Correspondence

Hidradenitis suppurativa is acne inversa
Dear Sir,

We have examined the analysis proposed by Klaus Sellheyer and Dieter Krahl. This work is of particular interest to us because hidradenitis suppurativa is a fairly common and very disabling affection for which treatments are still mostly ineffective. Etiological circumstances remain unknown even though responsibility for the pathological process appears to be situated in the excretory canal of the apocrine gland, with no participation of sebaceous nor eccrine glands and no keratinization anomaly. The apocrine gland appears to be unaltered at the onset as demonstrated by the authors of this article. However, why mention acne since the sebaceous gland is not concerned any more than the eccrine gland; furthermore, if it is not acne, why use the term inverse? To speak of inverse acne solely on the argument of inversed topography could lead to the belief that there are common points between hidradenitis suppurativa and acne, whereas everything seems to oppose them:

- Heredity: the affection is transmitted on a dominant mode with variable expressivity; moreover, often associated with Dowling Dega disease, which follows the same dominant hereditary transmission.
- Trigger factor responsible for the expression of this affection is tabagism, which is found in 75–85% of cases. Passive tabagism has not been mentioned, but in a family where several members appear to be or might be carriers of the defect, non-affected subjects were nonsmokers.
- Onset circumstances: puberty, when the apocrine glands appear; prepubescent observations are so exceptional that they are published and no association with hyperandrogenism is found.

From their line of reasoning, the statement made by Plewig considers microcomedos as typical for acne inversa but also states that these microcomedos usually can be identified only histopathologically. The statement made by Bazex et al. that the “responsibility for the pathological process seems to be situated in the excretory canal of the apocrine gland” is based on erroneous speculations which we had hoped to rectify with our study of 176 cases of acne inversa. The authors have failed to produce any evidence whatsoever that an occlusion of the apocrine duct is central to the pathogenesis of acne inversa. In contrast, numerous authors from the UK, the Netherlands and Denmark, studying between 12 and 101 patients with acne inversa, came to the same conclusion as we did, namely that “hidradenitis suppurativa” is a disease of follicular occlusion rather than of the apocrine glands. Bazex et al. are incorrect in their assessment.

Dear Sir,

We appreciate the interest of Bazex, Bayle and Sans in our article about acne inversa. From their line of reasoning, however, it is unclear to us if the respondents have studied our histopathological observations carefully enough. We would like to respond to the various points mentioned by the authors:

1. The common denominator of the early pathogenetic events in acne vulgaris and acne inversa (“hidradenitis suppurativa”) is the occlusion of the follicular infundibulum. The authors of the letter are incorrect in their assessment that “comedos are not a characteristic element of hidradenitis suppurativa”. Not only have we demonstrated comedos histopathologically in acne inversa (see Figs 1 and 2 of the original article), their presence is also noted by other authors and are regarded as the hallmark of early disease develop-

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Reference

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that we simply “speak of inverse acne solely on the argument of topography”, as we have produced histopathological results to support our proposal. Bazex et al. are in line with the French surgeon Verneuil who noted in 1854: “[He] [Verneuil] make this observation only with reservations, for it is chiefly the curious distribution of these collections that made me adopt the interpretation which I give here [= that the apocrine glands are the center of events]. It is therefore a point to be restudied and to be demonstrated in a more satisfactory manner.” Bazex et al. have not produced new evidence “in a more satisfactory manner” to support their claim but continue to speculate on the pathogenesis of acne inversa.

2. As Bazex et al. pointed out that acne inversa can be associated with Dowling-Degos disease6–11 – although not “often”, as they stated, but rarely. This uncommon autosomal-dominant genodermatosis presents with an intratriggrous reticulon hyperpigmentation but is additionally characterized by comedos and periorial pitted acneiform scars. Co-manifestation of Dowling-Degos disease with acne inversa and multiple keratoacanthomas,12 or with acne inversa and multiple epidermal cysts,13 renders further evidence towards a primary defect of follicular keratinization. We also would like to point out that not only nonintratriggrous forms of acne but that also acne inversa can be part of autoimmune inflammatory diseases such as the SAPHO syndrome.14 Of note is also an association of acne inversa with pityriasis rubra pilaris.15 Although there is a genetic component to acne inversa,6,14 it is simplistic to reduce acne inversa to a genodermatosis, as the statement of Bazex et al. implies.

3. Smoking is clearly a triggering factor in acne inversa,18,19 but so is lithium.20,21 The latter can also be observed in association with papulopustular acne.22,23 Does the merely more common (and likely even relevant) association of a specific lifestyle factor with a disease entity exclude a close relationship of this entity to another one, in which this association is not so commonly observed? We also would like to point out that acne inversa can precipitate detrimental social problems which may enhance the likelihood of inducing soothing behaviors such as smoking. Do we know how many of the “75–85% of cases” of acne inversa quoted by Bazex et al. can be viewed as being triggered by smoking? How many of their (illusory) number of patients with acne inversa who smoke developed their behavior in response to the disease?

Exogenous triggering (e.g. by lithium therapy) and endogenous predisposition (as observed in the SAPHO syndrome) of acne inversa as well as acne vulgaris point to a close relationship of both entities and confirm our concept that they are part of a disease spectrum of acne.

4. “Onset circumstances” do not allow differentiating acne vulgaris and acne inversa. Adult onset acne is not uncommon and patients with this disease are of the same age group as those with acne inversa.14 While it is widely accepted that acne vulgaris is an androgen-mediated disease, so is the evidence that this might also be true at least in part for acne inversa.35–37

5. We wished the respondents had presented evidence for their statement that “the lesions [of acne inversa] are often preceded by acute hidradenitis”. Our experience is the opposite and the histopathological data we presented (which included also early lesions) prove the absence of a primary involvement of the apocrine glands, hence the absence of “acute hidradenitis”. The “fibrous retractions of the axillar or inguinal areas”, as mentioned by Bazex et al., are not of an “unknown cause”. They are the direct consequence of an extensive inflammatory process which cannot be contained and results in scar formation.4,8 Bazex et al. are incorrect in their statement that “the constitution of fistula [commonly present in acne inversa] are never observed in acne”. Fistulas are a common feature in cystic acne, acne conglobata and acne fulminans.6,15 Would the authors deny a relationship of cystic acne, acne conglobata or acne fulminans with acne vulgaris?

6. We agree completely with the respondents to our study that surgery is often justified in acne inversa; however, not because the entity is conceptually different from acne but because of the more extensive scar tissue formation. On the other hand, in our experience antibiotic therapy is rarely successful in containing the disease process. This is also in agreement with the majority of the literature.3,4,6,11

7. We agree with Bazex et al. that acne inversa usually “does not respond to any of the treatments for acne”. We outlined the reasons earlier but this does not argue against the relationship between both entities. It only indicates a more severe tissue destructive process in acne inversa vs. acne vulgaris.

In summary, we were surprised by the lack of reference Bazex et al. made to the results of our histopathological study and their seemingly existing ignorance of the plethora of histopathological evidence produced by numerous other authors (quoted in 1) in support of the concept of acne inversa as a disease of follicular occlusion rather than as a primary disease of the apocrine glands. In our study, we purposefully included early disease states of acne inversa and we were stunned by the similarity of the histopathological findings of early acne inversa with acne vulgaris. This included a lack of involvement of the apocrine gland and the presence of follicular occlusion in both entities. The former justifies the replacement of the term “hidradenitis suppurativa” in favor of acne inversa. This should help in the clinical management of these difficult to treat patients and, specifically, should avoid long-term antibiotic therapy of a disease process erroneously perceived to represent a primary inflammation of the apocrine glands. In sum, “hidradenitis suppurativa” is acne inversa!

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An example of photo-aggravated lichen sclerosus
A 14-year-old girl presented with a 4-year history of an asymptomatic eruption. The lesions first appeared on her forehead after episodes of moderate sunburn whilst on holiday abroad. Further lesions appeared on sun-exposed areas, such as the shoulders and upper arms, after further episodes of intense sun exposure or sunburn. She was otherwise well. Her maternal great aunt suffered with lichen sclerosus and it was suspected that her maternal great-grandmother also had a history of the same condition.