SCABIES - A MIGHTY PERSISTENT PESTERING PUZZLE DESPITE PESTICIDES

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With the availability of modern pest control measures, effective scabies therapy should be within the reach of every household or a community and endemic or epidemic scabies should be in the realms of history. That we have lagged behind such a goal, portrays a lack of our ability in application of science. In this paper I shall review the might of scabies and its mite, its persistent pesterings recorded in history, the natural course of untreated scabies, its seven clinical faces creating the puzzles of diagnosis and the pesticides used for its control.

Persistent pesterings of scabies are shrouded in the history of medicine. In the treatise of Indian Medicine known as "sushruta samhita" dated 500 B. C. we find descriptions of an itchy eruption distributed on buttocks, hands and feet which was named "Pama" by Charaka around 800 B. C.[1]. We find in Greek medicine of Hippocratic era dating 460–370 B. C. the term Psora meaning "the itch", denoting a number of itchy skin problems. In 447 A. D., Cassius Felix named it scabies in "De Medicine" and separated it from other skin diseases. The mite was first alluded to in 970 A. D. by an Arab physician Al Tabari as an " animalcule that can be recovered on the point of a needle" but he did not refer to it as the cause of the disease[2]. Another 7 centuries passed by till Thomas Moffet in 1643 of England and Bonomo and Cestoni of Italy in 1687, observed the size, shape and the burrowing habits of the mites[2]. The Italians accurately studied and measured it, noted the ova, conjectured the sexual reproduction and spread of the disease and described its treatment. Accurate entomologic descriptions were given after another century by a Swedish naturalist Dee Geer in 1778 after whom the mite was known as Sarcoptes Scabei der Geer[2]. However, Medical men — a traditionally opinionated lot — were against the causation of scabies by the mite until 1813[3].

It was Rennucii in 1834 who again dug out the acarus from its burrow, and Beaude demonstrated the live parasites to the French Academy[2]. Bazin demonstrated the spread of acari over the whole body and established its effective treatment in 1878. His contemporary, Hebra from Vienna, pestered by more than 8000 cases of scabies per year accurately described the life cycle of the mite and published the first book on this disease in 1844. On a visit to Norway, he saw the first

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unusual case of extensive chronic scabies with immense number of mites in a leprosy patient described by Boeck and Danielssen and named it "Norwegian Scabies — Boecki"[4]. It was thought that the mites were different in these scabies, but now we know that this is not the case. A century later Mellanby in 1944[6] during the Second World War wrote a fascinating book on scabies. With his associates, he studied the parasitology and transmission of scabies based on a mighty endeavour of counting 9978 ovigerous mites in 886 patients[7,8]. He observed the life history of experimental scabies on 56 volunteers noting variations of mite populations for 265 days[9], and also examined the histologic responses. Further resurgences of scabies from time to time in different populations during peace and war catalogued by Orkin[10] demonstrates the might of this mite.

The Life History of the Mite

The life–cycle of Sarcoptes is accurately known since Hebra's unsurpassed studies, but some disputes remain regarding the infective potential of its different stages[11]. Recently Kutzer[12] has restudied the life-cycle of animal scabies and shown that eggs take 3 to 4 days to hatch, followed by a larval and two nymphal stages – proto – and tritonymphs.

The male and female tritonymphs can be identified. The adult male develops after about 14 days and the female after 21 days. After fertilization the female burrows down in the stratum granulosum but it does not penetrate the rete–malphighii. According to Johnson's theoretical estimate[9] approximately three quarters of a million mites can develop in 7 weeks but in practice considerably less than 1/2000th of this is noted showing a large casualty of the immature forms.

Diagnosis

The parasite is easy to demonstrate by 10X magnification in KOH, normal saline or in mineral oil. Besides the live acarus or its larvae, and ova faecal pellets of brown colour with a red fringe are also diagnostic[13,14].

Transmission

It is widely believed on Mellanby's evidence, that the transmission is mostly by person to person contact. The role of fomites was not considered important enough to advise their disinfestation. However, this question requires reappraisal particularly under endemic situations, which Mellanby had not faced.

"Scabies" occurs in almost all species of animals; (including zebra & lion!) In man it rarely develops from contact with infected animals although dogs, cats and goats have been incriminated[15,16,17,18]. The disease results from hypersensitivity reactions to the acarine bites and not due to direct colonization of the human skin.

Life history of Untreated Scabies

I shall summarize this important information from Mellanby's experiments on human volunteers[9]. (Fig 1). The course of the first infection produced in 56 votunteers, was sharply different from that of a re-infection. Scabies could be produced even by a single fertilized acarus, and no matter where the parasite was introduced, it caused typical clinical symptoms on the traditional sites. The first four weeks, were asymptomatic, although the mite population increased and could be visualized with a lens. From the fourth to the sixth week itching increasd and became unbearable by about 100 days. The build up of the mite population was slower than expected and adult females of the

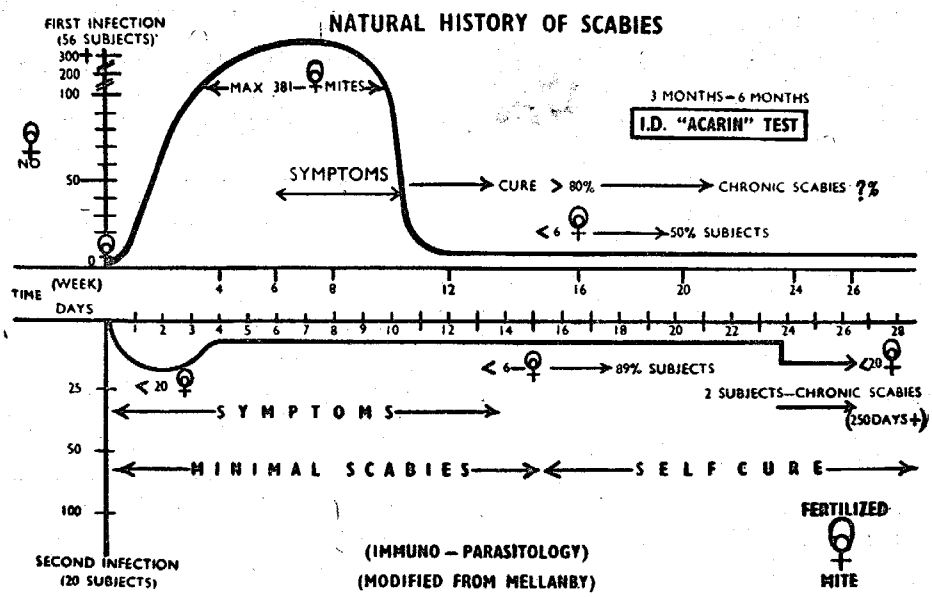

Fig. 1 Natural History of first and second experimental infections of scabies in volunteers. The time scale for primary infections is in weeks while that for the reinfection is in days. Experimental infections were achieved by transfer of single fertilized female acarus.

second generation were not observed in most hosts until the 28th day. This suggests a high mortality of the larvae of the first generation during the developing phase of scabies. The number of "ovigerous" mites, which were counted as an index of the invasive mite population, varied from 50 to 381 between 80 to 115 days after which there was a sharp decline. In fully developed scabies the average count was 8–12 ovigerous mites only. In many subjects with severe reactions, the disease was thrown off after this. Chronic infection involving very low parasite rates may continue indefinitely in some untreated individuals. The first injections were terminated on the 150th day.

Re-infections were studied in 22 volunteers who had suffered from scabies. Some subjects could not be reinfected suggesting a developed immunity. Lesions occurred within twenty-four hours at the site of introduction of the parasite. The mite population remained low and fluctuating after which day disappeared spontaneously in a couple of days, either by scratching or reactions.

However, vacant burrows which maintain itching, were seen for a long time in protected sites such as the genitals and axillae. Only in two subjects successful chronic infections developed. In spontaneously reinfected subjects the average parasite rate was only 3.2, as compared with 11.3 at the first time. The parasite rate was less than 6 in 60% of the first, and 89% of the second infections. Mellanby believed that this was too low for transmission of the disease to new hosts, which occurs readily with parasite rates of 20–50 and above. Hence symptomless individuals in the early asymptomatic stage of the first infections with large numbers of parasites are probably the main transmitters. They can be termed as the "carriers" of the disease.

Scabies is to some extent a "self-limiting disease": this was attributed to three factors, viz, scratching, immunological and patholigical changes in the hosts and possibly, by development of secondary bacterial infections[15].

Clinical Types of Scabies

Very few clinicians are aware of the seven clinical faces of scabies which I have compiled from the literature (Table I) and tried to correlate it with parasitologic and immunologic features.

(i) Subjects with primary infections are those with minimum symptoms of more than 2 months' duration particularly if parasites are easily demonstrable because severe symptoms begin after 80–100 days. The duration of the disease probably varies in different races. In a survey of 50 families of scabetic patients at K. E. M. Hospital, 90% of subjects had the disease for only about one month's duration and recurrences after treatment occurred in more than 37% of subjects[19]*.

(ii) Subjects with scabies of less than 2 months, but with severe symptoms and low mite population are probably reinfections. Partial or self-treated chronic cases also belong to this category.

(iii) Scabies in babies and children have a florid picture with extensive pyoderma, which we commonly see in the poor patients in the tropics. The duration is usually less than 2 months; and the mite population is low because of severe inflammatory reactions accompanying both scabies and pyoderma. Both are probably primary infections, and they also have a high recurrence rate[19].

(iv) Scabies in the healthy adults may be difficult to diagnose[20] and some of its current problems are elucidated by Orkin[21]. Persistent chronic nodules, or papule-vesicles or groups of vesicles are seen in subjects who have developed varieties of reactions due to hypersensitivity to the products of an

TABLE 1
7—Clinical Types of Scabies

Symptomatology	Itch	Duration	Number of Mites	Immunity Status
Asymptomatic — Mild / Severe	(±)	3 — 4 Months	High > 20 / Low < 10	Primary Infections
Papules+Vesicles ++	(+++)	< 2 Months	Low < 10	Reinfections
Pediatric Scabies + Pyoderma Vesicles +++	(+++)	1 — 3 Months	Low	Both Primary Infections
Nodular Persistent Nodules Vesicles ±	(++)	Chronic > 6 — 8 Months	Rare	Sensitization
Geriatric Psoriasiform: Dyskeratosis (Norwegian Scabies)	(±)	Chronic 1 Year +	Very High > 1000 per Cm_2	Negative Anergy
Latrogenic Many Crusted Burrows	(±)	4 — 6 Months	Very High	Immuno-Suppression
Animal Scabies Urticarial Papules Vesicles ++ on Trunk	(+++)	< 2 Months	None / Rare	Sensitization

acarus. Extraordinary variations of the clinical and histologic pictures confusing it with dermatitis herpetiformis, nummular eczema, neurodermatitis, insect bites, lichen planus[22] and sometimes even reticulosis have been recorded[23].

(v) Norwegian scabies is the non-responsive negative end of the immunologic spectrum as we now understand. It is easy to demonstrate acari in these patients, if Kligman's dictum of "No acarus-no Norwegian Scabies" is remembered[24]. Diverse backgrounds of etiological factors are reported e. g. senile dementia, imbecility, lack of sensations as in syringomyelia, leprosy, tuberculosis, beri-beri, malnutrition and malignancy all of which possibly result in lack of immune-reactivity[25].

The diagnosis is often missed because of unusual clinical features which may elude diagnosis even for years. Many chronic dyskeratotic bizzare syndromes, with subungual, palmar and plantar hyperkeratosis, or atypical psoriasiform lesions with accumulated keratin debris may turn out to be this variety. Hyperpigmentation, depigmentation and alopecia may also be present. Burrows are masked by the keratin masses which harbour more than 2,000,000 mites[24] in a patient or about 200 mites per sq. cm[25]. The patient may seed about 2520 mites to 6312 mites per gm. weight of environmental dust[26], explaining sudden ward or family epidemics[24,27] arousing suspician of a patient with Norwegian scabies among the contacts.

Clinical pictures intermediate between Norwegian scabies and chronic scabies should be particularly expected in endemic areas with its many adverse ecologic factors.

(vi) Patients showing diffuse crusted lesions alone, with easily demonstrable mites, particularly among those undergoing prolonged topical or[28] systemic[5,29,30] immunosuppressive therapy are increasingly reported. Suppressed sensitization explain unusual pictures.

This may appropriately be called Iatrogenic scabies.

(vii) The last picture of scabies with severe symptoms on the trunk and avoiding the usual scabetic locations, is that of animal scabies[16,17,18]. Finally, one should remember infected scabies in children as a cause of nephritis which should be looked for.

Treatment

Gordon and Unsworth[31] mentioned about 450 references to treatment prior to 1945 out of which he reviewed the significant data. While going through the literature I found very few comparative assessments of the efficacy of different scabicides, and data on the possible development of drug resistance by[32] the acarus which should be expected from similar experiences in the mass treatment of lice[33,34]. Five traditional scabicides are mentioned by current text books[35,36]. Benzyl Benzoate 20% Benzyl Benzoate compound with DDT 1% and Benzocaine 10%, Gamma Benzene, Hexachloride (GBHC) 1% (Lorrxane in U. K ; Kwell in U. S. A.), Mono-sulphiran 25% successfully used in 1942 war, (Tetmosol in U. K.) as a solution for treatment and as a soap for prophylaxis and Crotamiton (Eurax).

The following table brings out Nair's[19] experience on the treatment of 184 patients from 50 families with

TABLE 2

Response of Scabies to Treatment

(Benzyl Benzoate 3 Applications)

	Adults (53)		Children (131)	
Relieved in	Itch	Lesions	Itch	Lesions
	%	%	%	%
1 — 10 days	44 (83.02)	52 (98.12)	95 (75.53)	127 (96.96)
10 — 20 days	9 (16.98)	1 (1.88)	36 (24.47)	4 (3.04)
Recurrences	7 (13.4)		32 (24.23)	

scabies at the K. E. M. Hospital, Bombay. Patients were treated with 20% Benzyl Benzoate, the only scabecide available following the sulfur era, at the public hospitals. (Table II)

Symptomatic response to treatment is not absolutely satisfactory nor apparently equal in both the adults and the children. Sizable groups of both (40%) showed persistent itching but less than 5% had lesions after 20 days. The recurrence rate after treatment is substantial (37%) being 13% in adults and 24% in children. About 10% of patients complain of more itching or irritation after benzyl benzoate application. Our results reflect either the failure of Benzyl Benzoate or the ecologic difficulties in an endemic, susceptible and underprivileged population with reinfective cycles from contacts. This situation leads to urban endemic scabies.

We had no opportunity to use the other scabicides in the hospitals. It is difficult to assess the results of treatment in private practice as the symptoms often persist due to hypersensitivity. However, I have found 1% GBHC to be satisfactory: I advocate 2 applications one week apart which I believe takes care of newly-hatched larvae from eggs which may remain unaffected. Patients of scabetic eczema with severe symptoms are often seen among children in private practice; they require additional steroids for 8 to 10 days. Those with severe pyoderma require antibacterial therapy in addition.

Crotamiton alone has not proved efficacious for treating scabies in our hands; nor does it relieve the pruritus.

Several questions remain unanswered namely the efficacy of various agents on the ova, (as the live forms such as larvae and the adult acarus easily succumb to pesticides) duration of their action on the skin, the potential of the scabicides going in chemical combination with keratin and sensitization and absorption and toxicity on repeated usage[37,38]. The use and efficacy of pesticides for mass treatment, and for disinfestation of fomites and the duration of their action in the environment are also to be worked out. Contrariwise, the effect of widespread use of pesticides including DDT & GBHC in man's environment on the incidence and prevention of scabies, is also not known.

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