EPIDEMIOLOGY, PATHOLOGY, AND TREATMENT OF AXILLARY HIDRADENITIS SUPPURATIVA

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Abstract—The epidemiology, pathology, and management of axillary hidradenitis suppurativa were examined in 11 patients with this disease. On the basis of this study, the following predisposing factors were incriminated in the epidemiology of the disease: predilection of the disease for women, obesity, hair removal by a safety razor, antiperspirants and deodorants, and local pyogenic infections distant from the axilla. Because antiperspirants do not effect transepidermal water loss of axillary skin, their potential deleterious effects are probably related to their chemical irritant effects on cut, nicked, or irritated axillary skin.

Treatment of this disease will vary according to its severity. Incision and drainage are usually needed to treat the localized disease. Specific measures must also be instituted to prevent progression of the disease (eg, weight reduction, avoidance of safety razor and antiperspirants and deodorants, and control of localized pyogenic infections distant from the axilla). Management of the chronic phase of the disease is primarily excision of infected axillary skin.

Keywords—axillary hidradenitis suppurativa; obesity; safety razor; antiperspirants and deodorants; pyogenic infections

Introduction

Axillary hidradenitis suppurativa (HS) is an inflammatory disease of the skin and subcutaneous tissue. The condition typically has an insidious onset with pruritus and hyperhidrosis being the earliest symptoms. As time progresses, the inflammatory process spreads to adjacent axillary skin, eventually resulting in extensive epithelialized sinus tracts within fibrotic dermal tissue. Recurrent infections result in a putrid, foul-smelling drainage that causes affected patients to limit their social contacts to the immediate family and security of their homes. On the basis of a comprehensive review of the literature, Mustafa et al concluded that there was a paucity of information about axillary HS. The etiology is unknown and there is no standard treatment.

In an effort to gain insight into the epidemiology, pathology, and management of this disease, the medical records of 11 patients with axillary HS were reviewed. Each patient was interviewed to identify factors that predisposed to this condition. The patient’s pathologic specimens were
R. F. Edlich, K. A. Silloway, G. T Rodeheaver, P. H. Cooper

examined to determine more precisely the histopathology of axillary HS. Experimental studies in normal human subjects were also initiated to ascertain the possible role of antiperspirants and deodorants in this disease process. The results of these clinical and experimental studies constitute the basis of this report.

Material and Methods

Clinical Data

Patients were selected for this study by surveying the medical records of the University of Virginia Hospital over a ten-year time interval (1973–1983). Eleven patients with HS were identified during this period of time. Clinical data were derived from a retrospective review of their hospital medical records and from personal interviews of all patients with axillary HS. The following information was obtained: race, sex, age, patient weight when symptoms of HS first appeared, anatomic location of HS, initial treatment prior to hospitalization, operative treatment, and associated local and systemic illnesses.

Personal interviews were directed at identifying possible factors that contributed to the development of HS. The patients were asked to estimate the rate of sweat production from their axillae. The diagnosis of hyperhidrosis was based on the subjective impressions of the patients and was judged to occur when sweating was typically noticeable under conditions where it would not normally be expected. The women were questioned as to their method of hair removal from their axillae (ie, safety razor with stainless steel blade, electric razor). The patients were asked if they encountered any adverse reactions to hair removal (nicking or cutting of skin) that were clinically disturbing and seemed to contribute to the development of HS. The patients were also asked if they used any antiperspirant or deodorant prior to their illness. Information regarding the brand name of the product and the vehicle of application (ie, roll-on, aerosol, etc) was ascertained. The patients were questioned about any adverse reactions (ie, pruritus, inflammation, etc) to the antiperspirant or deodorant that may have preceded the onset of HS.

Histopathology

Microscopic sections of 14 excision specimens of axillary skin from nine patients were reviewed. The number of slides per specimen ranged from four to 15. Many of the excisions included normal axillary skin with abundant apocrine glands, so that direct comparisons between normal and abnormal tissue were often possible. Particular attention was paid to the type and extent of inflammatory changes (chronic, subacute, and acute), the location of such changes within the specimen, and their spatial relation to squamous-lined sinus tracts. The presence or absence of eccrine and apocrine coils within affected skin was recorded, and acute or chronic inflammation of either eccrine or apocrine coils was specifically evaluated.

Antiperspirants and Transepidermal Water Loss

One possible mechanism by which antiperspirants and deodorants predispose to HS is that they form an occlusive film that interferes with transepidermal water loss (TEWL), thereby increasing moisture on the skin surface, which in turn encourages bacterial proliferation.¹ The validity of this hypothesis was tested by measuring the changes in TEWL following topical application of either antiperspirants or deodorants. The Evaporometer EPI developed by Nilsson and Lamke* was chosen because it obtains noninvasive, reproducible measurements of TEWL.⁴ Calibration

of the instrument was accomplished prior to the measurement and at 3-month intervals as suggested by the manufacturer.

Reproducibility of measurements obtained by different users of the instrument was demonstrated during preliminary trials. To ensure maximal reliability, all measurements were made by the same investigator on the same day in a closed room in which the humidity and temperature were approximately constant. Care was taken to ensure even contact pressure of the probe to the skin, a significant variable that must be controlled.

Transepidermal water loss was determined in seven healthy women volunteers (three black, four white) whose ages ranged from 18 to 20 years and who were not obese, as judged by the guidelines of Blackburn et al. Healthy volunteers were selected for this phase of this study because the deodorants and antiperspirants can be spread uniformly over their axillary skin. In contrast, the scarred and infected axillary skin of patients with axillary HS is not susceptible to a uniform application of these deodorants and antiperspirants. The subjects were not permitted to use either antiperspirants or deodorants for seven days prior to the study. Each subject remained in the room for at least 30 minutes before the experiment. They were required to remain seated prior to measurements. During the measurements the subjects lay supine with both arms extended to 180° to provide exposure of both axillae. A 2 × 2-cm area in each axilla was identified by making four dots with a marking pen in each corner of the square to ensure that the site of measurement was the same in each axilla.

The measurements of TEWL were taken ten minutes prior to and 30 minutes after the topical application of roll-on antiperspirants. One axilla of each subject was subjected to Right Guard™ antiperspirant and deodorant, while the other axilla was treated with Mitchum™ antiperspirant. The active ingredient in each antiperspirant was an aluminum zirconium tetrachlorohydrex glycine complex. The site of application of each antiperspirant in each patient was randomized. Using similar contact pressures, the roll-on antiperspirants were applied uniformly over the axillary skin. A new container of antiperspirant was used for each axilla. The statistical significance of the measurements was determined by the Student’s t test.

Results

Demographic and clinical details of the 11 patients under study are shown in Table 1. Each had one or more predisposing factors that could have contributed to the development of HS. When the patient’s weights were compared with the “ideal weight” proposed by Blackburn et al., all the women were judged to be overweight to a degree that ranged from 2 to 47 kg (Table 2). The two men were not obese. Four women and one man had subjective complaints of hyperhidrosis that preceded the onset of HS.

All patients used an antiperspirant or deodorant prior to developing HS. Six complained of adverse reactions (ie, pruritus, burning sensation, inflammation, infection) that they thought were related to the development of HS. The types of antiperspirants that elicited adverse reactions are listed in Table 3. In each case, the vehicle for transfer of the active ingredient was a roll-on applicator. Acne vulgaris was noted in one male patient prior to the onset of HS.

All women used razors with stainless steel blades to remove hair from their axillae. Three of the women had complaints of nicking or cutting the skin after shaving.

*Gillette Personal Care Division, Boston, Mass.
†Revlon Inc, New York, NY.
prior to the onset of HS. These adverse reactions to the razor caused them to discontinue the use of their antiperspirants or deodorants.

Treatment with Right Guard™ antiperspirant and deodorant resulted in no consistent change in TEWL of axillary skin (Figure 1A). In three volunteers there was a slight increase in TEWL (3%–16%), while a decrease in TEWL (6%–58%) was detected in the remaining subjects. Similarly, Mitchum™ antiperspirant had no

Table 1

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<th>Patient No.</th>
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*Initial Onset of HS.
†History of acne vulgaris.

Table 2. Weight of Patients With HS

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Table 3. Antiperspirants

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<tr>
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*Carter Wallace, New York, NY.
statistically significant effect on TEWL of axillary skin (Figure 1B). Topical application of this antiperspirant was associated with an increase in TEWL of axillary skin in three subjects (7%-40%), and a decrease in TEWL in four subjects (10%-40%).

Treatment of each patient consisted of surgical excision of the diseased skin and all hair-bearing skin with a 1-cm margin of normal uninvolved skin. The excision was extended down to deep fascia. In nine patients the wound was closed with minimal undermining (about 3 mm along each edge) using interrupted 3-0 absorbable suture in the dermis and interrupted 4-0 monofilament nylon skin sutures. In two patients split thickness skin grafts were required to resurface axillary wounds. Because the wounds in all patients were infected with a penicillin-resistant strain of 

\[ \text{Staphylococcus aureus} \]

a cephalosporin or a penicillinase-resistant penicillin was administered intravenously immediately before surgery, during surgery, and continued for two days postoperatively. Prophylactic drainage of the primarily closed wounds was not employed. Healing occurred without infection in all patients. No shoulder disability, contractures, or recurrence of the disease was encountered.

Microscopically, all specimens exhibited marked chronic inflammatory changes. There was extensive scar concentrated in the deep dermis and subcutaneous tissue. The scar contained patches of lymphocytes and plasma cells and scattered multinucleate giant cells, a few of which contained fragments of keratin. Both eccrine and apocrine coils were essentially absent within scarred areas and intact hair follicles were sparse. Embedded within the scar were variable numbers of sinus tracts lined by keratin-forming, stratified squamous epithelium. Some of the sinuses connected with surface epidermis via orifices that, except for their larger caliber, were indistinguishable from infundibula of hair follicles in adjacent, uninvolved skin. Rarely, such orifices contained hairshafts. The foregoing changes had a distinctly segmental distribution. The borders between normal-appearing skin and abnormal tissue were often abrupt. Rare eccrine and apocrine coils in affected areas exhibited chronic in-
flammation and fibrosis of their stroma when situated at the margins between normal and involved tissue, but none exhibited acute inflammation.

The specimens from two of nine patients had histologic changes limited to those just described. In addition to chronic changes, specimens from the remaining seven patients exhibited both subacute and acute inflammatory reactions. These were typically situated adjacent to and appeared to involve epithelial-lined sinuses. There were large patches and horizontally oriented tracts of edematous granulation tissue containing numerous capillaries, large numbers of lymphocytes and plasma cells, and scattered multinucleate giant cells. In some instances the lining of the adjacent sinus was intact, but in other foci there were segmental erosions of the lining. The lumens of the affected sinuses were either empty or contained remnants of keratin and sparse inflammatory cells. Granulation tissue adjacent to sinuses not infrequently contained flakes of keratin. Acute inflammatory changes, when present, consisted of dense neutrophilic infiltrates. These were typically mixed with the granulation tissue. Short segments of epithelium lining sinuses were heavily infiltrated by neutrophils and showed prominent degenerative changes. Purulent exudate was sparse or absent within sinuses, and there was little tendency for the exudate to extend upward within them toward the epidermis. Direct extension of acute inflammatory exudate through the superficial dermis to the epidermal surface was not seen.

Only three sections in two cases exhibited acute inflammation of rare apocrine coils. These were situated in skin that was otherwise normal. There were neutrophils in the stroma and lumens of secretory units and marked degenerative changes of apocrine epithelium. Portions of the coils were completely obliterated by inflammatory changes and lacked recognizable epithelium. Despite serial sections, it was not possible to relate inflamed apocrine structures to a specific follicle. In no sections did we observe superficial suppurative folliculitis.

The epidermis throughout most of the sections had a distinctly folded, accordion-like configuration. It was covered by a prominent layer of dense keratin. The orifices of normal follicles were typically wide, and follicular infundibula had goblet-like or distended contours. Typically, they were partially or completely filled with keratin.

**Discussion**

The development of infection is determined, in part, by a delicate balance between the number of bacteria present and the tissue's resistance to infection. Experimental studies performed in our laboratory demonstrated that the critical infective dose of pure cultures of obligate and facultatively aerobic bacteria was one million bacteria or greater per gram of tissue.6 The type of aerobic bacteria was less important in the development of infection than the number.

Host defenses of patients with axillary HS are usually normal.7 Nevertheless, since the axilla is warm, moist, and contains abundant secretions, it is a veritable haven for microorganisms sufficient in number to cause infection if the integument is disrupted. In their study of axillary microbial axillary flora in 25 healthy subjects, Shehadeh and Kligman8 found that most organisms were gram-positive, coagulase-negative staphylococci and coryneform bacteria. The latter bacteria are gram-positive, nonspore-forming pleomorphic rods. The aerobic coryneform bacteria of the skin are commonly known as diphtheroids, a name coined to indicate a close association with the diphtheria bacillus. The general term coryneform is more appropriate to these skin bacteria, because they resemble *Corynbacterium diptheriae* in little, but morphology. Staphylococci were present in nearly
100% of axillae; coryneform bacteria in more than 75%. The resident flora of these healthy subjects also included the gram-negative Aerobacter species and Alkaligenes fæcalis in almost one half and one third the individuals, respectively. The average bacterial counts of the right and left axillae of these 25 subjects were almost identical (2.36 millions/cm and 2.59 millions/cm, respectively). On the whole, individual variations from week to week were tolerably small, while the average weekly counts were strikingly constant.

Marples and Williamson found that, on average, persons with a predominance of diphtheroids in their axilla harbored total populations of several million per square centimeter, whereas those with more than 50% staphylococci averaged several hundred thousands total bacteria per square centimeter. They also showed that if the diphtheroids were selectively suppressed by antibiotic treatment, the staphylococci resistant to the antibiotic increased proportionately. Thus, diphtheroids appeared to limit the growth of staphylococci in the normal axilla.

In our studies several factors that appeared to predispose the patients to axillary HS were identified. There was a predilection of the disease for women. Nine of our 11 patients with axillary HS were women. Pigott and Ellis also reported that axillary HS primarily affected women (six of eight cases). In other series of axillary HS, however, the men and women were equally affected.

The susceptibility of a woman's axillary skin to HS may be related, in part, to the practice of axillary hair removal with a safety razor. Removal of axillary hair is aesthetically pleasing to women and is helpful in preventing axillary odor. The presence of hair greatly increases axillary odor, because it acts as a collecting site for axillary secretions, debris, keratin, and bacteria. Shaving and careful washing of the axilla reduces odor for more than 24 hours.

Shaving the skin with a safety razor is associated with well-documented deleterious effects. A safety razor consists of a blade held in a fixed geometry by the head. The razor blade transects the infundibulum of the hair follicle so that the wounded hair follicle provides access and substrate for bacteria. In addition, the impermeable corneal layer is damaged, resulting in an exudate that supports bacterial proliferation. In our experimental animals, bacteria inoculation of shaved skin resulted in dermatitis. In contrast, skin whose hair was removed by either an electric clipper or electric razor was refractory to bacterial contamination and did not develop dermatitis. The implications of the damaging effects of hair removal on the skin are being recognized by surgeons. In a prospective, randomized study by Alexander et al., the wound infection rate of patients whose hair was removed by a safety razor was significantly greater than that encountered in patients in which hair removal was accomplished by electric clippers. On the basis of these findings, these investigators recommended that electric clippers should be used to remove hair from the skin at the operative site and indicated that hair removal by a safety razor is potentially dangerous, inviting the development of infection. Prior to the development of axillary HS, three of our patients complained of nicking or cutting of axillary skin by a safety razor prior to the onset of HS. This interruption of the skin allows access to the millions of the microorganisms that reside on axillary skin. As in the case of the surgical patient, the razor compromises the local tissue defenses and appears to invite the development of HS. These deleterious effects of the razor could be prevented by using an electric razor for hair removal.

The absence of axillary hair resulting from shaving appears to be a peculiarity to Americans as an aesthetic standard. The incidence of axillary HS in women in other cultures who rarely shave axillary hair has never been documented. An odorifious study focusing on obscene pa-
patients who refrain from shaving and using deodorants should provide further insight into the epidemiology of this disease.

The number of bacteria in the axilla is probably enhanced further by increasing the moisture on the axillary skin. Experimental and clinical observations recently summarized by Kligman et al. consistently emphasized the importance of moisture in colonization of skin by potential pathogens. The relative dryness of normal skin in most parts of the body contributes to the marked limitation of growth of bacteria, especially gram-negative bacilli with their greater moisture requirements (Escherichia coli, Pseudomonas aeruginosa, etc).

A variety of factors that can potentially enhance the moisture content of axilla have been identified in patients with HS. Anderson and Perry reported that 20 (77%) of their 26 patients with HS were overweight. The excess weight ranged from 2.3 to 40.7 kg, with an average of 12.7 kg. Obesity was evident also in all women with axillary HS in our study. Obesity is prone to increase the moisture on axillary skin by maintaining an occlusive skin cover over the axillary skin. In the obese patient, the axilla is buried between the enlarged lateral thoracic wall and the upper arm. In the individual of normal body weight, the arm does not gain intimate contact with the thoracic cage in a resting position, thus allowing exposure of the axilla and permitting TEWL. Subjective hyperhidrosis preceded the onset of axillary HS in five of our patients. Brunsting observed several patients with HS whose illness was exacerbated by the use of aluminum-containing deodorants. Six of our patients thought that the application of antiperspirant preparations predisposed to the development of HS. They noted that topical application of these agents resulted in burning, itching, dermatitis, and infection, causing them to discontinue their use. Several mechanisms may account for the deleterious effects of these products on axillary skin. Some antiperspirants may produce an occlusive film over the axillary skin, impairing TEWL and increasing the moisture content on the underlying skin. This hypothesis was not supported by our measurements of TEWL before and after application of antiperspirants in seven healthy women. In our study, topical application of two different antiperspirants did not significantly alter the TEWL from their axillary skin. Steiner and Grayson believed that antiperspirants predisposed to HS through chemical irritation, inducing poral closure, or changing axillary flora. The warning labels on antiperspirants lend credence to their belief that antiperspirants may indeed be chemical irritants. These labels indicate to the consumer that antiperspirants should not be used on broken or irritated skin.

Long-standing pyogenic infections have also been associated with the development of HS. A history of acne vulgaris preceded the development of axillary HS in a male patient in our clinical study. Pigott and Ellis described a male patient who suffered severe acne as an adolescent, with the development of axillary HS at age 18. The frequent occurrence of HS among persons with seborrheic type of skin and associated acne was noted by Brunsting. Eleven of his 22 patients with HS showed severe acne scars of their face and trunk. Conway et al. noted that seven of their patients with HS showed severe acne scars of their face and trunk. An eighth patient displayed severe epidermophytosis and an active chalazion. Shelley and Cahn provided experimental evidence suggesting that occlusion of the follicular orifice (which also conducts apocrine sweat to the surface) was important in the pathogenesis of axillary HS. In each of 12 normal male adult sub-
jects between the ages of 20 and 40 they applied a perforated belladonna adhesive tape to one axilla that had been manually epilated. Three of the 12 developed suppurative apocrine hidradenitis that was thought, by the authors, to be histologically similar to spontaneously occurring axillary HS. Although we cannot exclude that this may be an important event in the early stage of the disease, we only observed rare similar changes in two patients. Moreover, it has not been proven that such lesions progress to the chronic condition encountered by clinicians and pathologists and recognized as HS.

All specimens in our study demonstrated chronic changes, and we believe that our observations provide some further understanding of the pathophysiology of HS. The major abnormalities consisted of deep dermal and subcutaneous scars; epidermal-lined sinuses; and chronic, subacute, and acute inflammation. The superficial half of the dermis was essentially spared. Although embedded in scar, the sinuses traversed normal dermis to connect with epidermis that was usually normal. It thus appears that the superficial portions of the sinuses, including their orifices, represent dilated and elongated, but otherwise unaffected, infundibula of preexisting hair follicles. The orifices of the sinuses rarely contained hair shafts, and the shafts were rare within scarred areas. It appears that early in the disease, the hair-producing portion of the follicle is destroyed. This correlates with the presence of alopecia in most of the axillary specimens and would indicate that residual hair shafts in the dermis have little role in perpetuating the chronic stage of HS. Both eccrine and apocrine coils were essentially absent from affected areas and are probably also destroyed relatively early during evolution of HS.

The existence of the sinus tracts, generated from follicular epithelium, probably accounts for the persistent clinical course of HS. The inflammatory changes were concentrated around the sinuses, and the acute and subacute changes appeared to have been secondary to segmental or extensive erosion of the sinus lining. The histologic appearances were indistinguishable from those observed in persistent and recurrently inflamed epidermal cysts. Increased activity of the disease may coincide with periodic overgrowth of bacteria residing within the sinuses and resultant damage and erosion of sinus lining epithelium.

The method of treatment varies with the stage of the disease. The acute phase should be treated early and intensively to avoid progression into the chronic phase. Incision and drainage are usually needed to treat the localized disease. Specific attention must be paid to each factor that predisposes to axillary HS. A strict weight reduction program must be instituted for all obese patients. Axillary hair must be removed with an electric razor rather than a safety razor. Antiperspirant and deodorants should be avoided. Local pyogenic infection (i.e., acne vulgaris, etc) must be aggressively treated.

Treatment of the chronic stage of axillary HS is primarily surgical. Almost all surgeons agree that the only chance for a lasting cure is complete removal of the hair-bearing area and underlying diseased deep dermal tissue in the axilla. After excision of the diseased tissue, a number of surgical techniques have been recommended for wound closure. Primary closure has been recommended by some surgeons. Split-thickness skin grafts have also been advocated to resurface large defects resulting from excision of the skin. Satisfactory results have also been obtained using local flaps. O'Brien et al used an anteriorly based Limberg flap in women and a posteriorly based flap in men.

Summary

A comprehensive study was initiated to determine the epidemiology, pathology, and management of axillary hidradenitis suppurativa (HS). In 11 patients with this
disease, specific predisposing factors were incriminated in the development of this disease. These factors included a predilection of the disease for women, obesity, hair removal by a safety razor, antiperspirants and deodorants, and local pyogenic infections distant from the axilla. The potential deleterious effects of antiperspirants and deodorants on the axillary skin do not appear to be related to its effect on transepidermal water loss, but rather to their chemical irritant effects on cut, nicked, or irritated skin.

The method of treatment of axillary HS varies with the stage of the disease. The treatment objectives of the acute phase is early incision and drainage of the localized infection and institution of specific measures to prevent progression of the disease (eg, strict weight reduction program, avoidance of safety razor and antiperspirants and deodorants, and control of localized pyogenic infection distant from the axilla).

Management of the chronic phase of the disease is primarily surgery. All hair-bearing skin with a 1-cm margin of normal uninvolved skin should be excised. The excision should extend down to deep fascia since the infected sinusoidal tracts involve primarily the deep dermal layers. The technique of closure of the resulting wound defect is dictated by the size of the defect.

REFERENCES