Diet in the prevention of hidradenitis suppurativa (acne inversa)

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Full control of hidradenitis suppurativa requires the prevention of new lesions. These appear to be induced by a complex series of hormonally driven molecular activities that lead to obstruction of the follicular duct, rupture and destruction of the sebaceous glands, the development of deep dermal sinuses that subsequently rupture to the surface, and production of an invasive subcutaneous mass that is resistant to medical therapy. Preliminary observations suggest that the use of a healthy and fully natural zero dairy and low glycemic-load diet may provide relief from progression of the lesions and possibly prevention of new lesions, even when medications fail. (J Am Acad Dermatol 2015;73:S52-4.)

Key words: acne inversa; diet; hidradenitis suppurativa; hormones; prevention.

Since Verneuil's description of hidradenitis suppurativa (HS) in 1854,[1] the main thrust of medical and surgical care has been directed at the therapeutic part of the management algorithm. As a result of work done in acne vulgaris—the miniature prototype of acne inversa (AI)—we are now in a position to look at the preventive side of that algorithm.

The deep sinuses, secondary pain, discharge, scarring, and odor in patients with HS—all are downstream effects of a rupture of the folliculopilosebaceous unit (FPSU). The initial plugging of the duct appears to be mediated by hormonal influences from 2 components of our "Western" diet: dairy products and highly refined simple carbohydrates.[2]

Dairy products contain 3 components that drive the process that blocks the duct and contributes to its leakage, rupture, and ultimate explosion. First, casein induces elevated levels of insulin-like growth factor (IGF)-1. Second, whey and simple carbohydrates raise insulin levels. Acting alone or in combination, insulin and IGF-1 stimulation (IIS) derepresses (opens) the androgen receptor, making it available to androgens from numerous sources: the testes, ovaries, stressed adrenals, from the FPSUs themselves, from various contraceptive hormones, and anabolic steroid supplements. Third, in addition to the classic driver of the various functions of the FPSU, 5α-reduced dihydrotestosterone (DHT), there is a 5α-reduced hormone from the human ovary (premenstrually) and at least 5 others present in cow milk itself—products of the bovine placenta and mammary glands. Even in those who consume no dairy, elevated insulin induced by diet predisposes to open androgen receptors that respond to endogenous and exogenous androgens.

The rupture induces an innate inflammatory reaction that destroys the pilar unit and the sebaceous glands themselves, leaving only small residual stubs of the sebofollicular canal attached to the follicular duct.[3] This residual follicular structure, with neither hair nor sebaceous gland attached, is still under the influence of androgens, so it continues to fill with tightly packed lamellae of incompletely differentiated ductal keratinocytes. These expand the ductal diameter, are unable to leave the duct, and so are recognized as the "tombstone comedones" that are the hallmark of the burned-out FPSU.

That same rupture produces 3 subcutaneous elements that are the prime movers of the disorder: the purulent inflammation, the development of single and interconnected epithelialized sinus tracts and the formation of the invasive proliferative gelatinous mass (IPGM).

While the purulent inflammation can be effectively reduced using anti-inflammatory drugs, even the most effective of them—the current biologics—are best considered a temporary symptomatic and

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calming measure.[4] This cooling of the purulent inflammatory processes allows time for conservative dietary and hormonal therapy to have their effect and provides the surgeon with an operative field that is better defined, minimized in extent, and more likely to heal effectively. These medications do not reduce or eliminate the sinuses or the IPGM.

Regrettably, following even the best-controlled studies, discontinuation of the active anti-inflammatory drugs (whether biologic, steroid, antibiotic, or some combination of these) is usually followed by recurrence. None of these modalities has ever shown a cure rate that exceeds the natural burn-out rate of spontaneous resolution, a relatively uncommon outcome. There is, however, in a growing lay literature, an increasing awareness of the effectiveness of dietary management. Social media have allowed sufferers from this disorder to find each other; various diets have been informally trialed and the results are being shared within the HS/AI community. There are now hints of success showing up in our clinical awareness and in informal trials, with patients acting as their own controls. In a personal dairy-free series, none of the 47 patients worsened; 83% improved to various degrees. No glycemic load restriction was used.

CAPSULE SUMMARY

- Hormones, particularly androgens, activate hidradenitis suppurativa.
- A dairy-free diet in 47 patients allowed 83% to improve and none worsened.
- Avoidance of dairy and high glycemic load foods that trigger androgen-driven obstruction of the follicular duct may reduce morbidity in hidradenitis suppurativa.

Formal clinical trials of such a complex problem are a major challenge. The ethics of asking patients to discontinue a major portion of their normal diet, to discontinue their present medications, to agree to avoidance of surgery—not to mention the impossible task of designing a truly blinded placebo diet—all conspire against the classic randomized clinical trial model. The lay population is therefore out in front, leading the charge, unfettered by the dictates of the US Food and Drug Administration, the formalized requirements of evidence-based medicine, the legal constraints of medical practice, and the need to have an institutional review board bless their investigative protocols. They are left with the freedom to truly experiment on themselves.

Self-experimentation is an age-old tradition. We can learn from it, guide the experimental subjects/volunteers toward science-based investigation, provide guidance in avoiding dietary disaster, and basically assist this community in their expression of translational research. They are not moving basic science just to the clinic but also beyond, to the patients themselves. Weisse[5] stated, "[T]he courage of those involved and the benefits to society cannot be denied."

There is a serious cultural barrier to such work, a misconception that stands in the way of many patients who are touched by this disease. This is the belief that the well-documented association of obesity with HS/AI represents a cause and effect relationship. This is a misconception shared with and perpetuated by their physicians and other care givers. We do see thin individuals with HS/AI. The classic picture is a lean, tense, and intense smoker who subsists on a diet laced with sugary sodas/soft drinks, often but not always caffeinated.

More commonly we see patients whose *dietary choices have induced 2 problems in parallel.* Such patients, often nonsmokers, consume the classic Western diet laden with dairy and simple carbohydrates—and it induces HS/AI as described above and also causes significant obesity. These patients may also be stressed (for reasons other than HS itself), and most have had many encounters with physicians during which the "weight" question has been brought up. This is a source of stress and anguish for many such patients. Physicians must tread carefully here. All parties must come around to the understanding that it is not overeating or obesity itself that causes HS/AI. It is the fact that, in a person with a structural sebofollicular weakness, *the foods that stressed and broke the duct also caused the obesity.*

Stopping the dairy whey/sugar >hyperglycemia >elevated insulin >hypoglycemia >hunger >sugar dietary cycling will correct the chronic hyperinsulinemia, insulin resistance, and all its downstream problems. Stopping all dairy foods will correct the casein/IGF-1—mediated side of the equation and will reduce both the androgens and the 5α-reduced precursors in dairy foods.

Certainly, surgery is sometimes essential to remove active areas and active lesions. Surgical cures have been achieved, but surgically removing all the susceptible FPSUs is out of the question. It is now possible, and much safer, to look to Dr Kligman's advice: help your patients "to actually achieve the ultimate goal in medical practice, namely prevention."[6] As patients search for an effective path to clearance, they need support and guidance to follow the most healthful diet available, free of

dairy and highly processed sugar and flour. Nothing could be more natural.

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