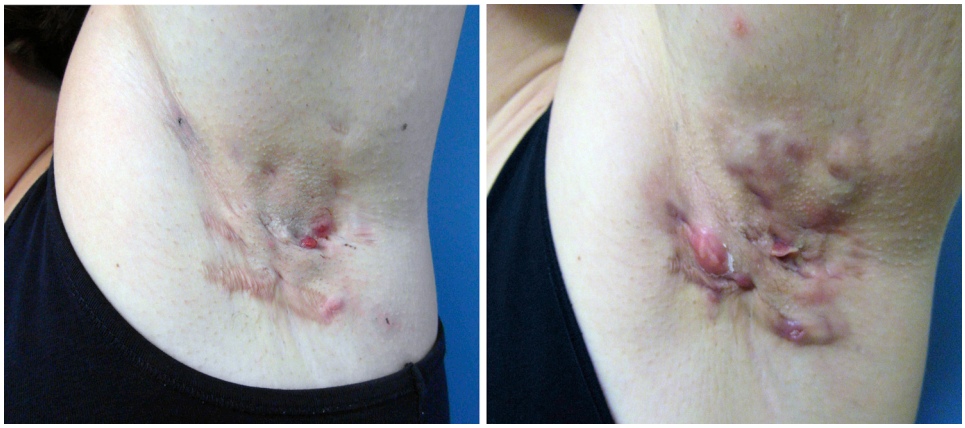

Fig 1. Baseline condition (*left*) and 3-month response (*right*) to miraDry treatment in the left axilla of patient 5 with hidradenitis suppurativa.

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Port-wine stains on the neck respond better to a pulsed dye laser than lesions on the face: An intrapatient comparison study with histopathology

To the Editor: Neck port-wine stains (PWSs) usually respond better than facial PWSs to treatment with a pulsed dye laser (PDL).[1] Until now, however, self-controlled studies with objective evaluation of efficacy and anatomic features have not been reported. In this study, we investigated the morphologic and anatomic features of both facial and neck PWS lesions and put forward a possible explanation for the difference in the therapeutic efficacy of PDL.

A total of 26 patients with untreated facial and neck PWSs received similar numbers (range, 2-3) of PDL treatments with a Vbeam laser (Candela Corporation, Boston, MA), with a radiant exposure of 12 J/cm^2, 1.5-ms pulse duration, 7-mm spot size, and cryogen spray cooling for 40 ms with a 20-ms delay on both sites. Clinical efficacy was evaluated by using a CR-400 chromameter (Minolta, Tokyo, Japan). Two months after the final treatment, the L*, a*, and b* color system of the International Commission on Illumination[2] were monitored, including the values of ΔE (color expressed change or improvement), Δa* (change in the vascular erythema), and blanching rate.[3] Additionally, pretreatment biopsies of both facial and neck PWS lesions were obtained from 9 patients. The PWS vascular diameter and depth and thickness of the epidermis were measured by using medical ImagePro Plus image analysis software (Media Cybernetics Inc, Rockville, MD).

After PDL treatments on both sides, the neck PWS lesions showed significantly better clearance than

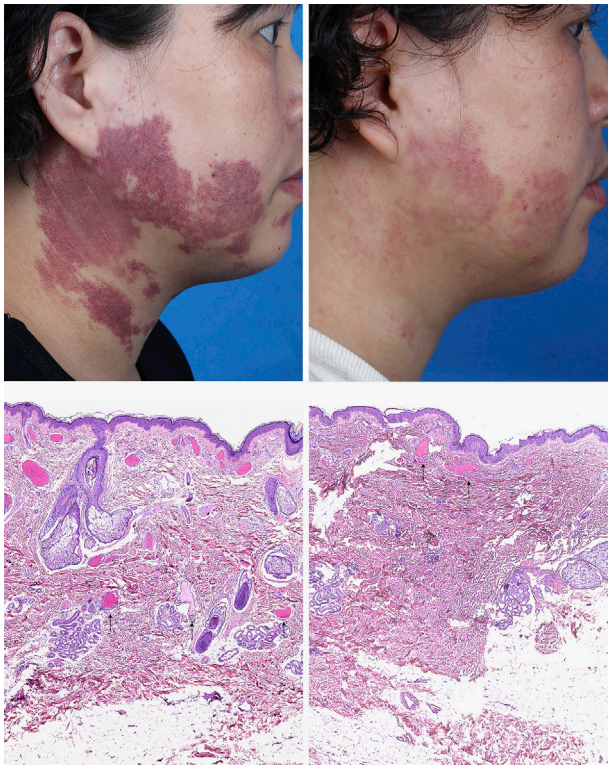

Fig 1. Patients treated with 3 sessions of pulsed dye laser treatment of both neck and facial port-wine stain (PWS) lesions were followed up 2 months after the final treatment. The results of the follow-up revealed that the response of the neck PWS lesions to the pulsed dye laser treatments was better than that of the facial PWS lesions (*upper left and right*). The histologic vascular features show that the ectatic vessels in the face were widely distributed from the papillary dermis to the reticular dermis (*lower left*) and that the ectatic capillaries were primarily distributed in the papillary dermis in neck PWSs (*lower right*). The epidermis was also thinner in neck PWSs than in facial PWSs. (Hematoxylin-eosin stain; original magnification: ×40.)

did the facial PWS lesions according to Δa* (5.66 vs 3.22 [$P = .001$]), ΔE (10.97 vs 7.45 [$P < .001$]), and blanching rate (59.9% vs 37.5% [$P < .001$]) (Fig 1).

The vessels in the neck PWS lesions were significantly more superficial (0.154 vs 0.248 mm [$P = .038$]) than those in the facial PWS lesions, and the epidermis at the neck sites was also thinner than that at the facial sites (0.018 vs 0.022 mm, respectively [$P < .01$]). A negative correlation existed between the clinical efficacy and the vascular depth ($r = -0.578$ [$P < .05$]) and the thickness of the epidermis ($r = -0.461$ [$P < .05$]) (Fig 2).

Our findings indicate that the superior efficacy of the PDL treatments in neck PWSs may result from the more superficial distribution of vessels. These results

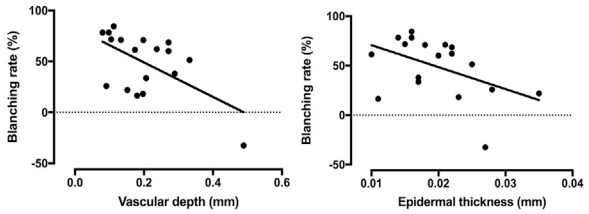

Fig 2. The Pearson correlation coefficient of vascular depth and thickness of the epidermis, which suggests that the vascular depth (*left*) and thickness of the epidermis (*right*) were negatively related to the efficacy outcome of the pulsed dye laser treatment.

were consistent with those of an earlier subjective study[4] indicating that it was easier to coagulate the more superficial distribution of vessels.

In a study by Ackermann et al,[1] a correlation between the therapeutic outcome of laser therapy and penetration of light into skin was found by investigating the respective anatomic sites. Theoretically, the increase in the thickness of the epidermis may thus lead to an increase in scattering and absorption of laser light, resulting in poorer efficacy in ameliorating facial PWS lesions.

In conclusion, the histologic manifestations in PWS lesions and their relationship with the efficacy of PDL treatment in neck and facial PWS lesions shows that the variation in both the epidermal thickness and the depth of the PWS lesion vessels may be responsible for the difference in therapeutic outcomes in the same patient.

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Ustekinumab treatment for neutrophilic dermatoses associated with Crohn's disease: A multicenter retrospective study

To the Editor: Systemic corticosteroids and tumor necrosis factor-alpha (TNF-α) antagonists are first-line therapies for neutrophilic dermatoses (NDs) associated with Crohn's disease (CD), the most frequent being pyoderma gangrenosum (PG).[1] Ustekinumab, a monoclonal antibody directed against the interleukin 12/interleukin 23 p40 subunit, was recently approved for use in CD.[2] This multicenter retrospective study conducted in 9 French hospitals between January 2013 and July 2017 assessed the efficacy of ustekinumab in CD-associated ND. Consecutive patients referred for active CD-associated ND and treated with ustekinumab following ND onset were included. Diagnosis of ND was based on previously published diagnostic criteria for NDs[3] and confirmed by skin biopsy. The efficacy of ustekinumab was assessed at week 16 after initiation: efficacy in relation to ND was evaluated by determining whether it resulted in a complete, partial (a >50% reduction in symptoms), or no response, and efficacy in relation to CD was evaluated by using the Harvey-Bradshaw index.

The study included 7 patients from among the 320 of 8298 patients with CD who received ustekinumab; the 7 patients included 5 women, 4 patients with PG, 2 with amicrobial pustulosis of the folds (APF), and 1 with chronic recurring Sweet syndrome (Table I). The result of microbiologic skin sampling was negative except in the case of patient 6, who had staphylococcal superinfection, as is frequently reported in APF.[3] The median ages at onset of ND and CD were 28 years (range, 6-43] and 22 years (range, 13-38), respectively. The median durations of ND and CD at initiation of ustekinumab therapy were 4 and 8 years, respectively. Of the 7 patients studied, 4 had active CD at initiation of ustekinumab therapy; 6 of the 7 patients had previously received TNF-α antagonists that were discontinued because of side effects or inefficacy. A history of lupus erythematosus contraindicated in patient 5. Patients received ustekinumab, 90 mg every 8 weeks subcutaneously, with slightly varying induction protocols for both CD and ND treatment (Table I).

At week 16, although most NDs were refractory to multiple therapies and ustekinumab was used as the third to eighth line of treatment, ustekinumab induced remission of ND in 6 of 7 cases (86%) (Table I): complete response in 4 of 7 cases (57%), partial response in 2 of 7 cases (29%), and no response in 1 case (patient 7 with refractory recurring Sweet syndrome). At week 16, the CD of 4 patients who had active CD on day 0 was in complete remission, 2 patients had stable CD with prolonged remission, and 1 patient had a worsening of CD activity that had previously been controlled by adalimumab. No serious side effects were reported. The median follow-up time was 36 months. One patient with ND eventually relapsed (patient 2). A literature review yielded 12 additional cases of ND that had been successfully treated with ustekinumab; of the total of 13 patients (11 with PG and 2 with APF), 9 (69%) were complete responders[4,5] and 3 (23%) were partial responders (Table II[6-13]). The limitations of our study are linked to its retrospective nature. Open labeled or randomized studies are needed to confirm these results. Associated immunosuppressants were sometimes required because of the severity of the ND (in patients 3 and 5); however, ustekinumab allowed tapering and/or discontinuation of the associated therapies.

Ustekinumab may control both ND and CD. It should therefore be considered as alternative therapy for refractory ND in cases of resistance, intolerance, or contraindication to corticosteroids or TNF-α antagonists.

We thank the members of the Ustekinumab–Crohn's Disease–Neutrophilic Dermatosis Study Group (Benoit Coffin [Colombes], Benjamin Pariente [Lille], and Vered Abitbol, Marion Simon, and Elise Chantelou [Paris]) for their prompt response to our request.