In conclusion, *E pasteuriana* can infect immunocompromised patients. There is potential risk that more Asian cases may emerge. Further investigation of *E pasteuriana* geographic distribution and reservoirs would reveal more information.

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A Case of Dominant Dystrophic Epidermolysis Bullosa Responding Well to an Old Medication

Epidermolysis bullosa (EB) is an inherited disease characterized by fragile skin and bullae or erosion formation, either spontaneously or on minor skin trauma. Molecular defects within the epidermis or the skin’s basement membrane indicate which subtype of EB a patient has. Collagen VII is affected in dystrophic EB.1,2 The mainstay of disease management involves wound care, symptom palliation, and prevention or treatment of complications. However, there is still no consistently effective treatment that minimizes or prevents formation of bullae on minor skin trauma.

Report of a Case | A 58-year-old man with a history of biopsy-diagnosed dominant dystrophic EB since childhood presented to the dermatology clinic with a 6- to 8-month history of increasing spontaneous bullae formation. Clinical manifestations were primarily cutaneous, acral in distribution, and affecting mainly his hands, legs, feet, and nails. His eyes, oral cavity, gastrointestinal tract, and genitourinary tract were not involved. Family history revealed that his father and older brother were also affected, and there was no history of consanguinity. Patient was treated with oral minocycline, 100 mg, twice daily.

At a 6-week follow-up appointment, the erosions were healing, and he had not had any new bullae formation. At a subsequent 6-week follow-up appointment, the patient reported...
that a few new bullae had formed but with substantially less frequency than before treatment with minocycline was started. Owing to his relative improvement, his minocycline dose was decreased to 100 mg daily.

At a subsequent 8-week follow-up, the patient reported a marked reduction in spontaneous bullae formation, with no new bullae for the 2 weeks prior to the appointment. Four months later, he reported that he had not had any bullae formation for the preceding 2 months and an overall 90% reduction in spontaneous bullae formation since starting minocycline treatment, showing no difference in effect between the twice-daily and daily dosing regimens.

One year after his initial presentation, the patient self-discontinued his minocycline regimen after marked clinical improvement. Within 1 month, he began spontaneously developing 3 to 5 new bullae per week and presented again in follow-up to our dermatology clinic. His original minocycline regimen, 100 mg, twice daily was restarted. The frequency of the patient’s bullae formation subsequently decreased within the first 4 weeks of minocycline treatment reinitiation. At a subsequent 6-week follow-up appointment, he had clinically apparent healing of his previous erosions with no active bullae or erosions.

Discussion | Patients with dystrophic EB have been found to have increased levels of matrix metalloproteinase-9 (MMP-9), as reported by Lettner and colleagues. Golub and colleagues reported that along with its anti-inflammatory properties, minocycline is also an MMP inhibitor. Thus, the effectiveness of minocycline in reducing the formation of bullae may be due to a reduction in MMP activity and a subsequent stabilization of the skin’s basement membrane zone. This theory is further supported by Bauer and colleagues in their work showing clinical improvement of patients with recessive dystrophic EB under treatment with phenytoin and the effects of phenytoin on decreasing collagenase activity.

We conducted a literature review using PubMed, MEDLINE via OVID (from 1946 to the present), EMBASE, and The Cochrane Library and found 2 other cases of dystrophic EB, reported by White in 1989, in which bullae formation was successfully reduced with minocycline. To our knowledge, the present case report details the first documented successful treatment of dystrophic EB using minocycline since 1989. Perhaps this old medication can be revisited as a potential treatment option for patients with dystrophic EB.

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COMMENT & RESPONSE

Concerns About Presence of a Wild-Type BAP1 Allele in Absence of Nuclear Protein Expression

To the Editor | As fellow researchers investigating the recently described BAPI tumor predisposition syndrome (OMIM 614327), we read with great interest the recent report “Multiple Cutaneous Melanomas and Clinically Atypical Moles in a Patient With a Novel Germline BAPI Mutation” by Gerami et al.1 The evidence of dysplastic nevus syndrome in the presence of germline BAPI mutations could prove indeed very useful in the clinical setting.

The authors also reported that all the melanomas from the proband did not show any BAPI nuclear staining in tumor cells—a finding suggestive of loss of heterozygosity, and expected for a tumor suppressor gene. However, at least 1 of the melanomas still showed a wild-type BAPI allele on the electropherogram. Absence of wild-type protein expression in the presence of a wild-type allele seen on electropherogram could be a mere artifact—due to contaminating DNA from stromal cells surrounding the malignant ones—or real, possibly from epigenetic inactivation of the wild-type BAPI allele. In our group’s recent investigation of sporadic malignant mesotheliomas,2 we did not detect any changes in the methylation status of the BAPI gene, and loss of BAPI nuclear immunohistochemical staining was always paralleled by partial or total genomic alterations of the BAPI gene.

We believe it would therefore be of exceptional relevance to the scientific community if this finding reported by Gerami et al were to be true. We would be grateful if the authors could clarify this intriguing point, providing more details on whether the DNA was extracted specifically from tumor cells after laser microdissection—therefore reducing the risk of an artifact—and, if that were to be the case, whether analysis of the methylation status of the BAPI gene could be performed. Otherwise, it may be helpful if the authors noted that this unusual finding was likely caused by contaminating stromal cells.

Finally, while we agree with the authors on the important roles, in the presence of germline BAPI mutations, of strict sun protection and regular dermatologic and ophthalmologic evaluations for the early diagnosis of potentially curable melanomas,2 we would also like to extend the list of prescrip-