Table II. Factors affecting reporting of most recent sharps injury to occupational health service

Variable	Reporting N/ total N	% Reporting	Reference group reporting/total N	% Reporting	Odds Ratio of reporting (CI)	*P* value
Female resident	126/180	70%	51/87	59%	1.65 (0.97-2.81)	.066
High-risk patient Injury (ever)	39/50	78%	137/216	63%	2.04 (0.99-4.2)	.052
High-risk patient Injury (most recent)	30/32	94%	146/227	64%	8.32 (1.9-35.7)	.004*
Self-induced	151/234	65%	25/32	78%	0.51 (0.21-1.22)	.133
Needle sharp	141/216	65%	34/48	71%	0.77 (0.39-1.5)	.471
Inpatient consult	12/16	75%	164/250	66%	1.57 (0.49-5.0)	.453
Ambulatory room	70/119	64%	107/158	68%	0.86 (0.51-1.43)	.563
Procedural room	88/130	68%	89/137	65%	1.13 (0.68-1.8)	.650
Medical Dermatology Procedure	34/54	63%	143/213	67%	0.83 (0.45-1.55)	.574
Surgical Dermatology Procedure	140/205	68%	37/62	60%	1.45 (0.81-2.6)	.211
Cosmetic Dermatology Procedure	2/7	29%	175/260	67%	0.19 (0.03-1.02)	.053

*Statistically significant with $P < .05$.

patient care. Current Accreditation Council for Graduate Medical Education dermatology milestones require residents to "recognize the reasons for protocol-driven procedural safety" and emphasize the need for residents to contribute to program improvement.[5] Sharps safety should be an important area for such future improvement efforts.

Portions of the results were presented at the 2014 Residents and Fellows Symposium of the Annual Meeting of the American Academy of Dermatology, Denver, Colorado.

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Does biopsy accurately assess basal cell carcinoma (BCC) subtype?

To the Editor: The treatment of choice for basal cell carcinoma (BCC) is predominantly based on the histologic subtype, location, and tumor size.[1,2]

Previous studies have reported discordance between subtypes identified by biopsy and subtypes identified in the excision specimen, with subsequent aggressive subtypes in the excision specimen not recorded in the initial biopsy specimen in 11% to 21% of cases. Previous studies were retrospective chart/database studies with different dermatopathologists having judged the biopsy and excision specimens without systematic histopathological review of both all biopsy and final excision specimen slides.[3-5]

We conducted a prospective study to evaluate the adequacy of BCC subtyping by punch biopsy in relation to the Mohs excision specimens and to evaluate interobserver variation. Patients were included if a biopsy specimen was obtained before Mohs micrographic surgery. None of the tumors were curetted or debulked before the Mohs excision. The cases consisted of 149 primary, 21 recurrent, and 19 incomplete excised tumors. From the frozen Mohs specimen serial sections of 4 μm (with intervals of

Table I. Frequency table of subtyping aggressive and nonaggressive basal cell carcinoma subtype in biopsy and relating Mohs specimen for both dermatopathologists with subsequent classification tests

	DP 1	DP 2	DP 1	DP 2
	Aggressive subtype Mohs		Nonaggressive subtype Mohs	
Aggressive subtype biopsy	99	103	28	19
Nonaggressive subtype biopsy	25	22	31	38
Mean classification tests [(DP1 + DP2)/2]	Sensitivity (79.8 + 82.4)/2 = 81.1% Specificity (52.5 + 66.7)/2 = 59.6% False positive rate (47.5 + 33.3)/2 = 40.4% False negative rate (20.2 + 17.6)/2 = 18.9%			

DP, Dermatopathologist.

Table II. Kappa cross-table for determining kappa interobserver variability between the dermatopathologists in both biopsy and Mohs specimens

		Biopsy specimens		Mohs specimens	
		DP 2		DP 2	
		Aggressive BCC subtype	Nonaggressive BCC subtype	Aggressive BCC subtype	Nonaggressive BCC subtype
DP 1	Aggressive BCC subtype	118	14	113	11
DP 1	Nonaggressive BCC subtype	10	46	12	46
		κ = 0.701		κ = 0.708	

BCC, Basal cell carcinoma; *DP*, dermatopathologist.

100 μm) were cut through the whole tissue block, thereby encompassing the whole tumor for observation. Both biopsy and Mohs specimens were stained with hematoxylin-eosin.

All slides were individually scored for tumor subtype(s) in an independent and blinded fashion by 2 dermatopathologists. Subtypes were defined as nonaggressive (superficial/nodular) or aggressive (infiltrative/micronodular/morpheiform/metatypical). Mohs slides were considered as reference, because they allowed an overall view of the tumor. In all, 188 cases were included. Baseline characteristics showed that 54.3% of patients were female. Most BCCs were located on the nose (41.0%). Outcomes of subtyping and the results of the classification tests are shown in Table I.

Most important, in almost one fifth of the cases, the biopsy specimen showed only a nonaggressive subtype, whereas an aggressive subtype was observed in Mohs specimen. A logical explanation is that the whole tumor is observed in the Mohs specimen, whereas a biopsy specimen represents only a small part of the tumor. Another possible explanation could be the fact that an aggressive subtype cannot always be judged properly in a biopsy specimen because of the limited size, and could therefore be underreported. Alternatively, in 40.4% of cases an aggressive subtype was observed in the biopsy specimens but not in the Mohs specimens.

To test which factors influenced agreement between biopsy specimen and Mohs micrographic surgery specimen, a generalized estimating equation model was used. There was no difference between primary and recurrent tumor, but both showed significantly more concordance than incomplete excised tumors. Tumor location or size were not significantly correlated.

The calculated Cohen kappa score was 0.701 for biopsy specimens and 0.708 for the Mohs micrographic surgery specimens, indicating a substantial agreement between both dermatopathologists (Table II).

The strength of our study lies in the separate and blinded systematic review of both the original biopsy specimen slides and the Mohs micrographic surgery slides by 2 individual dermatopathologists.

This report underlines that biopsy specimens may not identify all growth patterns of a BCC, that an aggressive component may be absent or fail to be observed, and that subtyping is subject to interobserver variation.

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Surgical treatment of pyoderma gangrenosum with negative pressure wound therapy and split thickness skin grafting under adequate immunosuppression is a valuable treatment option: Case series of 15 patients

To the Editor: Pyoderma gangrenosum (PG) is a rare inflammatory disease of unknown etiology. Only about 50% of patients achieve remission after 6 months of immunosuppressive therapy and even when patients respond well to treatment, relapses can occur in 30% to 60% of cases.[1-3] Progression of existing lesions or development of new lesions after trauma is reported in up to 30% of PG (pathergy phenomenon).[3] Therefore the role of surgical interventions is controversially discussed in that they might further aggravate the condition, especially if performed without immunosuppression.[2] On the other hand, PG ulcers without skin grafting require a prolonged time to heal, being prone to secondary infection, which potentially represents an additional trigger for pathergy. In addition, long-term systemic immunosuppressive therapy is associated with adverse reactions in about 65% of patients.[2,3] Therefore an urgent clinical need exists to close open wounds in these patients as fast as possible. Negative pressure wound therapy (NPWT) has become an important tool in the management of complex wounds and is used to secure split thickness skin grafts (STSG) in difficult recipient wound beds.[4] Here we report a multicenter case series of 15 patients with PG who were treated successfully with surgery under adequate immunosuppression, with STSG secured by NPWT after effective conditioning with NPWT (Fig 1).

Patient details, treatment, and outcomes are reported in Table I. All patients were hospitalized before surgical intervention to initiate immunomodulatory therapy. NPWT (VAC) was begun up to 1 week before surgical intervention. All interventions were performed under local tumescence anesthesia as previously described.[5] STSGs were secured by NPWT for 3 to 5 days. Ten patients healed completely and primary healing without recurrence after the first treatment cycle occurred in 9 of those patients, while patient 5 healed after 2 recurrences. One local recurrence healed after modification of the immunosuppressive treatment with NPWT alone and a second PG at a different localization healed after a second cycle of NPWT followed by STSG, secured by NPWT. In 3 patients, there was marked improvement of more than 90% of the wound surface healed; minor recurrence developed in 2 of those patients, which was managed with local wound dressings and increased immunosuppression, resulting in 95% improvement seen at follow-up. Two patients still have ulcers: ulcers healed in patient 4 but recurred twice after successful treatment cycles with NPWT followed by STSG secured by NPWT. Patient 11 never improved more than 30% despite treatment. No pathergy or reactivation of PG was observed, neither in the vicinity of the PG ulcers nor at the skin graft donor sites. NPWT followed by STSG secured by NPWT induced healing within 1 month after grafting in 67% (10/15) of cases and improvement of more than 90% in 4 (27%) cases, being significantly superior to the reported healing rates of 50% after 6 months when a conservative approach is performed while showing a similar recurrence rate of 30%.[1-3]

This large case series of surgical intervention in PG confirms that wound preparation with NPWT and