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Hot Tub Folliculitis: A Clinical Syndrome

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With the increasing use of hot tubs, patients are being seen with a distinct clinical syndrome that appears several hours or days after hot tub exposure. It consists of a maculovesicular, often pruritic rash, and commonly occurring associated symptoms including fever, upper respiratory tract complaints, axillary adenopathy and breast tenderness. Cultures in the cases described here grew out Pseudomonas aeruginosa, *giving a diagnosis of* Pseudomonas *folliculitis. The illness clears spontaneously without any treatment. Proper attention to hot tub chlorination and use are probably important in preventing this problem, and awareness of the syndrome by physicians may prevent unnecessary and costly diagnostic studies and treatment programs.*

THE USE OF home hot tubs and spas is popular in Northern California and is becoming popular elsewhere for relaxation and even social activities. Although this pastime is generally safe, certain health hazards exist. One such problem, *Pseudomonas* folliculitis, has been previously reported in several large groups of people using public hot water soaking facilities in health spas or motels.[1-5] It is now apparent that this can also occur sporadically in persons using home hot tubs. Our report is intended to alert physicians to this entity that may be seen more frequently as home hot tub use becomes more prevalent. We compare our cases with those previously reported to define a distinct clinical syndrome; we also discuss the possible causative factors, the course and complications and the implications of this syndrome for some patient populations.

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Report of a Case

A generally healthy 35-year-old man presented with a pruritic rash and general malaise, which had developed over one day. Physical examination elicited a temperature of 37.9°C (100.2°F). The skin of his trunk, upper arms and thighs had multiple papulovesicular lesions, some of which had pustular apices. The rash did not affect his palms, soles or mucous membranes. Axillary lymph nodes were enlarged, soft and very tender. There was no other adenopathy and the remainder of the examination showed no abnormalities. The pustules were lanced and the material cultured, growing out *Pseudomonas aeruginosa*. Cultures of a blood specimen were negative.

Later the same day, a tender rash developed on the patient's wife and 9-year-old son. Examination confirmed that their skin lesions resembled the patient's, and cultures of specimens from their rash grew the same organism.

Questioning indicated that these three family

members had been in their fiberglass hot tub with two friends about 36 hours before the onset of the patient's rash. A telephone check revealed that the friends had noted a mild rash of similar description, beginning at about the same time as the patient's and clearing spontaneously within two days. In addition, the man and woman both had sore breasts. Cultures were not obtained. An 18-month-old child in the patient's family had been in the hot tub off and on during the day of exposure and each of the following two days. One spot developed on her face that may or may not have been similar to the others' lesions.

A specimen of water from the hot tub was cultured and grew two *P aeruginosa* sp, one of which was identical in growth characteristics to that isolated from the patients' pustules. Serotyping of the *Pseudomonas* organisms was not done.

Affected family members were treated with local application of gentamicin ointment, but because of the axillary adenopathy and fever, the patient also received carbenicillin given orally. The 9-year-old child's symptoms cleared in two days, but the parents' symptoms lasted about seven days. There were no recurrences.

Discussion

Since 1975 five epidemics of documented *P aeruginosa* folliculitis have been reported from whirlpool spas and have involved groups of 27 to 75 people who were clinically ill.[1-5] One report[6] in addition to ours involved cases of two people exposed in their home hot tub. The clinical and bacteriologic aspects of all of these cases are similar. Hot tub folliculitis appears to be a discrete clinical syndrome and may occur in epidemics or as isolated cases.

In all instances there has been an incubation period from exposure to onset of symptoms. In the epidemics reported, the onset occurred from six hours to five days after exposure[1-4] and rashes developed in all five of our patients between 36 and 48 hours after being in the hot tub. The predominant complaint has been rash; lesions are papulovesicular on an erythematous base, 2 to 5 mm in diameter, and some vesicles develop pustular apices. The rash is present on the trunk, buttocks and proximal limbs, usually sparing the face and neck, soles and palms and mucous membranes. It is often but not always pruritic. As with our patients, associated symptoms occur in about half the reported cases and include weakness, myalgia, chills and fever, headache, earache, sore throat, enlarged axillary nodes or tender breasts.[1-5] In three of the previous reports[1,3,6] the rash cleared within ten days—which was similar to our cases. In one series the duration averaged 8 days but varied from 1 to 21 days.[4] Spontaneous healing is the rule though recurrences after initial healing may occur,[4] and Sausker and co-workers[2] described one apparently healthy woman who had several recurrences of follicular pustules over a period of three months following the initial folliculitis.

Few laboratory studies previously reported have included normal complete blood counts; our patient's leukocyte count was 11,900 per cu mm, with 71 percent polymorphonuclear leukocytes. Although Gram stain of material from the pustules was negative, we had no difficulty culturing *P aeruginosa* in specimens from all three of our patients in whom it was attempted. Burkhart and Shapiro[6] also cultured that organism from both of their patients. In larger studies, cultures were less frequently positive for *P aeruginosa* but in those situations many cases were evaluated retrospectively or the patients may have been seen later in their courses. Skin biopsies were done by Sausker and colleagues.[2] They found that the epidermis had minimal acanthosis and spongiosis; the dermis had a heavy inflammatory response with both round cells and polymorphonuclear leukocytes, and there was active folliculitis with disruption of follicular epithelium. Organisms were not identified in the biopsy specimens.

Prevention of hot tub folliculitis ought to be directed at its causes; however, the pathogenesis of this syndrome is not as clear-cut as one might expect, though it certainly is related to *P aeruginosa*.

Disinfection of hot baths would seem paramount and the Centers for Disease Control (CDC) have formulated guidelines for public spas and hot tubs.[7] They specify a free residual chlorine level of at least 1.0 mg per liter (ppm) and suggest regularly hyperchlorinating. One of the previous reports indicated an acceptable chlorine level,[2] though it was determined by the orthotolidine method, which is a less accurate measure of free chlorine than the DPD kit method recommended by the CDC.[7] The hot tub in our report had been chlorinated in the usual manner the day before, but the whirlpool jets had not been turned

on, and the chlorine may not have been well circulated; also, water in the jet system, which grew *Pseudomonas,* may not have been exposed to the chlorine.

The effectiveness of chlorination may depend on additional factors such as organic debris present in the tub from heavy usage and the temperature of the water. Kush and Hoadley[8] have shown that growth of *P aeruginosa* in warm water tubs is enhanced by organic substrate available in the water. Interestingly, in two epidemics[1,4] those exposed persons in whom symptoms developed were clustered in periods of high usage, and in a third epidemic more of the affected people had used the tubs late in the day.[2] Both circumstances might increase the organic substrate available for bacterial growth in the water. All reports of this syndrome are related to high temperature facilities and not to regular swimming pools, though some swimming pools investigated also grew *Pseudomonas.*[1] It has been suggested that high temperatures dissipate chlorination more rapidly than lower temperatures[2] and many *Pseudomonas* sp grow well at 41°C (105.8°F).[9] Japanese standards for tubs require temperatures of at least 42°C (107.6°F), which may suppress bacterial growth.[8] Information is not available in previous reports, but it seems very unlikely that any of the cases referred to in our paper involved pool temperatures that high.

Bathing with soap after soaking may be another deterrent; it reduced the likelihood of rash from 64 percent to 42 percent in Washburn and associates'[1] patients, but it was not significantly protective in that study, two others[2,4] or our own experience.

It has been suggested that *Pseudomonas* is the organism responsible for the folliculitis because of a specific exotoxin or combination of exotoxins that some serotypes might produce.[2] Many *Pseudomonas* exotoxins have been described.[10,11] Furthermore, one serotype, 0:11, has been isolated from patients' lesions and whirlpool water in four of five epidemics.[1-3,5] Against the serotype-specific theory, however, the same serotype was also isolated from whirlpool water at other spas or motels in investigative areas in two reports,[1,2] and at least two other serotypes have been reported in epidemics.[4,5] In other clinical settings, no relationship between site or type of *Pseudomonas* infection and serotype of the infecting organism has been shown.[12]

Not all persons exposed get ill, so host-resistance factors must play a role in whether infection and illness develop. In normal circumstances the invasion of healthy skin by *Pseudomonas* is rare, even though the organism may be present on the skin for a long time.[10] Experimental superhydration of the skin can induce a dense inflammatory *Pseudomonas* folliculitis similar histologically to hot tub folliculitis.[13] Although the experimental conditions are quite different from soaking briefly in a hot spa, it is possible that immersion is important in the breakdown of usual skin resistance in some people.

A predilection for infection might be suspected among persons lacking certain immune responsiveness to *Pseudomonas.* In general, this has been a clinical problem only in diseased patients with leukopenia or other immune deficient states and not in healthy persons.[14]

We are unaware of any report of hot tub folliculitis occurring in a person with diminished immune responsiveness, chronic renal disease or diabetes, or a case of *Pseudomonas* septicemia occurring following hot tub folliculitis. However, we are concerned that such persons might be more susceptible to the disease and perhaps should be warned of the potential risk.

There is no indication that antibiotic treatment, whether systemically or topically administered, is necessary or useful. The illness appears to run its course whether or not antibiotics are used, and it is important for a physician to realize, as we did not, that the presence of associated symptoms does not imply systemic spread of infection.

Conclusion

Hot tub folliculitis may become a more prevalent problem and awareness of the syndrome is important. When a patient presents to a physician without a history of epidemic exposure, especially if that patient frequently uses a home hot tub, but the physician overlooks the possibility that the patient has hot tub folliculitis, other more serious diagnoses will be considered. These include meningococcal or gonococcal septicemia, atypical viral illness, insect bites or scabies, herpes, other bacterial folliculitis, contact dermatitis and iododerma.

The diagnosis of hot tub folliculitis is made by an alert physician by history and physical examination. Usually no laboratory studies are necessary. The treatment is limited to reassurance and

an explanation to the patient of proper hot tub procedures.

REFERENCES

1. Washburn J, Jacobson JA, Marston E, et al: *Pseudomonas aeruginosa* rash associated with a whirlpool. JAMA 1976 May 17; 235(20):2205-2207
2. Sausker WF, Aeling JL, Fitzpatrick JE, et al: Pseudomonas folliculitis acquired from a health spa whirlpool. JAMA 1978 Jun 2; 239(22):2362-2365
3. Rash associated with the use of whirlpools—Maine. Morbidity Mortality Weekly Rep 1979 Apr 27; 23:182-184
4. Outbreak of *Pseudomonas aeruginosa* serotype 0:9 associated with a whirlpool. Morbidity Mortality Weekly Rep, 1981 Jul 17; 30:329-331
5. McCausland WJ, Cox PJ: Pseudomonas infection traced to motel whirlpool. J Environ Health 1975; 37:455-459
6. Burkhart CG, Shapiro R: Pseudomonas folliculitis—Development after the home use of personal whirlpool spa. Cutis 1980 Jun; 25:642-643
7. Centers for Disease Control: Suggested health and safety guidelines for public spas and hot tubs, HHS publication No. 99-960. Atlanta, CDC, 1981
8. Kush BJ, Hoadley AW: The association of *Pseudomonas aeruginosa* with commercial whirlpool bath waters, *In* Proceedings American Society Microbiology, New Orleans, May 9-13, 1977, p 271
9. Hoadley AW, Ajello G, Masterson N: Preliminary studies of fluorescent pseudomonads capable of growth at 41°C in swimming pool waters. Appl Microbiol 1975 Apr; 29:527-531
10. Young LS: The use of exotoxins in the pathogenesis of Pseudomonas infections. J Infect Dis 1980 Oct; 142:626-630
11. Liu PV: Extracellular toxins of *Pseudomonas aeruginosa*. J Infect Dis 1974 Nov; 130 Suppl (O):S94-99
12. Baltimore RS, Dobek AS, Stark FR, et al: Clinical and epidemiological correlates of Pseudomonas typing. J Infect Dis 1974 Nov; 130 Suppl (O):S53-59
13. Hojyo-Tomoka MT, Marples RR, Kligman AM: Pseudomonas infection in superhydrated skin. Arch Dermatol 1973 May; 107:723-729
14. Young LS: Role of antibody in infections due to *Pseudomonas aeruginosa*. J Infect Dis 1974 Nov; 130 Suppl (O):S111-118

Medical Practice Questions

EDITOR'S NOTE: *From time to time medical practice questions from organizations with a legitimate interest in the information are referred to the Scientific Board by the Quality Care Review Commission of the California Medical Association. The opinions offered are based on training, experience and literature reviewed by specialists. These opinions are, however, informational only and should not be interpreted as directives, instructions or policy statements.*

Challenge Food Testing for Respiratory Disorders

QUESTION:

Is it accepted medical practice to perform diagnostic evaluation of food allergy as a cause of respiratory disorders?

If yes, is it accepted practice to hospitalize a patient for a 96-hour fast followed by reintroduction of foods into the diet one at a time to observe symptoms? Are other diagnostic methods accepted for evaluation of food allergy as a cause of respiratory disorders?

OPINION:

In the opinion of the Advisory Panels on Allergy, Chest Diseases and Internal Medicine, it is accepted medical practice to perform diagnostic evaluation of food allergy as a possible cause of *certain* respiratory disorders. Although rare, some patients with atopic bronchial asthma and allergic rhinitis are allergic to one or several foods which may, under certain circumstances, exacerbate the respiratory symptoms.

Hospitalization for a 96-hour fast followed by reintroduction of foods to observe symptoms is not an accepted method of diagnosis. There are no scientific data available to support its use; it is unproved and possibly harmful.

Conventional elimination diets and/or double-blind food challenges on an outpatient basis are the accepted medical procedures. Where such methods fail, skin testing by a well-trained specialist and the RAST (radioallergosorbent test) when skin testing cannot be performed are additional accepted diagnostic methods.