Newer Studies on the Epidemiology of Fungous Infections of the Feet

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Aside from the obvious values in its practical applications, this paper is a wholesome reminder of the longevity of any hygienic misconception if, and when, it finds wide enough acceptance.

For years some measures for the prevention of fungous infections of the feet (tinea pedis, "athlete's foot," ringworm of the feet, epidermophytosis) have been based on the assumption that when an individual develops an attack of clinical fungous disease on the feet this represents a freshly acquired infection contracted directly from infected persons or indirectly from contaminated objects in the patient's environment. Among the supposed sources for these causal fungi are said to be carpets, bath mats, slippers, towels, etc., in the patient's home and particularly in areas in and around swimming pools and shower rooms.

If active fungous disease of the feet were really produced in this manner, it would justify the continuation of such attempted prophylactic measures as: the exclusion of persons with fungous infection of the feet from the use of public bathing facilities; the use of disinfectants on the floors of shower and locker rooms; the installation of antiseptic foot baths; and the use of individual disposable slippers. These and other rituals have been recommended over and over again for use not only in schools, gymnasiums, and other public places but even in the patients' homes.

This type of prophylactic measure has been advocated also in some textbooks and articles in the recent medical literature. How do these theories which form the basis of many public health measures against fungous infections of the feet agree with the available evidence from clinical and experimental studies? A survey of published reports dealing with the experimental transmission of tinea pedis has revealed a paucity of research. Von Graffenried,[1] Weidman,[2] and Peck [3] were able to induce clinical lesions in subjects by placing masses of culture material of fungi between the toes into skin which had been macerated for several days and was kept in a macerated state for sometime after inoculation, or by encasing the inoculated areas by binding with adhesive tape. Such severe, highly artificial measures, however, are no more likely to have bearing on the actual manner in which acute attacks of fungous infection of the feet are produced under natural conditions than does the fact that a pyogenic infection of the skin probably can be caused by scarifying the skin and then applying a dressing with material from a culture of Micrococcus pyogenes var. aureus to the area.

Many attempts have also been made to demonstrate the presence of pathogenic fungi on the floors of shower rooms and public baths. It was hoped that demonstration of their presence would support the assumption that acute attacks of fungous infections of the feet are due to an infection which has been picked up in these potential breeding

places. The vast majority of examinations, however, have been unsuccessful.[4-8] Recently, Ajello and Getz [9] were able to isolate a single culture of Trichophyton mentagrophytes (T. gypseum) from the tile floor of a shower stall, using a medium containing cycloheximide [10, 11] which is more or less selective for obtaining growth of certain pathogenic fungi. They also cited an earlier isolation of a pathogenic dermatophyte from a bath mat.[12] It can be argued that the failure to isolate pathogenic fungi from these sources may to a large extent have been due to overgrowth of saprophytic molds on the culture media—even though these media contained cycloheximide (our own unpublished studies and those of others). Nevertheless, it is remarkable that despite literally hundreds of unsuccessful attempts at isolating dermatophytes from areas in and around swimming pools and shower rooms the idea that these areas are important sources of infection producing acute attacks of fungous infections of the feet has persisted in the minds of public health authorities.

The shoes and socks of individuals with tinea pedis also have been blamed as a source of infection and especially of reinfection. It is not surprising that fungi have been found repeatedly in shoes and socks since these articles usually contain organic materials and are, in infected persons, constantly exposed to the fungi.[9, 13-15] However, their actual role as causes of infection or of acute attacks of fungous disease of the feet has never been scientifically investigated and proved.

For many years certain dermatologists have held the opinion that most of the available evidence speaks strongly against the causal role of exogenous exposure in acute attacks of fungous disease of the feet.[8, 16-20] They have postulated that the occurrence of clinically active disease is principally due to the lowered resistance to the fungi of certain sites on the feet of some subjects who previously had been carrying fungi in a latent, opportunistic, facultatively pathogenic state on their own feet.

If this theory is correct, and there is much evidence in its favor, exogenous infection would play a minor or negligible role in the causation of the acute attacks of fungous infections of the feet. In further support of this idea it is to be noted that Schramek [21] in 1916 already demonstrated fungi in clinically entirely normal skin areas in patients with fungous disease of the feet. Moreover, pathogenic fungi often have been demonstrated on the feet of persons who had no clinical symptoms of tinea pedis.[7, 19, 23-28]

The important role of local susceptibility is most obvious in subjects who have had clinically active tinea pedis recurrently or continuously over a long period of time. Often only a small part of the total surface of their feet is involved—despite what must be overwhelming exposure of the other parts. Hopkins, et al.,[29] found that the species of fungi causing fungous infections of the feet at a military post occurred in the same ratio in every group examined. If contagion were an important factor in the spread of the disease, one species of fungi would be expected to predominate in different groups of men in the

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same military post. Moreover, in recurrent attacks of fungous infection of the feet the organism responsible for the first attack was usually found in subsequent infections, strongly suggesting that in most cases recurrences were flare-ups of latent infections. In reporting on the results of a questionnaire answered by 88 of America's leading dermatologists,[18c] it was found that of the probably many thousands of patients with fungous infections of the feet and groin examined through the years by these specialists, a definite possibility of conjugal or familial transmission was proved in only four instances.

It seemed to us that while all the available evidence appeared to be against any important role of exogenous infection in the causation of acute attacks of fungous infections of the feet, a definitive answer to this problem could be obtained only through experimental investigation. Our studies which were carried out under a grant from the U. S. Army have not yet been completed. We are presenting here some of our results to date which have a particular bearing on the public health aspects of fungous infections of the feet.

Experimental

Studies were made to ascertain whether pathogenic fungi actually are shed in the scales from the feet of subjects with clinically active tinea pedis.

Seventy-eight subjects with clinical evidence indicating fungous disease of the feet were examined, and 28 subjects clinically free from the disease were also included.

Each subject immersed his feet for 15 minutes under standard conditions in 200 ml of sterile tap water in a sterile enamel pan. The foot bath water thus obtained was then centrifuged and the sediment was examined microscopically by the potassium hydroxide method and with the Hotchkiss-McManus stain, as well as in culture on Sabouraud's glucose agar and other media.

Results: In 54 of 78 subjects with clinically active lesions (69 per cent) fungous elements were seen microscopically in the foot bath water. Of these, 13 yielded cultures of pathogenic fungi (eight of Trichophyton rubrum, four of T. mentagrophytes, and one of Epidermophyton floccosum).

In four of 28 subjects (14 per cent) clinically free from fungous disease of the feet fungous elements were seen in the foot bath water and in one instance a culture of a pathogenic fungus (T. rubrum) was isolated. It is noteworthy that these four subjects when previously examined by the usual mycologic procedures had failed to show fungous elements on their feet.

Studies were made to ascertain whether foot bath water containing fungi is capable of causing acute attacks of fungous infections of the feet in subjects free from mycologic evidence of tinea pedis.

Forty-five subjects were included in these tests. Their feet were free from fungous elements at two microscopic and cultural examinations done at weekly intervals. One foot of each subject was exposed for 30 minutes to foot bath water containing fungi.

In 14 of 45 subjects the exposure was to the foot bath water freshly obtained from proved sufferers of active fungous infection of the feet. The remaining 31 subjects were exposed to foot bath water deliberately contaminated with viable fungi obtained from culture tubes. The number of viable fungous elements, as roughly estimated by the method of Friedhoff and Rosenthal,[30] varied between 680 and 52,000 per 200 ml foot bath with an average of 19,075. The other foot in all volunteers served as a "control" and was placed similarly in a foot bath which contained fungus-free sterile tap water. Subsequently, both feet of the volunteers were checked

mycologically once weekly for six weeks with scrapings examined both microscopically and by culture.

Results—Of the 45 subjects thus exposed not one developed clinical fungous disease. Eighteen (40 per cent) also failed to show either by microscopic examination or by culture fungi in scrapings from their feet at any time during the six-week period following deliberate exposure. In 27 (60 per cent) of the 45 volunteers fungous filaments were seen in one or more scrapings subsequent to the deliberate exposure to fungous contaminated water. In 23 volunteers fungi were seen in the scrapings from the exposed foot and in 15 from the control foot. In some of the subjects positive scrapings were seen more than once in the six weekly examinations. The total number of positive microscopic findings was 38 for the test foot and 26 for the control foot.

From two subjects cultures of T. rubrum were isolated on one occasion, although they had been exposed to water contaminated with T. mentagrophytes. This indicates that these two volunteers had a previous latent infection with T. rubrum, not discovered in the two preexperimental mycologic examinations, and that exposure to T. mentagrophytes did not result in a culturally detectable or clinically altered infection. In still another subject five weeks after deliberate exposure to T. mentagrophytes the same species was recovered in culture.

Among the 14 subjects who had been exposed to foot bath water from fungous infected volunteers, which presumably contained relatively minute quantities of viable fungous elements, only three showed mycelia upon subsequent examination (three on the test foot and two on the control foot). Among the 31 subjects who had been exposed to foot bath water deliberately contaminated with presumably larger quantities of fungous material, 24 showed mycelia upon subsequent examination (20 on the test foot and 13 on the control foot).

Comment

Fungous elements were found in the foot bath water of 69 per cent of 78 subjects with clinical fungous infection of the feet indicating that pathogenic fungi are readily shed from the feet of a majority of subjects with clinically active fungous infection. It should be noted that many in this group of subjects had been under treatment for their fungous disease. Perhaps in an entirely untreated group of subjects an even higher incidence of positive findings might have occurred. In addition, fungous elements were found in the foot bath water of 14 per cent of 28 subjects without clinical signs of fungous infection confirming earlier reports that persons may harbor pathogenic fungi on their feet in the absence of clinical changes. Moreover, our experimental results demonstrate that even in uninfected persons the quantity of fungi on the feet may be sufficiently great to be shed into the surroundings.

That the incidence of positive microscopic findings on clinically normal feet varies tremendously is shown by comparing our 14 per cent figure with the 40.8 per cent figure of Strickler and Friedman [23] and the 1.7 per cent figure of Ajello, et al.[25] Perhaps these differences can be explained on the basis of very marked variation in the degree of exposure to fungi between the groups of subjects examined. These very striking differences in the incidence of positive microscopic findings can be readily explained on the basis of our studies in which we attempted to infect subjects free from mycologic evidence of tinea pedis. The more massive we had made the exposure to pathogenic fungi, the more frequently it was possible to demonstrate fungous elements microscopically. Thus, mycelia were found

in only one-fifth of the volunteers who had undergone a light fungous exposure as contrasted with two-thirds of those with more massive fungous exposure. However, no matter how massive we made the exposures we were unable to detect any association between the finding of fungous mycelia on feet and any tendency to develop clinically active fungous infection during the subsequent six weeks.

The deliberate exposure of 45 subjects to fungous contaminated foot bath water (under conditions which probably were much more favorable to the development of tinea pedis than the usual actually occurring natural conditions) did not result in a single acute attack of fungous infection nor in any other gross clinical changes. It appears to us that our experimental results give overwhelming support to the previously available strong clinical evidence favoring the theory that acute attacks of fungous infections of the feet are usually due to a flare-up of a pre-existing latent, clinically or mycologically often not detectable infection. Contagion, i.e., exogenous exposure to pathogenic fungi, appears to play a minor or negligible role in causing clinically active, e.g., acute attacks of fungous infection of the feet.

The easy transmission of these microorganisms from one foot to another (and we may assume to other people's feet as well) is demonstrated by the fact that 15 of the 23 subjects who showed mycelia on their fungous exposed foot also showed mycelia on their "control" foot. The fungi on the clinically normal feet may be termed "opportunists," which probably when the local conditions are right (lowered skin resistance), multiply and cause clinical disease. Indeed, it is the letting down of the defenses on the part of the human host which leads to acute attacks of tinea pedis and not the contagion from the swimming pool, shower room floor, bath mat, or the thousand and one other objects which have been blamed and which have consequently been repeatedly sterilized, by exposure to heat, formaldehyde, and other methods.

It may be assumed that most human beings living in the United States begin to carry fungi on their feet in childhood or adolescence. They must be constantly contacting them in the many places where infected individuals deposit them daily. These young people develop clinical fungous disease only when unusual conditions occur. Until such time the fungi remain opportunists or facultative pathogens on their feet—of no more danger to their health than the staphylococci which also live on their skins. The fungi cause fungous disease on the feet and the staphylococci cause boils, folliculitis, etc., when the individual's resistance becomes lowered. In view of all this evidence some of the hitherto commonly instituted public health measures could well be discarded.

It is useless to attempt to sterilize articles in and around bathrooms, showers, swimming pools, etc., or to impregnate them with fungicidal agents. There is no evidence whatsoever that those areas are breeding grounds for pathogenic fungi despite the fact that fungi are apparently continuously deposited there from human beings with and without manifest fungous disease.

It is useless to attempt to sterilize shoes, socks, and other fomites of patients with fungous disease of the feet. They may often contain pathogenic fungi, but sterilizing them cannot sterilize the patient's feet which continue to carry and disseminate the fungi and which are inevitably undergoing frequent exogenous exposure. On the contrary, attempts at sterilization with chemicals are, in general, likely to do more harm than good. These chemicals may cause primary or allergic irritation of the skin. When this happens they lower the resistance of the skin to such an extent that the previously present fungi,

true to their character as opportunists, now find a fertile soil for multiplication with consequent development of the attack of clinical fungous disease. It is naive to expect that wading for a few seconds through a basin of antiseptic solution would help to prevent the transmission of fungous disease of the feet. These stagnating, unhygienic puddles should be abandoned.

There is no reason to practice exclusion of persons with tinea pedis from camps and other public facilities. The relatively small number of fungous elements which they deposit are unlikely to cause trouble, since even the large masses of fungi used in our attempts at experimental infection have been shown not to cause clinical disease in persons with adequate resistance.

What then are the measures which should be instituted to prevent active fungous disease of the feet? It appears obvious that one should make every effort to institute those procedures which will maintain or raise the resistance of the individual's skin. For example: (1) the use of perforated shoes whenever possible, and especially during the hot part of the year, in order to reduce the tendency toward moist conditions and maceration, and to prevent a relative shift of the skin pH on the feet toward the alkaline side; (2) perhaps the wearing of wool and cotton socks which are capable of absorbing moisture, rather than of nylon, rayon, and other nonabsorbing sock materials; (3) the regular use of a drying, mildly fungistatic foot powder (e.g., sulfur ppt, boric acid, tannic acid, zinc peroxide aa 5%, talc qs, or one of the commercially available fatty-acid containing powders); (4) the insertion of lamb's wool in the toe webs in persons with a tendency to interdigital maceration; (5) careful drying of the feet and changing to dry footgear whenever the socks and shoes become "soaked"; and as a logical measure (even though not yet experimentally proved) the use of nonalkaline soapless detergents for washing the feet rather than of ordinary toilet or other soaps which increase the tendency to alkalinity.

Summary

Experimental evidence has been presented that pathogenic fungi are readily shed from the feet of many individuals with and without clinically active fungous disease of the feet. Persons clinically free from fungous disease of the feet often—and perhaps usually—harbor pathogenic fungi on their feet, even if they cannot be detected in one or more careful clinical and mycologic examinations.

Attempts to induce acute attacks—or at least some evidence of clinical fungous disease of the feet in 45 subjects, mycologically free from the disease, by deliberate exposure to masses of pathogenic fungi in foot baths have been entirely unsuccessful. Not a single instance of fungous disease occurred within six weeks after exposure, although during this period fungi were found one or more times on the feet of 60 per cent of the exposed subjects. The ready transmission of fungi from the exposed feet to the unexposed "control" feet was demonstrated.

The results of these studies add convincing experimental evidence to the previously existing clinical data that exogenous exposure to fungi in swimming pools, shower stalls, bathrooms, etc., plays a minor or negligible role in eliciting acute attacks of fungous infections of the feet. As repeatedly stressed by certain dermatologists in the last 25 years, it is the decreased resistance of the skin of the human host with a resultant activation of the pathogenic fungi, previously lying dormant as opportunists on the patients' own feet, which is usually responsible for such attacks.

For the sake of re-emphasis mention is

made of certain hitherto commonly used, but ineffective and potentially harmful measures for the prevention of fungous infection of the feet which should be discarded. Similarly, suggestions are resubmitted for a few simple procedures designed to maintain and raise the local resistance of the feet and thus to prevent acute attacks of tinea pedis.

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