accept a new CareSource patient, with an acceptance rate of 17% (Table 4).

Comment. While privately insured patients enjoyed a 91% acceptance rate and relatively shorter wait times, the publicly insured patients faced significantly lower acceptance rates and longer appointment wait times.

Despite making up less than 20% of the workforce, academic dermatologists provide most of the care for Medicaid patients in Ohio. Not only does this limit access to care for the publicly insured, but it also places significant strain on academic dermatology. In addition, the lower reimbursement, and resulting lower physician compensation, may make it more difficult for academic centers to recruit and retain faculty.

The burden faced by academic dermatologists is evident in the data—with an average wait time almost twice that of private dermatologists. With implementation of the health care reform bill and expansion of Medicaid eligibility, longer appointment wait times are inevitable, not only for those with public insurance, but also for those who need the subspecialty care that is often available only at academic centers.

<table>
<thead>
<tr>
<th>Practice Type</th>
<th>No. of Requests</th>
<th>No. Accepted (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Academic</td>
<td>16/16 (100)</td>
<td></td>
</tr>
<tr>
<td>Private</td>
<td>15/88 (17)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>104</td>
<td></td>
</tr>
</tbody>
</table>

*Findings of the proportion test (z = 6.67, P < .001) suggest that the rate of acceptance for CareSource patients is higher among academic dermatologists (16/16 = 1) than it is among private dermatologists (15/88 = .30).

Table 4. Acceptance Rates for CareSource Patients Based on Dermatologist Practice Type

**Topical Aprepitant in Clinical and Experimental Pruritus**

oral aprepitant is a nonpeptide inhibitor of tachykinin receptor NK1 approved for use as an antiemetic drug for chemotherapy-induced nausea. Duval and Dubertret first reported that peroral aprepitant effectively reduced pruritus in 3 patients with Sézary syndrome. Similar results were obtained later for 6 of 7 patients with erythrodermic cutaneous T-cell lymphoma. Stander et al reported an open-label study of 20 patients with chronic pruritus treated for a week with peroral aprepitant. It has been suggested that the effects of aprepitant are related to its preventing of mast-cell activation in the skin. The aim of the present study was to test this hypothesis with topical aprepitant.

Methods. The experimental protocol for both patients and healthy subjects was designed as a randomized, double-blind, vehicle controlled, right-left study and was approved by the local ethics committee. All participants gave their written informed consent. Aprepitant was blended at a 5% concentration in a lipophilic vehicle in accordance with the European Union Good Manufacturing Practice rules. A visual analog scale (VAS) was used to score pruritus.

Thirteen patients (8 women and 5 men; age range, 33–82 years; median age, 57 years) treating 15 symmetrical skin regions completed the study (Table). The patients received 2 boxes marked “left” and “right,” respectively, each containing 3.5 g of a cream to apply topically on a single occasion. They were asked to evaluate their pruritus before, 30 minutes after, and 2 hours after the treatment.

Seven healthy nonatopic volunteers (6 women and 1 man; age range, 37–56 years; median age, 52 years) were enrolled. Aprepitant and the vehicle were applied to the volar surface of the left and right forearms, respectively, and left on for 30 minutes. The cream was then wiped off, and transepidermal water loss (TEWL) was measured using a closed-chamber device (VapoMeter, Delfin Technologies Ltd) to study the effects of the cream on the skin barrier. Thereafter, both forearms were pricked with histamine. The flare and itch were evaluated after 5 minutes, the weal after 15 minutes.
Results. Topical application of aprepitant, 5%, did not attenuate clinical pruritus (Figure). Erythema of dermatitis in those who had it was also unchanged. The mean (SD) VAS scores for pruritus were 4.5 (2.0) prior to treatment with aprepitant, 4.1 (2.2) after 30 minutes, and 2.8 (1.6) after 2 hours. The corresponding values on the vehicle-treated side were 5.1 (2.2), 3.4 (1.9), and 2.8 (1.9), respectively. Both treatments improved the skin barrier with significant reduction in TEWL: 4.0 g/m²h after aprepitant application; 5.1 g/m²h after application of vehicle alone; baseline TEWL, −10.4 g/m²h (P = .001).

The mean (SD) VAS scores for pruritus induced by prick-test reactions to histamine were 4.3 (3.4) on the aprepitant-treated side and 4.8 (2.4) on the vehicle-treated side (P = .22). Also, the mean (SD) area of the histamine-induced flare (900.0 [246.4] mm² vs 816.8 [296.1] mm²) (P = .17) and volume of the weal (47.5 [28.6] mm³ vs 54.6 [34.9] mm³) (P = .30) were unchanged.

Comment. A single topical application of the aprepitant in our study failed to attenuate clinical itch and erythema. This finding is in line with previous observations of aprepitant having no effect on erythroderma in 8 patients with T-cell lymphoma.1,2 The lack of effect of aprepitant in the present study could be explained by insufficient permeation of the skin by our formulation. However, the TEWL values indicate that both formulations were absorbed into the skin.

The rationale for treatment of pruritus with aprepitant is the occurrence of tachykinins in sensory nerve fibers as well as in the central nervous system. Injected into the skin, tachykinins induce a dose-dependent itch and flare that can be inhibited by antihistamines.3 In the present study, aprepitant failed to inhibit either pruritus or flare when histamine was pricked into the skin. This finding contradicts the explanation that aprepitant may prevent mast-cell activation.4

The discrepancy between our results using topical aprepitant and the reported effects of systemic treatment on itch associated with chronic skin disease may be due to the effects being mediated by central neuronal mechanisms.3 However, systemic aprepitant has been shown to be ineffective in the treatment of pain.5,7

Accepted for Publication: February 15, 2012.

Author Affiliations: Department of Dermatology, Skane University Hospital, Lund, Sweden.

Correspondence: Dr Wallengren, Department of Dermatology, Skane University Hospital, Getingev 221 85, Lund, Sweden (joanna.wallengren@med.lu.se).

Financial Disclosure: None reported.
Funding/Support: The study was supported by the Skane County Foundation and the Welander and Finsen Foundation.

Additional Contributions: Nurses Inger Nordgren, RN, Anne Nielsen, RN, Jonna Gammelmark, RN, Anna E. Mårtensson, RN, and Carina Lundblad, RN, helped recruit the patients. APL, Umeå, Sweden, manufactured the aprepitant formulation, which was purchased at regular price for purposes of this study.


VIGNETTES

Eczematous Drug Eruption After Ustekinumab Treatment

Ustekinumab is a human monoclonal antibody that blocks interleukin (IL)-12 and IL-23, 2 cytokines that play an important role in the pathogenesis of psoriasis.1,2 It was approved in 2009 for the treatment of adult moderate to severe chronic plaque psoriasis. We report herein the first case to our knowledge of a patient with an eczematous eruption that appeared after the administration of ustekinumab.

Report of a Case. An 82-year-old woman with no history of atopic dermatitis, eczema, or allergy was seen for cutaneous plaque psoriasis and palmoplantar pustular psoriasis, initially treated with phototherapy, methotrexate, adalimumab, and etanercept without improvement. A treatment with subcutaneous ustekinumab (Stelara; Janssen-Cilag) was then started and administered at weeks 0 and 4 and every 12 weeks thereafter (45 mg per injection). Slow but progressive improvement was observed over the first month of treatment.

Three days after the second injection, she developed a diffuse pruritic rash. Physical examination revealed diffuse maculopapular and erythematous lesions, especially on the trunk and medial aspects of the limbs. Histologic examination showed epidermal spongiosis, focal and crusted parakeratosis, edema in the epidermis and upper dermis, and a discrete lymphocytic perivascular infiltrate in the dermis, all consistent with the diagnosis of psoriasiform eczema. The eczematous lesions improved within 2 weeks following topical steroid treatment.

The third ustekinumab injection was followed by worsening of the eczematous reaction, while the psoriasis palmoplantar lesions remained unchanged. Ustekinumab treatment was discontinued, and all lesions completely resolved 2 months thereafter.

Comment. Ustekinumab is a human monoclonal antibody that specifically binds to the p40 subunit common to both IL-12 and IL-23. Ustekinumab prevents IL-12 and IL-23 from binding their cell surface receptor complexes, thereby blocking the T-helper cell 1 (Th1) (IL-12) and Th17 (IL-23) inflammatory pathways implicated in the immune responses in psoriasis. It is important to emphasize that IL-12 stimulates the production of several cytokines, including interferon γ and tumor necrosis factor (TNF) by T lymphocytes and natural killer cells. Furthermore, IL-23 induces upregulation of TNF via Th17. Thus, ustekinumab also partially acts as a TNF inhibitor. While TNF inhibitors have been associated with several cutaneous adverse effects, ustekinumab has not been involved in skin adverse effects except for injection-site reactions occurring in 1% to 2% of patients.3

Figure 1. Eczematous eruption of the trunk (A) and buttocks (B) 3 days after the second injection of ustekinumab.