific variety but in some lesions simulated that found in neurodermatitis.

Dermal Infiltrate

The dermal inflammatory reaction was in most instances characterized by concentrated masses of cells involving the entire thickness of the dermis and part of the hypodermis, or distributed in dense patches in various portions of the dermis. The reaction showed no predilection for the cutaneous appendages but was often primarily collected about blood vessels. Whereas pseudo-epitheliomatous hyperplasia was accompanied by abundant dermal reaction, the reverse was not necessarily true. The important features of the infiltrate included: (1) Its density and usually great extent. (2) The presence of numerous mature eosinophilic leukocytes reaching a concentration of as many as ninety per high-power field. (3) The large numbers of plasma cells which varied greatly in size, many with two nuclei and rarely three, and some of them indistinguishable from the giant plasma cells of mycosis fungoides. (4) The abundance of histiocytes with cytoplasm which appeared to have undergone diffuse hydropic swelling and reacted negatively to stains for glycogen and fat. Mast cells were not increased. (5) Finally, there were noted conspicuous lymph follicles

(5 of the 20 cases) with definite germinal centers. It is of interest that eosinophilic leukocytes were excluded from the germinal centers despite their presence in large numbers in the adjacent infiltrate.

In addition, mitotic figures were observed in histiocytes in 5 of the lesions; karyorrhexis with phagocytosis of chromatin was common. Binucleate histiocytes with partially overlapping nuclei closely resembling the Sternberg-Reed cells of Hodgkin's disease also were found occasionally. In 3 lesions subepidermal edema, almost urticarial, was present; in only one of these was the process of short duration (Fig. 14). In 3 instances there were subendothelial edema, swelling of endothelial cells, and a few polymorphonuclear leukocytes in the intima of dermal arteries and veins, a reaction reminiscent of one form of allergic inflammation of vessels. In 4 other lesions (1 tick, 1 mosquito, and 2 unidentified) epidermal inclusion cysts were found in the mid-dermis in the core of the infiltrate. In one lesion portions of the tick actually were included in the center of an epidermal cyst (Figs. 11 and 12). In 2 cases there was noteworthy proliferation of the squamous cells lining these cysts. Foreign body giant cells were present in the vicinity of the cyst, possibly representing a reaction to the keratin of the epidermal inclusion or to broken off remnants of the arthropod. The apparent integrity of the cyst with no obvious extrusion of keratin and the association of eosinophilic leukocytes with the giant cells suggest that portions of the arthropod or its products might be the inciting agent.

Comparative Lesions

The acute lesions of the eschars of scrub typhus of approximately 9 to 27 days' duration differed strikingly from the reaction to other arthropods, essentially in the almost complete absence of eosinophilic leukocytes from the dermal infiltrate.[5] In addition the constant superficial necrosis of the eschars was not noted in the lesions under current study. On the other hand the dermal reaction to the larva of Ascaris and to the ovum of *Schistosoma japonicum* was in basic respects similar to the bites of arthropods although the pseudo-epitheliomatous hyperplasia was lacking (Figs. 23 and 24).

Discussion

Differentiation from Lymphoblastomas

The basic histologic picture of the cutaneous reaction to a variety of arthropods is a dense dermal infiltration consisting principally of mature eosinophilic leukocytes and plasma cells admixed with histiocytes which are occasionally in mitosis or binucleated, resembling Sternberg-Reed cells. This histologic reaction has been confused with

Hodgkin's disease and mycosis fungoides, a possibility noted previously by others.[6] The occasional addition of circumscribed collections of lymphocytes, sometimes with large lymphoid follicles and actual germinal centers, may suggest giant follicular lymphoblastoma, Spiegler-Fendt sarcoid, or an ill defined atypical lymphoblastoma. The presence in histiocytes of mitotic figures, however rare, serves further to camouflage the true diagnosis inasmuch as mitotic figures in dermal infiltrates must be regarded as presumptive evidence of neoplasia until proved otherwise. The reaction to the bite of arthropods is a definite exception to this rule. Furthermore, contrary to the observations of others,[6] these studies indicate that the reactions to the bites of arthropods may persist as active dermal "eosinophilic granulomas" for at least 2 years, and perhaps longer. It is precisely this fact, hitherto obscured, which requires that these lesions be considered in an altogether new stratum of differential diagnosis characterized by diseases of relatively long duration, particularly neoplastic processes. It bears noting that while the existence of a single lesion may to some degree support the diagnosis of insect bite as against neoplasm, this type of evidence must be tempered by the fact that lymphoblastomatous involvement of skin may appear as an isolated lesion for a long time. Conversely, insect bites may be multiple.

Hodgkin's disease may cause the most difficulty in differential diagnosis. The main basis for the histologic differentiation of the insect bites from the neoplastic lesions and Spiegler-Fendt sarcoid (whatever its nature) is the abundance of mature eosinophilic leukocytes in association with plasma cells. Evidence of phagocytosis is additional presumptive, but not conclusive, evidence against the diagnosis of lymphoblastoma. However, there are other criteria which lend further aid in differential diagnosis. Frequently the reaction to the arthropod is accompanied by a degree of pseudo-epitheliomatous hyperplasia of the epidermis that may be mistaken for squamous cell carcinoma. Such a reaction in conjunction with the suspected dermal infiltrate is, *per se,* potent evidence in favor of the reaction to an arthropod. Moreover, the presence of epidermal inclusion cysts, especially with foreign body giant cells intermingled with eosinophilic leukocytes, even in the absence of identifiable parts of the arthropod, is significant evidence of the inflammatory basis for the lesion.

Differentiation from Other Eosinophilic "Granulomas"

Perhaps the source of greatest confusion lies in the recently publicized group of "eosinophilic granulomas of the skin."[3,4,7] There can be little question of the heterogeneity of this group in which there

appear to have been lumped lesions due to parasites of various sorts as well as to biting insects and acarines, lipoidal and reticulohistiocytoses including Letterer-Siwe's disease, periarteritis nodosa, and lymphoblastomas, particularly Hodgkin's disease. Surely, therefore, the tendency to consider the cutaneous "eosinophilic granuloma" an entity will serve only to add confusion. The differential histologic characteristics mentioned above, namely, eosinophilic plasmacellular infiltrate, epidermal inclusions, and pseudo-epitheliomatous hyperplasia, may aid the very practical purpose of segregating the reactions to the bites of arthropods from the other types of so-called eosinophilic granulomas. In the absence of pseudo-epitheliomatous hyperplasia, the reaction to the intracutaneous larvae of worms, often in the clinical form of "creeping eruption," may be indistinguishable from that of insect bites. The dermal eosinophilia in some cases of periarteritis nodosa may be fully as marked as in the lesions due to arthropods but the vascular change in the latter is relatively mild and does not present the fibrinoid degeneration that characterizes periarteritis nodosa, or, for that matter, erythema elevatum diutinum, another disease that has been included in the differential gamut. Various stains for fat that are clearly positive serve to rule out insect bites in favor of the lipoidal histiocytoses. However, the vacuolated cells of the former may be mistaken for lipoidal histiocytes in routine sections, although the granularity and fine vacuolization of the histiocytes is more evident in the histiocytoses, both lipoidal and nonlipoidal. The frequent presence of eosinophilic leukocytes in the histiocytoses calls for nice judgment in deciding that the histiocytes constitute the primary cellular response and, generally, are structurally different from those found in reactions to arthropods. Because of the identity of the names, the eosinophilic granuloma of skin has naturally been compared with the corresponding lesion of bone.[7] Here again, in the lesion of the bones, the histiocytes with the acidophilic, finely granular, often lipoid-filled cytoplasm appear to be the essential, primary cellular matrix rather than the eosinophilic leukocytes, however abundant they may be. This is not meant categorically to deny any possible relationship between eosinophilic granuloma of bone and other forms of cutaneous eosinophilic granulomas of skin, exclusive, of course, of those due to arthropods. In the previously reported mixed group of "eosinophilic granuloma,"[3] an eosinophilia of the peripheral blood commonly was present and was often at a high level. Data on the level of eosinophilic leukocytes in patients with arthropod bites included in the current study are incomplete, but in the 2 cases in which counts were made, no peripheral eosinophilia was present.

Pathogenesis

No constant or significant differences were noted in lesions varying in duration from 3 weeks to 2 years. Possibly a study of a larger series of cases in the early stages may modify this impression. In only one instance (mosquito bite) was a scar formed. The duration of this lesion was unknown. In any case, it is indisputably true that the prominence of eosinophilic leukocytes and plasma cells was as marked or even greater after 1 to 2 years as after 3 weeks. This constancy in the quality of the reactions notwithstanding their duration, which is at sharp variance with the observations of others,[2,6] is of considerable interest because it implies that the stimulating agent of the arthropod or its venom in some way manages to maintain its activity over many months. This activity is achieved in the absence of any recognizable remnants of the arthropods, although in several instances there was a history of incomplete removal of a tick. Moreover, the duration of vesiculation observed in more than one-third of the cases appeared to be in days or, at most, weeks rather than in months. The pathogenesis of this acute or subacute vesiculation, occasionally recurrent in a chronic lesion, remains to be explained on a basis more adequate than mere excoriation. Inasmuch as these vesicles are of eczematous types, action of the persistent allergen within the lesions seems a likely basis for their development. There was no correlation between the occurrence of vesiculation and the duration of the lesions.

In contrast to the reactions of the skin to ticks, mosquitoes, and chiggers is the histologic picture of the eschar or primary lesion of scrub typhus (tsutsugamushi disease). In the eschar, eosinophilic leukocytes are absent or rare. This disparity is of interest because of the close taxonomic relationship of the larval mite of scrub typhus (*Trombicula akamushi* and other species) to the chigger (larval mite of *Trombicula irritans*). Of course, in the eschar there is the added factor that rickettsiae are included in the lesion, but these organisms are said not to affect the histologic reaction qualitatively.[8]

One of the lesions, of 15 months' duration produced by a tick, occurred in a Negro. Histologically, there was no appreciable epidermal reaction but considerable dermal infiltration. The reaction did not differ basically from those in the white patients, which is of interest in view of the known resistance of the skin of the Negro to reactions to lice, mites, and other arthropods.

Pseudo-epitheliomatous Hyperplasia

Finally, there remains the interpretation of the principal epidermal reaction or pseudo-epitheliomatous hyperplasia. The term pseudo-

epitheliomatous hyperplasia is used commonly in the dermatologic literature, but appears aptly chosen notwithstanding the resistance to it by general pathologists. The term does not connote a "precancerous" state. It refers simply to hyperplastic epidermis which sends shoots of branching rete pegs deep into the dermis so as to *simulate* a squamous cell carcinoma. This simulation becomes especially marked when islets of epidermis appear invasive; actually they belong to the tentacular epidermis which has been cut obliquely. This reaction, which occurs often at the margin of chronic cutaneous ulcers, was observed frequently in the reactions to arthropods and usually was mistaken for carcinoma. It is stated in the literature [9,10] that the bites of insects have produced carcinomas. There is also a report of melanoma following a tick bite,[11] but this case appears to have had incomplete histologic study. However, the demonstration of a causal relationship in the bite of the arthropod to the development of the neoplasms, while of considerable fundamental interest and by no means disproved, still requires more direct evidence than is afforded by the available data.

Summary

A histologic study was made of the reactions to the "bites" of ticks, chiggers, mosquitoes, and unidentified arthropods.

The reaction, which consisted of a dense dermal infiltrate characterized by large numbers of eosinophilic leukocytes, plasma cells, and histiocytes, may be mistaken for Hodgkin's disease, mycosis fungoides, atypical lymphoblastoma, histiocytoses, and the heterogeneous group of "eosinophilic granulomas."

The lesion is often associated with a pseudo-epitheliomatous hyperplasia which may be confused with squamous cell carcinoma. The association with an eosinophilic dermal infiltrate and with epidermal inclusion cysts provides helpful differential clues.

With one exception, no basic difference was noted in the histologic reaction of the skin to the various arthropods studied. The striking exception is the almost complete absence of eosinophilic leukocytes in the eschar or primary lesion of scrub typhus caused by the larval mite (*Trombicula akamushi* and related species).

It is emphasized that the reaction to the "bites" of arthropods may persist for many months and that, in general, no appreciable difference is noted in the histologic reaction in lesions lasting from 3 weeks to 2 years. It is therefore concluded that the stimulating agent of the arthropod somehow must persist actively in the focus of these lesions for a remarkably long time.

A single cutaneous lesion with the histologic picture suggestive of

Hodgkin's disease or other lymphoblastoma should always be suspected as having been caused by the bite of an arthropod until conclusively proved otherwise. The history of an insect bite may not be volunteered after a lapse of many months.

The cutaneous reactions of individuals, even of the same race, to different arthropods varies not only in the acute stage but also in the persistence, degree, and quality of the histologic reaction.

It remains to be determined precisely what agent in the venom or embedded parts of the arthropod, or both, is responsible for the cutaneous reaction.

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[*Illustrations follow*]

DESCRIPTION OF PLATES

All photomicrographs were made from sections stained with hematoxylin and eosin.

PLATE 71

FIG. 1. Pseudo-epitheliomatous hyperplasia 1½ years after an insect bite. (Army Institute of Pathology negative no. 95762.)

FIG. 2. Pseudo-epitheliomatous hyperplasia 4 months after a tick bite. (A.I.P. neg. 95767.)

FIG. 3. Pseudo-epitheliomatous hyperplasia 8 months after a chigger bite. (A.I.P. neg. 99865.)

FIG. 4. Higher magnification of the tips of the pegs of epidermis seen in Figure 3, showing numerous mitotic figures. (A.I.P. neg. 100469.)

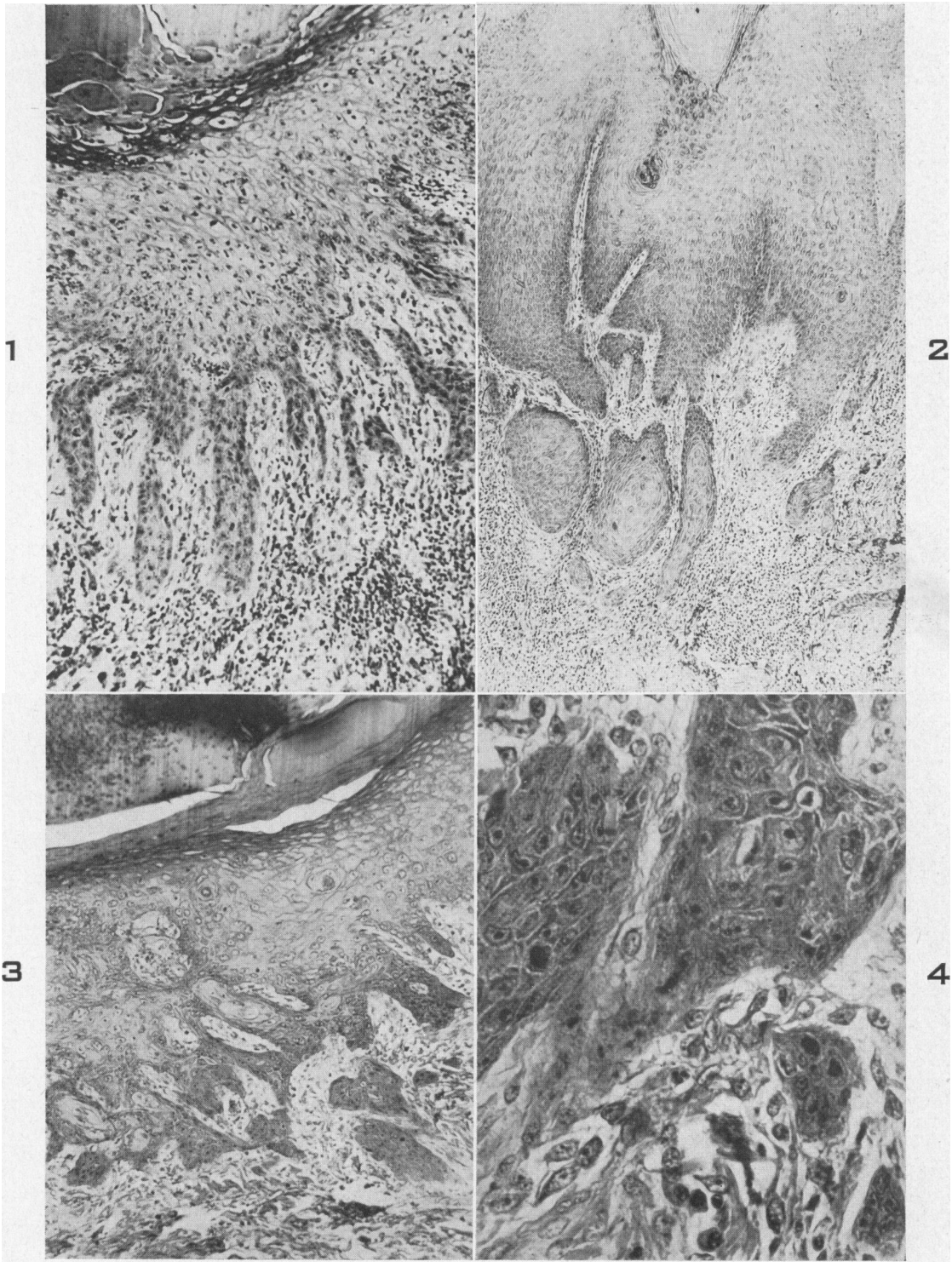
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PLATE 72

FIG. 5. Vesiculation, acanthosis, and dermal reaction 4 months after an insect bite. (A.I.P. neg. 99868.)

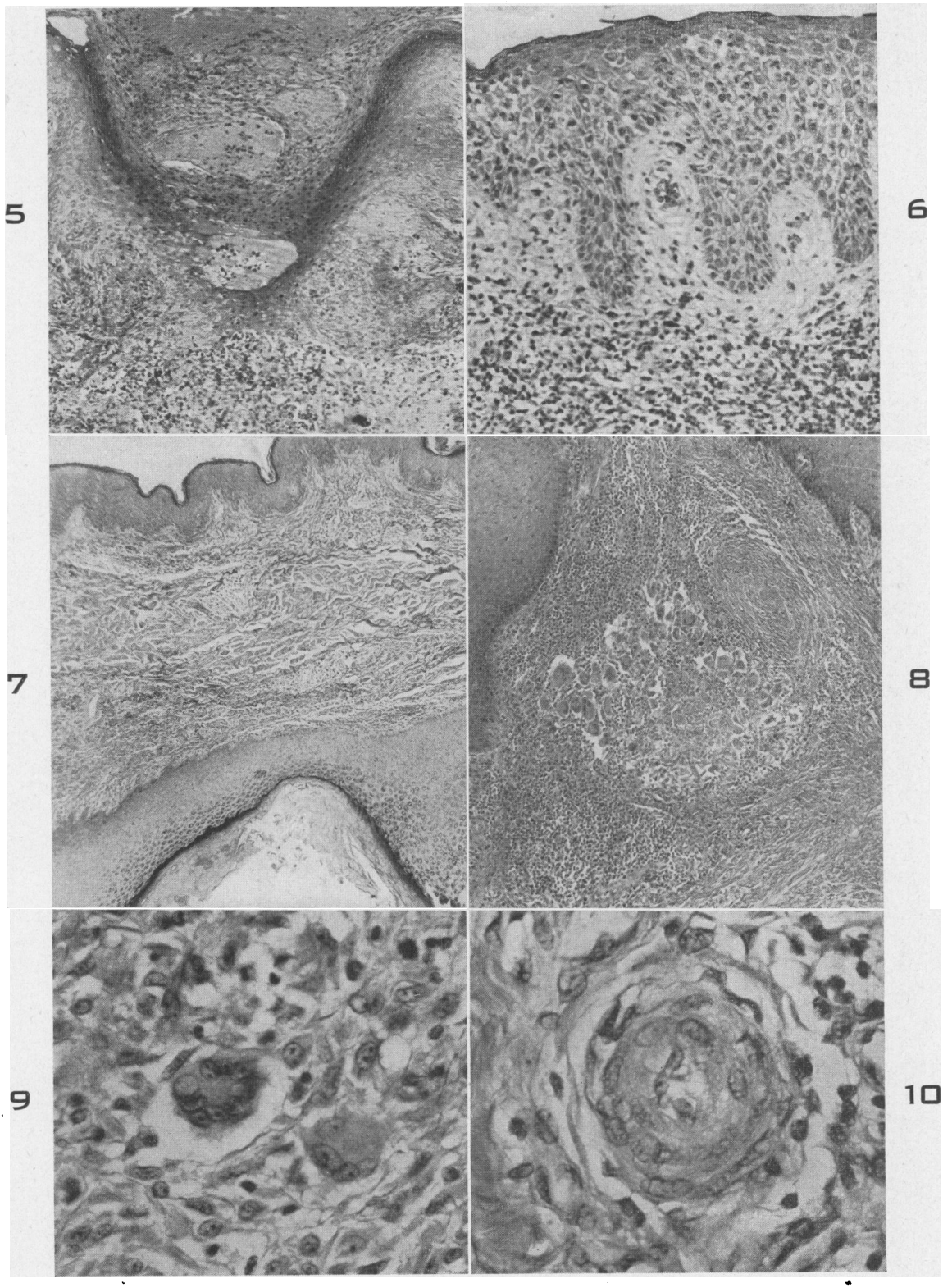
FIG. 6. Spongiosis and epidermal transmigration of eosinophilic and neutrophilic leukocytes 4 months after a tick bite. The dermal infiltrate is conspicuous. (A.I.P. neg. 100435.)

FIG. 7. Epidermal inclusion cyst 7 weeks following a mosquito bite. (A.I.P. neg. 99869.)

FIG. 8. Giant cell proliferation together with plasma cells, eosinophilic and neutrophilic leukocytes comprise the reaction to the epidermal cyst seen in Figure 7. No parts of the insect were detectable. (A.I.P. neg. 100442.)

FIG. 9. Giant cells admixed predominantly with eosinophilic leukocytes and histiocytes. This reaction was observed 7 weeks following a mosquito bite. (A.I.P. neg. 100471.)

FIG. 10. Arteriolitis with swelling of cells and fibers of the entire wall, 4 months after an insect bite. (A.I.P. neg. 100467.)



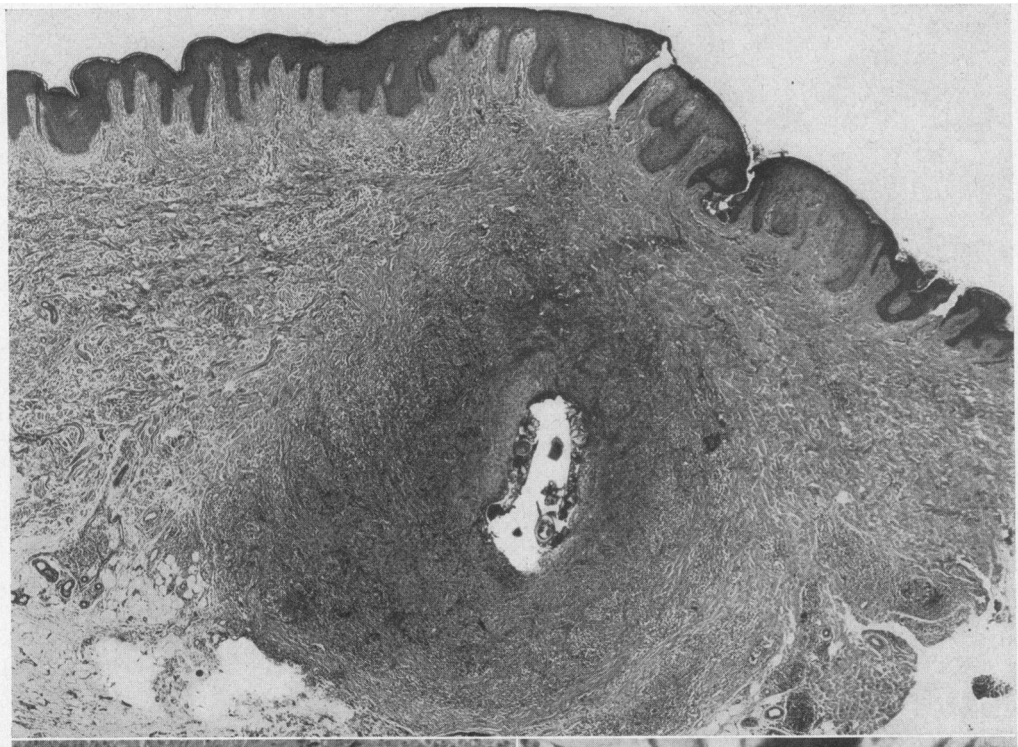
Allen Persistent "Insect Bites"

PLATE 73

FIG. 11. Reaction to a tick bite after 1½ years, showing the dense dermal infiltrate ("eosinophilic granuloma") surrounding an epidermal inclusion cyst. The contents of the cyst include portions of the tick. (A.I.P. neg. 77486.)

FIG. 12. Higher magnification of the epidermal inclusion cyst and the enclosed parts of the tick shown in Figure 11. (A.I.P. neg. 90949.)

FIG. 13. Higher magnification of Figure 12, showing parts of the tick and epithelium of the inclusion cyst, several cells of which are in mitosis. (A.I.P. neg. 100470.)



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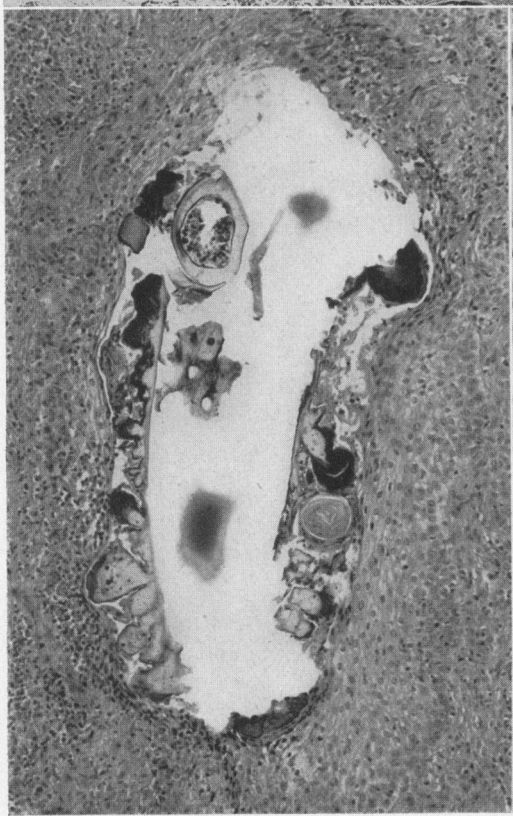

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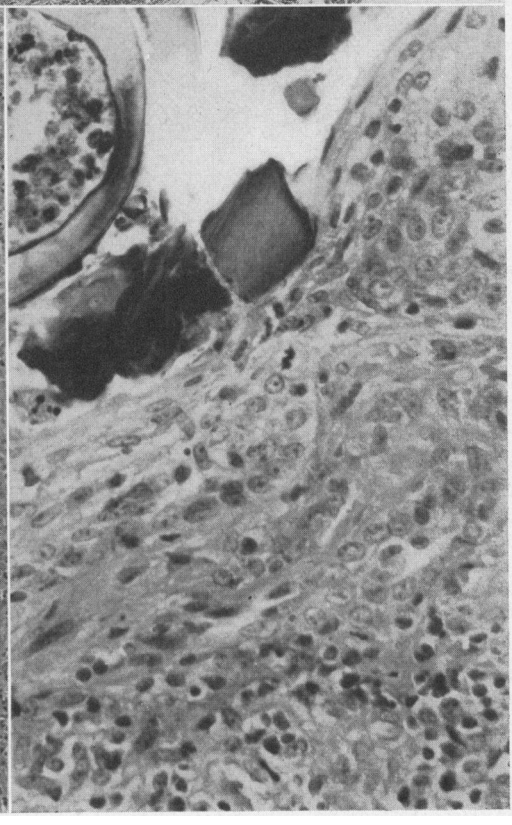

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PLATE 74

FIG. 14. Dermal reaction 4 weeks after a tick bite, showing subepidermal edema and a dermal infiltrate in which the predominant cell is an eosinophilic leukocyte. (A.I.P. neg. 100613.)

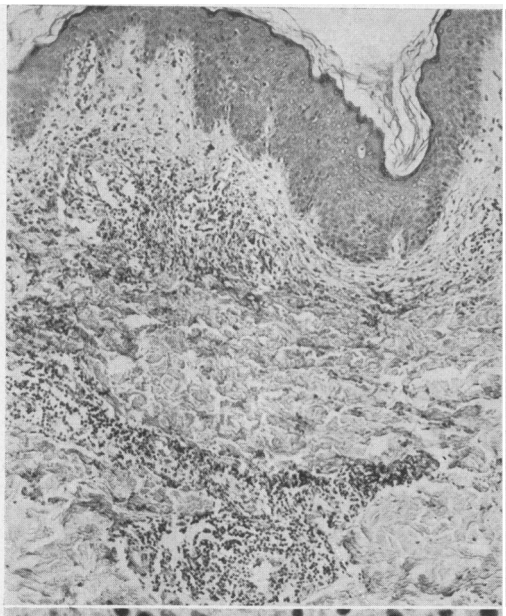
FIG. 15. Dermal reaction ("eosinophilic granuloma") to a chigger bite after 10 months. (A.I.P. neg. 95765.)

FIG. 16. Dermal polymorphous reaction ("eosinophilic granuloma") to a tick bite after 18 months. (A.I.P. neg. 78276.)

FIG. 17. Reaction to a chigger bite after 8 months. The plasmacellular response simulates a syphilitic infiltrate. (A.I.P. neg. 80169.)

FIG. 18. Large binucleated cell, and numerous plasma cells and eosinophilic leukocytes, in the inflammatory response 8 months after a chigger bite. (A.I.P. neg. 100439.)

FIG. 19. A mitotic figure and many histiocytes with hydropic cytoplasmic changes in reaction 15 months after a tick bite. (A.I.P. neg. 100466.)



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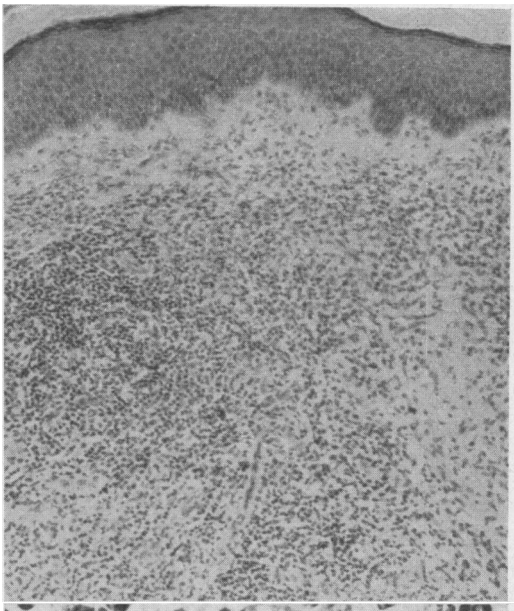

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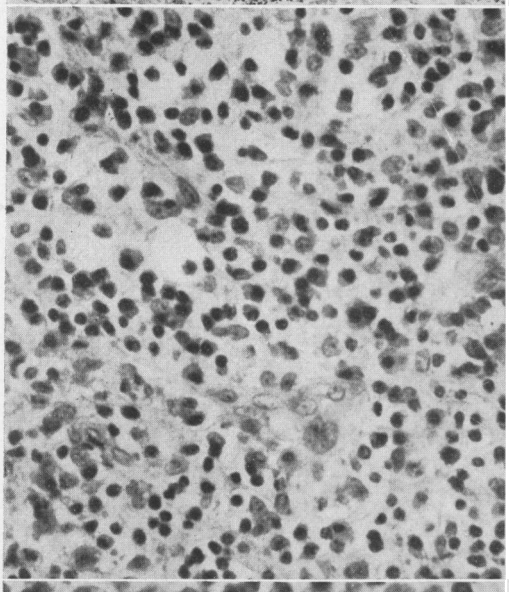

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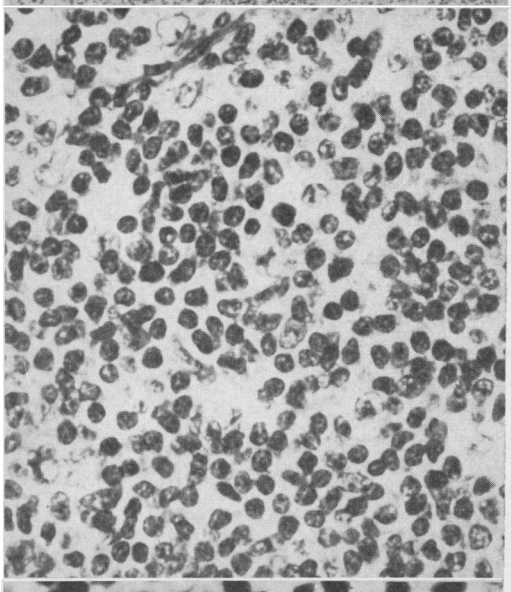

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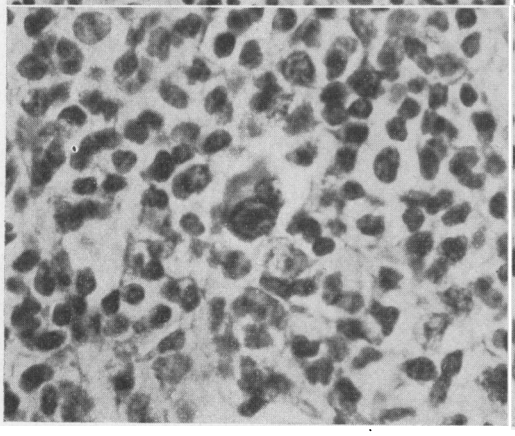

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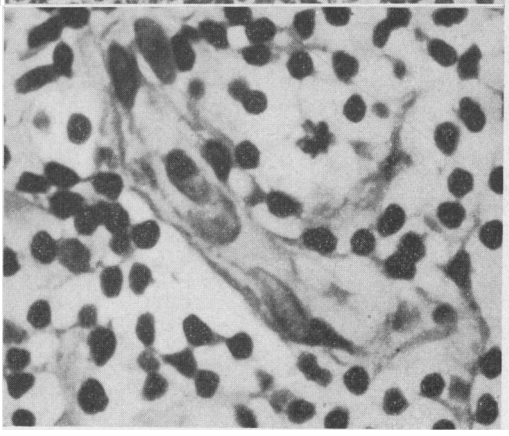

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PLATE 75

FIG. 20. Reaction 10 months after a tick bite, showing dermal infiltrate with large lymphoid follicles that have led to erroneous diagnoses, particularly of lymphoblastoma. (A.I.P. neg. 90953.)

FIG. 21. Prominent lymph follicles forming part of a dermal reaction 10 months after a tick bite. (A.I.P. neg. 100440.)

FIG. 22. From the same case as Figure 19, showing a lymph follicle in relation to sweat glands. (A.I.P. neg. 90954.)

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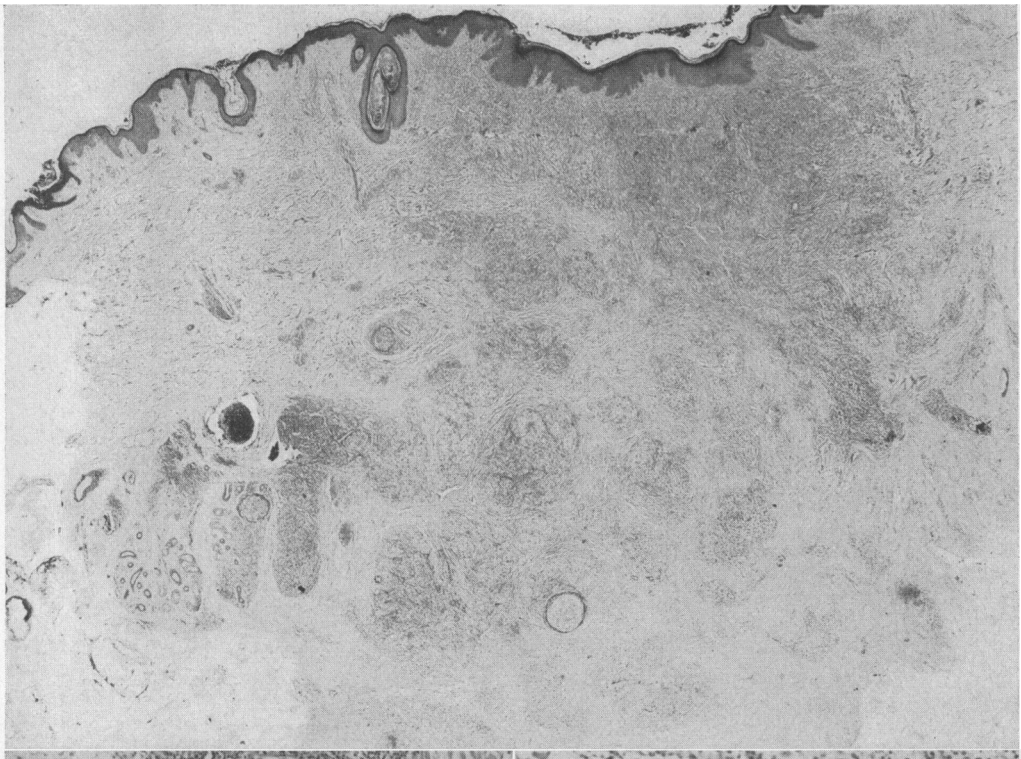

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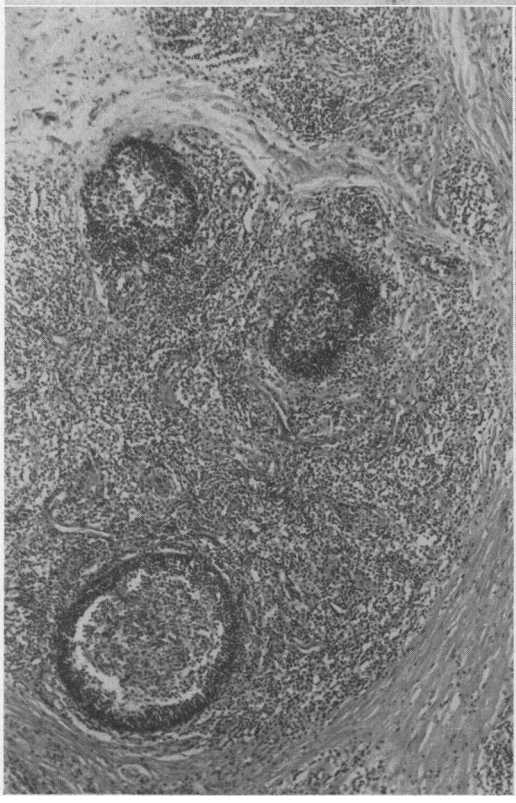

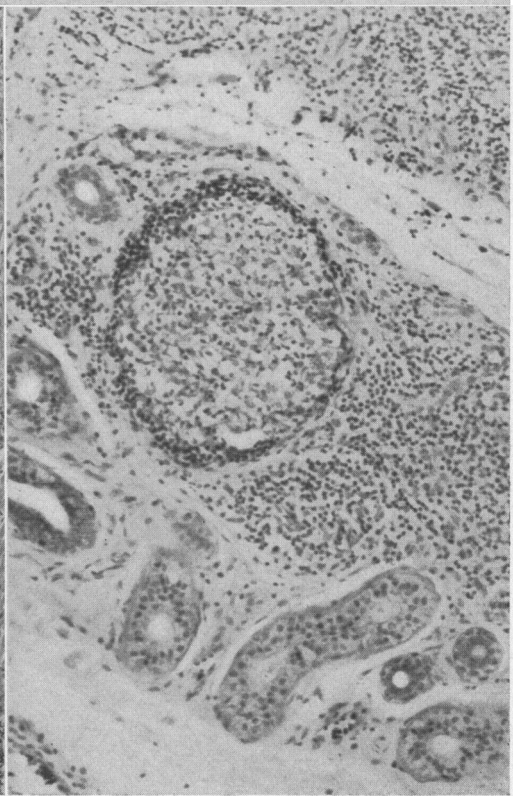

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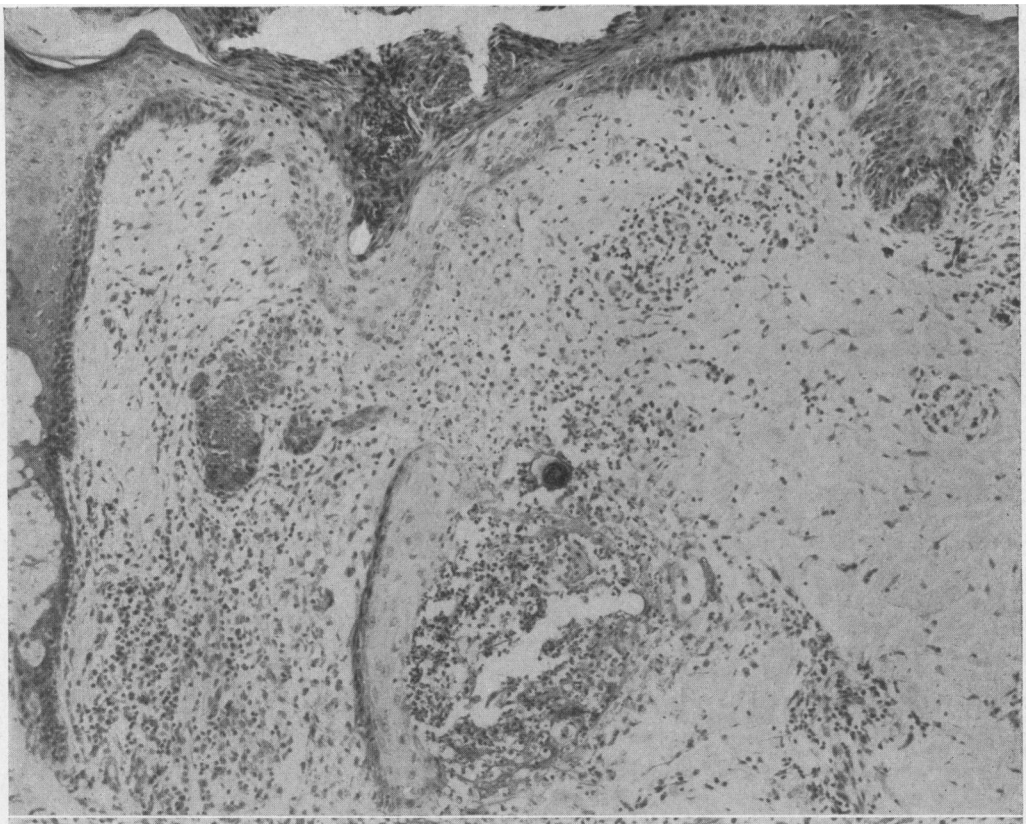
PLATE 76

FIG. 23. Dermal "eosinophilic granuloma," a reaction to the ovum of *Schistosoma japonicum,* showing superficial scaling, the ovum, adjacent epidermal inclusion, and inflammatory reaction including numerous eosinophilic leukocytes. (A.I.P. neg. 89563.)

FIG. 24. Dermal "eosinophilic granuloma," a reaction to the larva of Ascaris, showing numerous eosinophilic leukocytes and foreign body giant cells. (A.I.P. neg. 78694). (Courtesy of J. E. Ash and S. Spitz. Pathology of Tropical Diseases. W. B. Saunders Co., Philadelphia & London, 1945, 350 pp.)



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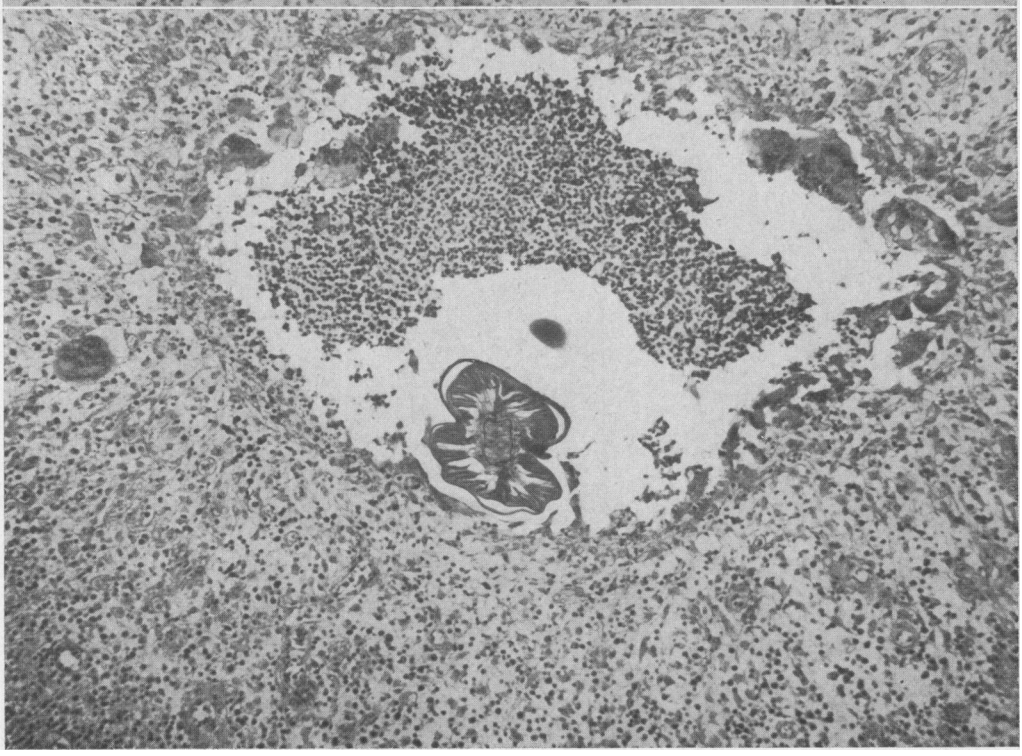

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