Pseudomonas aeruginosa Folliculitis Acquired through Use of a Contaminated Loofah Sponge: an Unrecognized Potential Public Health Problem

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Pseudomonas aeruginosa folliculitis is a well-known entity that occurs among users of closed-cycle recreational water sources such as whirlpools, swimming pools, and hot tubs. In the absence of this epidemiologic link, isolated cases are difficult to diagnose. We encountered a patient who developed P. aeruginosa folliculitis subsequent to the use of a loofah sponge grossly contaminated with the same P. aeruginosa strain (serotype 10; pyocin type 1/a, 4,b) that was recovered from her skin lesions. Furthermore, we demonstrated that sterile unused loofah sponges can serve as the sole growth-promoting substrate for P. aeruginosa. To obviate the potential public health problem of contaminated loofah sponges, it is strongly recommended that manufacturers append, and consumers adhere to, instructions as to the care of loofah sponges, which includes allowing the sponge to dry after use.

Pseudomonas aeruginosa in the immunocompromised host is an important cause of systemic infection which may be accompanied by cutaneous manifestations including erythematous nodules, abscesses, vesicles, and cellulitis, erysipelas-like lesions, and eczema gangrenosum.

In the nonimmunocompromised host, P. aeruginosa has been epidemiologically associated with folliculitis that occurs in individuals who bathe in water contaminated with this bacterial species. Usually, Pseudomonas folliculitis occurs in outbreaks involving the use of closed-cycle recreational water sources such as whirlpools, swimming pools, hot tubs, private spas, and water slides. From 1972 through 1982, more than 74 outbreaks of Pseudomonas folliculitis have occurred in the users of health spas. Furthermore, a nosocomial outbreak of P. aeruginosa folliculitis occurred in association with the use of a physiotherapy pool.

In the clear-cut setting of outbreaks of folliculitis associated with the use of hot tubs and spas, the diagnosis of small pusular lesions on an erythematous base involving mainly the skin of the trunk, buttocks, legs, and arms is readily established. In the individual case, however, the diagnosis of Pseudomonas folliculitis may be overlooked unless a history of hot tub or spa use is elicited.

Alternatively, in the absence of any epidemiologic link to suggest Pseudomonas folliculitis, an array of potentially more serious etiologies may be entertained, including meningococcemia, gonococcemia, viral eruption, and contact dermatitis.

Documented herein is a case of Pseudomonas folliculitis that occurred in a healthy female patient in which the route of acquisition of her infection was through the use of a loofah bathing sponge grossly contaminated with P. aeruginosa. The epidemiologic link was solidified by showing that the P. aeruginosa skin and loofah sponge isolates were of the same serotype and pyocin type. Furthermore, the ability of loofah sponges to serve as the sole growth-promoting substrate for P. aeruginosa is also described.

CASE REPORT

A 25-year-old female administrative assistant sought medical advice for the occurrence of small discrete pusular lesions (Fig. 1) that randomly occurred over various parts of her body over a 2-week period. She noted the appearance of the first lesion on her abdomen, along the bottom edge of her bra and, then several lesions on her right and left legs below the knee and on the right calf subsequent to shaving her legs. Two days later she developed extremely tender axillary lymphadenopathy which made raising her arms difficult. Medical opinion of the etiology of these lesions ranged from a presumed viral exanthema to contact dermatitis or disseminated gonococcal infection. Several pusular lesions were evaluated by smear and culture. They revealed the presence of numerous polymorphonuclear leukocytes admixed with slender gram-negative rods which proved to be P. aeruginosa on culture. Because of this finding, she was questioned extensively about the usage of whirlpools, hot tubs, or swimming pools, all of which she denied. Unable to clearly establish an aquatic epidemiologic link to account for her acquisition of P. aeruginosa, she was further questioned about the use of wash cloths or sponges and admitted to using a loofah sponge which hung constantly in her shower stall. With this information, the patient was asked to bring to the laboratory her sponge and any other beauty aids such as lotions. Strikingly, on culture, the loofah sponge was found to be florid with P. aeruginosa (Fig. 2), while the other beauty aids were sterile.

MATERIALS AND METHODS

Both the skin and loofah sponge P. aeruginosa isolates were found to be identical by serotyping (serotype 10) and pyocin typing (pyocin type 1/a, 4,b), which were determined through the courtesy of J. Michael Janda, Department of Health Services, State of California, Berkeley.

To test the growth-promoting potential of loofah sponges for P. aeruginosa, a new loofah sponge, made in El Salvador and marketed by Schroeder & Tremayne, Inc., St. Louis, Mo., was purchased. After establishing its baseline flora by touch inoculating the sponge to a 5% sheep blood agar plate,
the sponge was sterilized by exposure to ethylene oxide. The sterility of the sponge was assessed by inoculating thioglycolate broth and 5% sheep blood agar with loofah sponge shavings. Subsequent to sterility assessment, fragments of loofah sponge (approximately 2 by 1.5 cm) were aseptically placed into tubes containing 2.5 ml of sterile distilled water (Baxter-Healthcare Corporation, Deerfield, Ill.) and individually inoculated with $10^3$ to $10^4$ CFU of *P. aeruginosa* isolated from the patient's skin and the loofah sponge. The inoculum was prepared by emulsifying several colonies of each isolate in 10 ml of sterile distilled water and washing and centrifuging the suspension three times, after which the optical density of the final suspension was adjusted to a 0.5 McFarland standard ($-10^5$ organisms per ml). One loopful (0.001 ml) was then added to tubes containing the loofah sponge fragments. Controls consisted of tubes containing sterile distilled water inoculated with the *P. aeruginosa* isolates as described above. All tubes were incubated at 35°C for 24 h, after which aliquots were examined in the wet state by phase-contrast microscopy and colony counts were determined subsequent to making serial 10-fold dilutions. The exact protocol was also followed for filter (pore size, 0.45 μm)-sterilized tap water to determine whether tap water as used naturally in showering or bathing is equivalent or better than sterile distilled water for the enhancement of *Pseudomonas* growth in the presence of loofah sponge fragments. All tests were performed in triplicate.

**RESULTS**

Prior to sterilization, new unused loofah sponges grew only scattered colonies of *Bacillus* species and *Staphylococcus epidermidis*. This result was in marked contrast to the virtual sea of colonies of *P. aeruginosa* recovered from touch imprints of the patient's in-use loofah sponge (Fig. 2). Aliquots taken from tubes containing the loofah sponge-*P. aeruginosa* mixture examined by phase-contrast microscopy were remarkable for the increased numbers and vibrant motility of the *Pseudomonas* cells, as contrasted to the occasional nonmotile to sluggishly motile bacillary forms observed in the distilled water or tap water control tubes.

In the absence of any other source of utilisable growth substrate, loofah sponge segments were growth promoting for the *P. aeruginosa* isolates from the patient's skin and loofah sponge. After 24 h of incubation, counts of *P. aeruginosa* increased from the inoculated $10^3$ to $10^4$ CFU/ml to $10^6$ to $10^7$ CFU/ml in the presence of only the loofah sponge. In the unsupplemented distilled water control, counts remained at $10^3$ to $10^4$ CFU/ml. Similar potentiation of growth was observed for a *P. aeruginosa* strain recovered from an in-use loofah sponge. Test results with tap water instead of distilled water were identical. In these experiments, the loofah sponge remained intact and was not degraded by the *P. aeruginosa* strains even after 7 days of incubation.

**DISCUSSION**

Loofah sponges, according to one manufacturer (Schroeder & Tremayne, Inc.), are derived from vegetable gourds of the cucumber family through a drying process which results in a fine network of woven (cellulose) fibers. The sponges are produced in a variety of sizes and shapes and are sold as beauty aids designed to remove superficial dried epithelial cells during bathing and showering prior to
the application of body oils and lotions. There are at least 77 varieties of loofah gourds which grow on a vine (1).

Several factors conjoined to predispose our patient to a loofah sponge-induced Pseudomonas folliculitis. Subsequent to use, the patient hung her sponge on the hot water knob in the shower stall to dry. She showered twice daily and recalled using the loofah sponge during her evening showers. It is highly plausible that the interval between showers was inadequate to ensure adequate drying of the sponge, which was 8 in. (20 cm) long and 4 in. (10 cm) in diameter. Additionally, her spouse, who did not use the loofah sponge (and, hence, did not have cutaneous lesions), also used the shower, thereby prolonging the wet phase of the loofah sponge.

The source of the loofah sponge-contaminating serotype 10 P. aeruginosa is unknown. It can be speculated that because our patient was a hospital employee, she may have become colonized with the organism during her working day and subsequently contaminated the sponge during usage. This link, however, is not absolute because the patient did not work in a clinical area but worked in an administrative office. Furthermore, in a survey of the microbial flora of 10 dry loofah sponges in personal use by the bacteriology laboratory technical staff, none grew P. aeruginosa. The one P. aeruginosa isolate obtained was from an in-use moist sponge of the mother of a high school student who was a volunteer in the hospital; that sponge was tested 2 days after the student initiated a liaison with our laboratory.

P. aeruginosa is an ubiquitous inhabitant of moist environments and has been recovered from sinks, baths, and tap water (5). In this regard, we suspect that our patient’s sponge was naturally contaminated by an environmentally derived P. aeruginosa. Failure to ensure adequate drying and the concomitance of organic debris, e.g., sloughed epithelial cells, favored pseudomonal growth.

While the above sequence of events referable to the loofah sponge—constant moisture, Pseudomonas contamination, and the presence of Pseudomonas growth-promoting food source—is adequate to ensure Pseudomonas proliferation in the loofah sponge, we identified either the loofah sponge itself or a solute from the sponge as a growth substrate for P. aeruginosa. This observation is not totally unexpected because P. aeruginosa possesses an array of exoenzymes which apparently are capable of degrading loofah sponge constituents in the absence of any other food source. Perhaps this innate pseudomonal capability is related to the common environmental habitat of gourds and P. aeruginosa. Exactly which loofah sponge component(s), e.g., cellulose or P. aeruginosa exoenzyme(s), is responsible for growth enhancement remains to be determined. To date, preliminary assessment by mass spectrometry of distilled water harboring loofah sponge fragments for up to a month has not revealed any Pseudomonas growth-promoting substrate.

The distribution of our patient’s pseudomonal lesions on the skin of her arms, abdomen, buttocks, and legs parallels that described previously (5, 12), as did her tender axillary lymphadenopathy. Thus, although her route of acquisition of P. aeruginosa was through the contaminated loofah sponge, the distribution of lesions and symptomatology parallels that which has been described for P. aeruginosa folliculitis associated with the use of hot tubs and whirlpools. In our patient, however, one may actually envision her coating the skin with a layer of P. aeruginosa derived from the grossly
contaminated loofah sponge. Minor trauma, e.g., shaving of her legs, probably served as a portal of entry for the bacteria. Loofah sponges are widely used as beauty aids. Indeed, a nationwide chain enterprise devoted to beauty products with a facility in New York City has on display numerous loofah sponges of various sizes and shapes. These were repackaged after purchase from a manufacturer. None of these contained the original manufacturer’s instructions to the user for the care of the loofah sponge after use, which includes the instruction to allow the sponge to dry thoroughly. Interestingly, the bacterial flora of a dry in-use loofah sponge is predominantly that of gram-positive cocci (staphylococci, micrococi) and Bacillus species admixed with a small number of Flavobacterium species and other nonfermentative gram-negative rods. If the loofah sponge is allowed to remain wet, especially after use, the microbial flora becomes enormous and shifts to predominantly gram-negative species, including P. aeruginosa (unpublished data).

Because of the widespread use of loofah sponges in the United States and the apparent failure of users to adhere to manufacturers’ instructions when given, they present a potential public health problem. To resolve this occult source of skin infection, it is recommended that (i) manufacturers readily append instructions and stress the necessity for proper care of their items, (ii) all repackaging of loofah sponges include the manufacturer’s care guidelines, and (iii) loofah sponge users adhere to care guidelines. In this fashion, one might reduce the incidence, presently unknown, of loofah sponge-induced P. aeruginosa or other bacterial folliculitis.

REFERENCES