the patients we reported. However, several comments on Dr. Don's case report are in order:

1. We have not had experience with capsaicin in acute zoster pain. However, while we have observed patients with chronic postherpetic neuralgia who have reported relief of pain after 2 or 3 days of capsaicin treatment, these are a distinct minority. Chronic neuralgia patients treated in our open-blind study, as well as the vast majority of those we have treated in a large ongoing double-blind clinical trial, have not noted significant relief of pain until after 2 to 4 weeks of treatment. Therefore, we would not want the patient with chronic pain or the treating physician to have overly optimistic expectations of the onset of clinical response. Such unrealistic expectations could interfere with patients receiving a sufficient course of therapy.

2. The patient reported by Dr. Don had no further pain after discontinuing topical capsaicin following 4 days of regular usage. In our experience, which involves long-term follow-up of over 30 patients with chronic postherpetic neuralgia for periods of up to 18 months after they first were treated with capsaicin, such a dramatic response was not observed. A number of patients who had good pain relief on capsaicin treatment for lengths varying from 2 to 6 months had a recurrence of pain once capsaicin was discontinued and they had to be restarted on capsaicin.

3. It is, of course, possible (and maybe even probable) that the differences in the time course of the responses we have observed to capsaicin versus that described by Dr. Don can be explained by our patients having pain for 6 months or longer and his patient having pain of only 2 to 3 weeks' duration. It is generally thought that acute neuralgic pain involves exclusively a peripheral component, while neuralgias of longer duration may involve an additional central component to the pain. For this reason, capsaicin, which is thought to act principally on a peripheral chemoreceptor of pain, might be more effective in relieving the pain of acute neuralgia than that of chronic neuralgia. We have generally avoided treating such acute cases with capsaicin because of our concerns about application of the agent to open lesions, which possibly could cause exacerbation of pain.

Dr. Don's observation is thought-provoking. If his results can be replicated in a much larger sample under a double-blind paradigm, capsaicin cream might offer safe and effective prophylaxis against postherpetic neuralgia, rather than simply a safe and effective treatment for this chronically painful disorder.

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Acne necroticans (varioliformis) and Staphylococcus aureus

To the Editor: Kossard et al performed a great service to dermatologists by once again focusing attention on acne necroticans (varioliformis), a not uncommon and long-recognized dermatologic condition much neglected in the literature.1

One is surprised that the authors of the article, “Necrotizing Lymphocytic Folliculitis: The Early Lesion of Acne Necrotica (Varioliformis),”2 did not culture the lesions. The association if not the etiology of this condition with S. aureus has been well documented.3 In addition, they did not cite Maibach's fastidious documentation of scalp folliculitis induced by Corynebacterium (Propionibacterium) acnes.4

I have collected a series of thirteen patients with this condition. Eight are female and five male. One was in her teens, one in her twenties, two in their thirties, and the rest over 40. Bacterial cultures revealed oxacillin-sensitive S. aureus in eleven, trimethoprim sulfasensitive Klebsiella pneumoniae in one, and Acinetobacter calcoaceticum in the other. Questioning or observation by next of kin revealed one common factor in all, "the patient picks or manipulates the lesions." Several complained of itching of the lesions, an observation noted by others and one that induces scratching.5

A recent spate of correspondence in The Schoch Letter documents clinicians' therapeutic frustrations with this venerable condition.* My hypothesis is that the initial lesion of acne necroticans is a folliculitis, probably a rosacea variant, as speculated by Kossard et al, with Propionibacterium acnes as the microbiologic agent. The patient manipulates and excoriates the initial folliculitis, papules, and pustules, which progress to the crusting, necrotic eschars and ultimately scarring lesions constituting this condition, a situation emphasized by Calnan and O'Neil.6 Treating the folliculitis with appropriate antibiotics is inadequate. The therapist must contend with the auto-induced component that may be construed as neurotic or anxiety-induced. Patients admit to being "highstrung" or intensely anxious.7 In two elderly patients the onset came after diminished mentation following cerebrovascular incidents. In several instances, situational factors were cited, that is, loss of job or spouse and marital or family discordance.

I prescribed doxepin for the psychologic aspects, for this tricyclic’s combined antidepressive8 and, in addi-

tion, antipruritic effects, together with the appropriate antibiotic, dicloxacillin for those with *S. aureus*-positive lesions and trimethoprim sulfa for those culturing negative organisms. Topical therapy prescribed was clindamycin in alcohol solution (Cleocin T).

Dramatic and gratifying resolution occurred in most within 2 to 4 weeks. Three patients could not tolerate doxepin because of drowsiness but could tolerate amitriptyline (two patients) or perphenazine-amitriptyline (one patient). In addition, I instructed the patient to apply antibiotic ointment to the nares, axillae, groin, and under nails and to cut the nails closely to eradicate reservoir bacterial sites.

Follow-up by phone call or office visit 3 to 6 months later revealed nine patients disease-free and four with recrudescence. The two with cerebral compromise required intermittent antibiotic and doxepin therapy for lesions control (the relatives noted reflex picking), while the other two cleared with a repeat course of antibiotics and tricyclics. In three patients not responding to dicloxacillin within 4 weeks, a 10-day course of rifampin with dicloxacillin was prescribed according to the method of Wheat et al.

In conclusion, acne necroticans, as Kossard et al clearly and definitively demonstrate, begins as a lymphocytic folliculitis that may well be triggered by *P. acnes*. The patient manipulates and excoriates the lesions, probably because of a psychologic overlay, and the crusting, necrotic, scarring process ensues. Resolution of this distressing, unsightly, and disfiguring condition is possible with the use of the appropriate antibiotics, as demonstrated by culture, and a tricyclic compound, which may control the pruritus-induced manipulation and excoriation as well as the anxiety, depression, or agitated depression creating reflex excoriations.

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REFERENCES


Reply

To the Editor: We thank David Fisher for sharing his experiences with acne necrotica and in highlighting the possible role of bacteria, particularly *Propionibacterium acnes*, and the neurotic overlay found in many patients. However, the disorder described as acne necrotica appears heterogenous and at least two subtypes can be distinguished. There is the relatively common form of acne necrotica miliaris, which is usually confined to the scalp and is characterized by superficial follicular vegetulopustules. The primary lesion is difficult to sample because it is often excoriated. The finding of pustules has prompted bacterial culture, which have grown *Staphylococcus aureus*, particularly, as in Fisher's series, although Maibach isolated *Corynebacterium (Propionibacterium) acnes* in a group of patients who had superficial follicular pustules of the scalp when first seen.

The patients we studied had lesions that conformed more to a variant of acne necrotica termed *acne necrotica varioliformis*. It is characterized by umbilicated, erythematous papules undergoing necrosis, which healed with pitted scars. Pustules, when present, appeared as a late event. In contrast to acne necrotica miliaris, the lesions occurred particularly over the face and upper part of the trunk, as well as the scalp. Although some authors have described patients with both types of lesions, we did not observe this combination in our patients. Fisher does not provide sufficient clinical details to assign his patients to these subgroups.

Maibach reviewed six biopsy specimens from his patients with *Propionibacterium* folliculitis of the scalp and found superficial follicular pustules with a mixture of polymorphonuclear and mononuclear cells. This finding contrasts with our histopathology of acne necrotica varioliformis, showing a necrotizing lymphocytic folliculitis as the early event in this disorder.

Whether all cases of acne necrotica varioliformis will show this pattern of pathology remains to be seen because it is possible that even this morphologic presentation may represent a heterogenous group of disorders.

The role of bacteria in acne necrotica varioliformis