INDIAN JOURNAL OF DERMATOLOGY, VENEREOLOGY AND LEPROLOGY VOL. 42, NO. 5, 1976

HUMAN SCABIES - A CONTINUING PROBLEM *

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Summary

Scabies continues to plague mankind with cyclic fluctuations. Based on the findings of an epidemic among the Bangladesh refugees in 1971 in West Bengal and clinical experience in hospital practice, the significance of socio-economic, hygienic, nutritional and immunological factors permitting resurgence of scabies in wartime or otherwise is dicussed. Overcrowding resulting in intimate contact and improper and inadequate therapeutic measures are held responsible for the spread of scabies. While hypersensitivity and immune mechanisms appear to be operative in scabietic infestation, to establish the presence and the significance of immunity to the parasite, further investigation is needed.

Known for centuries, scabies continues to plague mankind even today. Though infestation with sarcoptes scabeii var hominis classically occurs through human contacts, similar disease at times may be acquired from domestic animals viz, dogs, cast, horses harbouring species specific acarus. The parasite does not honour any social or geographic boundaries and in both, endemic and non-endemic areas it appears to resurge in periodic epidemics. Many tropical and subtropical regions are either endemic for human scabies or are affected by epidemics periodically. The epidemiological studies[1],[2] suggest that epidemic outbursts occur at intervals of about 15 years. This cyclical resurgence of scabies is ill understood and appears to be related to various environmental, parasite and host factors.

During the 1971 Bangladesh war when millions of refugees were sheltered in West Bengal for medico-social help, a survey was conducted by the author of a sample of their population. This revealed an epidemic of scabies with prevalence rate of 68 percent. Presently the disease appears to be prevalent in and around Delhi.

Based on these experiences, an attempt is being made to delineate important factors that might influence the reservoir, spread and the resurgence of the disease in a particular area. The biological behaviour of the parasite and clinico-pathological features of scabies are also briefly reviewed.

Life-Cycle of Sarcoptes Scabiei

Acarine mite sarcoptes scabeii feeds, dwells and multiplies on the human skin. The adult female is approximately three times the size of the male and measures less than 0.4mm. in length. In general, the mite resembles a miniature tortoise and has 4 pairs of legs; the anterior two pairs carry the suckers while the posterior two pairs are provided with long bristles.

* Based on work presented at the workshop on Communicable Dermatoses during the International Symposium and Fourth Annual Conference of IADV&L at New Delhi in Feb. 1976.

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Received for publication on 25—3—1976.

Following fertilization, the female acarus burrows into the skin nearly lifting her body upright supported by the bristles and gradually cutting its way beneath the horny layer of the epidermis in a slightly downward horizontal direction. The rate of burrowing is extremely variable (1 mm to 5 mm per day) but the mite has been observed to reach beneath the stratum corneum in as short as 20 minutes[3]. Two days after fertilization the gravid female starts laying eggs along her course in the burrow at the rate of 1-3 eggs per day. On an average a female acarus deposits a total of 10-25 eggs following which concludes her 4-5 week long life by dying at the end of her burrow. It is interesting to note that once a female burrows herself beneath the stratum corneum the possibility of her leaving the burrow to move out seems extremely unlikely because of the presence of posteriorly pointing bristles. It is only through normal process of desquamation that dead mites or eggs approach the skin surface. Fecal pellets can also be found along the burrow.

After 3-4 days of incubation in the burrow, the egg hatches into a hexapod larva which starts moving out. On reaching the skin surface, larva starts making new burrow and then over 10-14 days time moults through various stages of octopod nymph into an adult mite (Fig. 1). Merely less than 10 percent of the total eggs laid become adult mites[4].

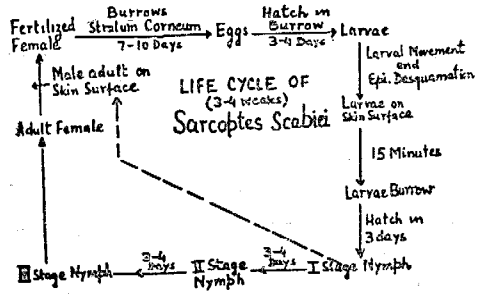

FIG. 1

The male goes through a similar cycle except that from the I-stage nymph it emerges directly as an adult male. The male spends most of his adult life on the skin surface, fertilizes female and dies. Rarely the male may enter shallow burrows before dying.

Clinical Features

In hospital practice the infestation predominantly affects the young and the elderly but under circumstances of epidemic outbursts it appears to affect all age groups (Table 1). The males appear to be affected twice as commonly as females. Most frequent manifestation is the intensely itchy rash that keeps the patient awake at night. More often than not, other members of the family

TABLE 1

Age–Sex incidence of scabies in a sample of Bangladesh refugees (1971)

Age (Years)	Number Examined	Number affected	Males	Females
— 1	28	21 (75)*	9 (43)	12 (57)
1— 5	46	32 (70)	12 (38)	20 (62)
6—10	84	67 (80)	37 (55)	30 (45)
11—20	140	100 (71)	68 (68)	32 (32)
21—30	116	86 (74)	70 (81)	16 (19)
31—40	94	51 (54)	30 (60)	21 (40)
41—50	62	31 (50)	11 (35)	20 (65)
+50	30	18 (60)	13 (72)	5 (28)
	600	406 (68)	250 (62)	156 (38)

* Figures in parentheses represent percentages

have similar complaints. In Bangladesh refugees survey it was observed that in 84 percent of cases the other family members or inhabitants of the same tent were involved.

The dermatologist informed by these hints in the patient's history is then guided by the morphology and the distribution of skin lesions to corroborate the diagnosis of scabies. Morphologically the only diagnostic lesion of scabies is the 'burrow'. A burrow appears as a small skin coloured thread like wavy elevation most commonly encountered in the fingerwebs or on the volar aspects of the wrists. Often hard to find without a magnifying lens and experience, the burrows were seen in 16 percent of 406 cases found to have scabies during the Bangladesh refugee survey (Table 2). Each patient displayed more than one type of lesions. The papules vesicles, pustules crusted excoriations or the urticarial lesions are not diagnostic of scabies but their particular distribution is helpful in diagnosis. Though the sites of predilection are well known, from the data obtained it appears that the sites involved are related to the patient's age. Certain specific sites are affected in different age groups (Table 3). It may be re-emphasized here that though finger webs, genitals, belt area and areolae in females remain the commonest sites of involvement, no area is immune to affection. The body areas above the neck are often spared except in infants where the clinical picture mimics infantile eczema.

A distinct morphological type of scabies manifests with unusually hyperkeratotic lesions of the palms, soles, face and the scalp teeming with the parasites. This is often referred to as 'Norwegian scabies' and appears to manifest in mentally defective or immunologically compromised individuals.

Laboratory Diagnosis

Though the diagnosis of scabies can be confirmed by the demonstration of the parasite from the burrows, the latter are only infrequently seen and the technique often proves to be time consuming especially in epidemics and large surveys. In office practice however,

TABLE 2

Type of lesions seen in 406 cases of scabies.

Type of lesion	Number	Percent
Papules and/or papulovesicles	386	95
Pustules and/or crusted lesions	160	39
Burrows	65	16
Wheals	41	7

TABLE 3

Age of patient and common sites of scabietic lesions.

Age group (Years)	Sites of lesions in order of frequency (406 cases)
—10	Finger and toe webs, Genitals, Buttocks, Umbelicus, Abdomen, Face.
11—20	Finger webs, Genitals, Belt area, thighs, Buttocks, Upper back, Axillary folds.
21—40	Genitals, Belt area, Upper back, Buttocks, Thighs, Webs of hands, Nipples and Areolae in females, Axillary folds.
+40	Genitals, Belt area, Buttocks, Thighs, Webs of hands, Axillary folds.

it is indeed the only fool-proof method of confirming the diagnosis. If a burrow is seen the parasite can often be found by gently digging the same with a needle while observing with a magnifying lens and transferring the same on a glass slide carrying a drop of 10 percent KOH or normal saline. A coverslip is placed and examined under light microscope. The findings of ova or scybala are also equally helpful and easily recognizable. In difficult situations where picking the burrows does not produce results, alternative methods of superficial shaving the burrow with a scalpel blade carrying a drop of immersion oil which tends to prevent fragmentation of the specimen and dissolution of scybala proves useful[5].

Histopathology

Histopathologic studies of scabietic lesions are of recent interest. It is mentioned that when an acute eczematous type of epidermal change is observed together with an erythema multiforme type of dermal reaction, a search for the parasite is justified[6]. Serial sections prove to be more fruitful for finding the parasite in biopsies. Vesiculation may be observed in upper stratum malpighii in suitable sections and this then represents the distal end of the burrow. Rarely, histopathology of scabietic lesions may simulate reticulosis[7].

Immune Mechanisms in Scabies

The cyclic fluctuations in scabies are alleged to be related to socio-economic factors, sexual promiscuity, demographic factors, increased travel and misdiagnosis[8]. Recently, the importance of immunologic factors have been realized but their exact nature is ill understood. The evidence of positive intradermal tests with extracts of sarcoptes scabiei about 6 months following infestation, second infestation producing an accelerated response, prolific infestation in immunologically compromised patients and specific anergy to intradermal tests in some patients speaks for the immune mechanisms in scabies[9],[10]. Moreover the histological findings of inflammatory lesions indicate a sensitizing phenomenon.

Treatment of Scabies

The various scabicidal agents available today include sulphur ointment (5–10 percent), Benzyl Benzoate emulsion or ointments (Ascabiol, scabalcid, scabindon, Uniscab), Mesulphen (Mitigal), Gamma benzene hexachloride (Lindane, Lorexane, 1 or 2 percent), Monosulfiran (Tetmosol) and crotamiton (Crotorax). While most of them are effective, a recent trial showed Gamma benzene hexachloride to be superior to others. This particular antiscabietic agent is widely used in the West under the brand name 'Kwell' with excellent results. It is alleged that usually one application is sufficient as against 3 or more with benzyl benzoate. However the trial[11] done in India showed 20 percent treatment failures even when the drug (2 percent) was applied daily for six consecutive days. The treatment with Lindane however, appears to be most economical to the patient. The use of DDT has been mostly abandoned because of its toxic effects.

Whichever antiscabietic preparation is used, it must be applied on the whole body below the neck after a thorough scrub bath (infants usually have lesions on the face, neck or scalp and medicine is also applied on these areas). Frequently only the visible lesions are treated which accounts for failure of treatment. The patient is asked to make two more applications of the medicine (Benzyl Benzoate-25 per cent mostly used) similarly after every 12 hours. The patient then takes bath and disposes his clothings for laundering and wears another set of clothes. Though the role of fomites in the spread of

scabies appears dubious[12] it is wise to ask the patient to discard the clothes for laundering at least to get rid of the odour of medicine. Secondary infection if present should be treated with suitable antibiotics immediately especially in children to obviate the possibility of poststreptococcal glomerulonephritis. Norwegian scabies needs similar but more vigorous treatment. The keratotic crusts may be removed before medicine is applied. The use of methotrexate[13] does not appear justified.

It is extremely important that every possible contact be treated at the same time. Some advocate treatment for the whole family and even the visitors to stop further spread. Frequently, the patient is given the treatment but the contacts are ignored. This results in treatment failure because of continuous reinfestation from the existing reservoir in the patient's environment. In wartime and in other situations of overcrowding the importance of simultaneous treatment of all affected is a must. Indeed, for proper control and eradication immediate treatment of affected persons and possible contacts and subsequent routine check up to get rid of residual reservoir is essential.

Lastly, the health education of medical and paramedical staff in first recognizing the disease in its many guises and then passing accurate instructions for treatment is greatly desired.

Comment

During natural calamities like wars, floods, famine etc., scabies is known to appear in epidemics. Likewise a survey conducted at the time of Bangladesh War in 1971 revealed an epidemic of scabies in a large population sheltered in West Bengal. Recent years have witnessed an ever increasing incidence of scabies in different parts of the country. This is partly reflected in an increasingly larger number of scabies patients attending the clinic every year (Table 4). The factors responsible for resurgence of scabies in war time and otherwise in general population are varied and need evaluation.

It is known that the disease spreads through personal contact and Mellanby[3] states that even under ideal conditions sarcoptes scabiei cannot survive more than a few days without a human host. Therefore, for spread of the disease one needs in addition to reservoir of infestation, a host and environments conducive for the transmission of parasite, the mite from person to person. In the presence of reservoir, the disease would spread far more easily in conditions of overcrowding that happens in the war time. It is clear from the Bangladesh refugee survey that all persons irrespective of their age, sex, caste and creed were affected. Overcrowding because of limited space being available appeared to be the most significant factor

TABLE 4

Annual outpatient data suggesting an increasing incidence of scabies.

Year	Dermatology outpatient annual attendance	Patients with scabies	
		Number	Percent
1970	10112	424	4.20
1971	10226	430	4.20
1972	9926	398	4.00
1973	10618	480	4.52
1974	10328	816	7.91
1975	12000	1700	14.17

responsible for the epidemic. Substandard hygiene and malnutrition were concomitant findings and one wonders if in the absence of intimate personal contact from overcrowding the disease would have taken an epidemic form.

In hospital practice on the other hand one finds a higher incidence in younger age groups[3]. This seems to be true also of some field surveys[12]. Again families living closely in a limited space are more susceptible to infestation. Often in a family independent of its socio-economic status, a child acquires the disease from a domestic help and in turn passes it on to the mother or some other family member. In such instances, the family members are usually in perfect health and demonstrate good personal hygiene. The infestation appears to spread through intimate contact; socio-economic, hygienic and nutritional factors being only secondary. Poverty, possibly influences the type of living facility thereby creating better chances for intimate proximity in a limited space. Based on large field surveys Nair et al[12] have also expressed similar views. Beside overcrowding resulting in intimate contact, improper and inadequate treatment and failure to treat all possible contacts are the most important factors responsible for spread of scabies.

The question of immunological susceptibility to scabies is rather fragmentarily understood. Primary infestation is reported to sensitize the individual and intradermal tests with extracts of Sarcoptes become positive after a few months. The inflammatory lesions that appear sometime after primary infestation are believed to result from hypersensitivity phenomenon, the view to an extent supported by recent histopathological findings[6,8]. An accelerated response on subsequent exposure, massive infestation of immuno-suppressed patients and alleged specific anergy in some patients indicate that immune mechanisms are involved[9,10]. However, the common recurrences seen clinically, affection of persons with varied social and nutritional status and infestation lingering for months or years without proper treatment cast doubts on acquired immunity to the parasite. The significance of low levels of serum IgA in predisposing to scabies is obscure[14]. Possibly, an understanding of the biochemical nature of the parasite and the chemical mediators that permeate down to the living epidermis to produce inflammatory lesions may provide a better insight into immune mechanisms involved in scabies.

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