patients were male; the majority (10; 67%) were non-Hispanic white with mean age 53 years. Six of the 9 participants who enrolled for 4 weeks continued for 12 weeks. There was no difference in the mean percent change in GLII mRNA levels between itraconazole and placebo groups at 4 weeks (132% vs 19.0% from baseline; \( P = .20 \)). There was no statistically significant difference in the percent change in tumor area between itraconazole and placebo at 4 weeks (0.04% vs −10.9% from baseline; \( P = .40 \)) and 12 weeks (8.9% vs 26.5%; \( P = .40 \)). In post hoc analysis, the itraconazole group showed reduced BCC tumor area in the subgroup of BCCs located on the back; however, given multiple testing for different anatomic locations, this difference was ultimately not statistically significant. Using liquid chromatography–mass spectrometry, intratumor itraconazole concentration was 133 μg/g of skin at 4 weeks and 96 μg/g at 12 weeks. There was no association between change in BCC tumor area and GLII mRNA levels. Other major metabolites such as hydroxy-itraconazole were not measured given their weaker half maximal inhibitory concentrations (IC50).

One patient had grade 1 liver function test abnormalities at weeks 4 and 12, but this patient had fatty liver disease. Plasma itraconazole levels were undetectable after 4 and 12 weeks, except for 1 patient with a plasma level of 4.12 ng/mL. Topical itraconazole caused only grade 1 to 2 adverse effects: application site reaction (\( n = 4 \)), pruritus (\( n = 4 \)), lesion pain (\( n = 3 \)), dysgeusia (\( n = 1 \)), and xerosis (\( n = 1 \)). These adverse effects resolved by the end of the study except in 2 patients who had persistent mild lesion pain, pruritus, and xerosis.

**Discussion** | Itraconazole, 0.7%, gel appears safe, is associated with intratumor drug concentrations after 4 weeks, and is not associated with systemic absorption. However, topical itraconazole failed to reduce GLII mRNA levels and tumor area. Topical and oral itraconazole are associated with BCC shrinkage in mice, but topical penetration in humans is more difficult owing to a thicker epidermis.

**Conclusions** | Itraconazole gel at the maximally soluble formulation of 0.7% did not reduce GLII mRNA levels and BCC tumor size. However, this study does not rule out whether other formulations of itraconazole at higher concentrations may be more effective.

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**Distinct Histopathologic Patterns of Finger Eruptions in Dermatomyositis Based on Myositis-Specific Autoantibody Profiles**

A number of myositis-specific autoantibodies have been identified in patients with dermatomyositis (DM), including anti-aminocyanyl-transfer RNA synthetase (ARS), antimelanoma differentiation-associated protein 5 (MDA5), and antinuclear antibodies, each of which is characterized by a cutaneous manifestation.1-2 We analyzed the histologic findings of finger lesions based on these 3 myositis-specific autoantibodies.

**Supplemental content**

**Methods** | This retrospective observational study was performed on patients with DM diagnosed with typical rash and the presence of anti-ARS, anti-MDA5, and TIF1γ antibodies detected using enzyme-linked immunosorbent assay kits (Medical and Biological Laboratories) in our dermatology departments from September 2007 to August 2018. We found 74 cases (30, 19, and 25 cases in the ARS, MDA5, and TIF1γ groups, respectively) where patients underwent skin biopsies of finger eruptions (eTable in the Supplement). The medical ethics review committee of each hospital exempted this study from ethical approval and waived the need for patient written informed consent because all data used were...
deidentified. Hematoxylin-eosin-stained skin specimens were classified into classic cutaneous histopathologic classifications, including interface dermatitis, psoriasiform dermatitis, eczematous reaction, and vascular injury (eFigure in the Supplement).

Immunohistochemical staining with antibody targeting myxovirus resistance protein A (MxA, sc-166412 by Santa Cruz Biology) was performed. Data were analyzed by the the χ² and Kruskal-Wallis tests using Prism 8 (version 8.1.0, GraphPad Software). P values <.05 were deemed statistically significant. The analyses were performed from September to December in 2018, then additional statistical analyses using Prism were performed in April 2019.

Results | Interface dermatitis was observed in more than half of the specimens from each group. Psoriasiform dermatitis was observed at a significantly higher frequency in the ARS group (17 cases, 57%) than in the MDA5 group (1 case, 5%) and TIF1γ group (1 case, 4%) (Figure, A). Patients in ARS group (18 cases, 60%) developed eczematous reaction at a significantly higher frequency than did the MDA5 (3 cases, 16%) and TIF1γ groups (1 case, 4%). Vascular injury was observed more often in the MDA5 group (14 cases, 74%) than in the ARS (13 cases, 43%) and TIF1γ groups (11 cases, 44%). These results were not significantly related to the finger location of skin biopsies.

The remains (the scores for dyskeratotic cells minus the scores for liquefaction degeneration of the basal layer) in the ARS group (median, 0.5; interquartile range [IQR], 0-1.7) were significantly higher than those in the MDA5 (median, 0; IQR, −0.5 to 0) and TIF1γ groups (median, 0; IQR, −1 to 0) (Figure, B). The expression of MxA, a type 1 interferon (IFN)-inducible protein, in the epidermal keratinocytes was significantly higher in the MDA5 (index median, 4; IQR, 4-4) than in the TIF1γ group (index median, 2; IQR, 1-3), and this expression was also rarely observed in the ARS group (index median, 0; 0-1) (Figure, C).

Discussion | To our knowledge, this study is the first histologic analysis of cutaneous manifestations to demonstrate that skin eruptions can be histopathologically classified into myositis-specific autoantibodies-associated groups, suggesting the systemic pathologies of the different types of antibody-associated DM, as shown in the eFigure in the Supplement. Anti-ARS antibody-associated DM is an independent subset characterized by a mixture of psoriasiform dermatitis and eczematous reaction with dyskeratotic cell-rich interface dermatitis. In addition, the eruption related to anti-MDA5 antibody-associated DM was characterized by vascular injury. We also found that MxA expression on epidermal keratinocytes was absent in the ARS group,
which is similar to findings from a previous study examining muscle fibers.4 In addition, high MxA epidermal expression was observed especially in the MDA5 group, as suggested previously,5 and also in the TIF1γ group.

Because the findings of this study were limited by the small number of Japanese patients, further large-scale studies are required to clarify the pathologies of DM.

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**Morphologic Switch From Psoriasiform to Eczematous Dermatitis After Anti-IL-17 Therapy: A Case Series**

The immunopathogenic role of type 17 T helper (Th17) cells in psoriasis has led to targeted therapies such as secukinumab, a human monoclonal antibody of interleukin 17A (IL-17A). We present 4 cases of eczema associated with secukinumab therapy for psoriasis.

**Report of Cases**
A woman in her 30s presented with psoriasis vulgaris with an inadequate response to phototherapy or to treatment with methotrexate, adalimumab, or ustekinumab. A Psoriasis Area and Severity Index (PASI) 100 (complete clearance) was achieved (baseline PASI, 19.4) after 3 months of secukinumab therapy. Concurrently, she developed flexural eczema on the wrists and antecubital and popliteal fossa. Treatment with potent topical steroids was followed by control, and secukinumab treatment was continued for a further year. Treatment was changed to cyclosporine for 18 months owing to pregnancy, during which her eczema resolved. Secukinumab treatment was reintroduced 8 weeks postpartum, followed by continued psoriasis control. Eczema again recurred on flexural sites within 8 weeks. Application of potent topical steroids was followed by control, and secukinumab treatment was continued. Three additional cases are summarized in the Table.

**Discussion**
We present a case series documenting the potential for secukinumab to induce eczematous dermatitis. Pertinent features that suggest a drug-induced adverse effect include the temporal relationship, resolution following treatment withdrawal, recurrence on rechallenge in case 1, and biological plausibility. Development of eczematous dermatitis coincided with the clearance of psoriasis. Patient 2 also developed eczematous dermatitis following both ustekinumab and secukinumab treatments (Figure). Interestingly, concurrent with the development of secondary failure of ustekinumab, her eczematous dermatitis resolved. The similar eczematous dermatitis following treatment with secukinumab in patient 2 provides evidence that this cutaneous adverse effect is specific to the immunopathogenesis of eczema, rather than a consequence of the molecular structure of either secukinumab or ustekinumab.

The coexistence of psoriasis and eczema in a single patient is uncommon given the distinct and opposing immune mechanisms. However, recent reports of the 2 conditions in the same patient have shown that specific T cells migrate into the skin and determine the outcome of inflammatory dermatoses, either Th2 cells for eczema or Th17 cells for psoriasis.3

Tumor necrosis factor (TNF)-inhibitors’ abilities to induce eczema have been well documented.2 Similarly, case reports have...