annual testing for LTBI only among high-risk patients (e.g., patients who live, work, or travel where TB exposure is likely).[4] Given that the estimated total medical expenditure among US employer-based privately insured individuals was $52.6 million in 1 year for the 3 most common TB tests,[5] significant cost savings may result by limiting testing to high-risk individuals and avoiding unnecessary tests.

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Practice habits of Mohs surgeons treating melanoma with Mohs surgery: A cross-sectional survey

To the Editor: Mohs micrographic surgery (MMS) is increasingly used to treat melanoma in the United States.[1] Recent studies have shown lower recurrence rates and improved survival outcomes for melanoma treated with MMS compared to wide local excision.[2,3] Given the variations in practice patterns of MMS for melanoma combined with a lack of best practice guidelines,[1] this survey of Mohs surgeons sought to elucidate the practice habits used by Mohs surgeons who treat in situ and invasive melanoma with MMS.

This study was a nationwide cross-sectional survey of Mohs surgeons with membership in the American College of Mohs Surgery (ACMS). An anonymous survey was developed and distributed to all ACMS members (N = 1630) during March 2020 through May 2020 by using ACMS mailing lists. MMS was defined as Mohs surgery with intraoperative

Table I. Practice habits of Mohs surgeons treating melanoma with Mohs surgery (N = 363)

Characteristic	Mohs surgeons treating melanoma with MMS, n (%)
Subtypes treated*	
LM	348 (95.9)
MIS (subtypes excluding LM)	332 (91.5)
T1	216 (59.5)
T2 and/or greater	76 (20.9)
Anatomic location	
Head and neck	360 (99.2)
Trunk and extremities (excluding hands and feet)	174 (47.9)
Hands, feet, genitalia	259 (71.3)
Number of cases treated with MMS per year	
≤40	204 (56.2)
41-120	118 (32.5)
121-200	21 (5.8)
>200	20 (5.5)
IHC staining	
Yes	273 (75.2)
No	90 (24.8)
IHC stains used	
MART-1	261 (95.6)
MITF	11 (4.0)
S-100	11 (4.0)
HMB-45	3 (1.1)

IHC, Immunohistochemical; *LM,* lentigo maligna; *MIS,* melanoma in situ; *MMS,* Mohs micrographic surgery.
*Based on American Joint Committee on Cancer, 8th Edition staging criteria.

Table II. Melanoma tumor processing during Mohs surgery

Characteristics	Mohs surgeons treating melanoma subtypes* with MMS, n (%)			*P* value†
	MIS (n = 363)	T1 (n = 216)	T≥2 (n = 76)	
Debulk				.005
Yes	310 (85.4)	199 (92.1)	73 (96.1)	
No	53 (14.6)	17 (7.9)	3 (3.9)	
Debulk technique				.94
Debulk with scalpel	305 (98.4)	212 (98.1)	76 (100.0)	
Debulk with curette	5 (1.6)	4 (1.9)	0 (0.0)	
Debulked specimen processing				.15
Permanent vertical sectioning	184 (60.3)	126 (59.4)	42 (55.3)	
Frozen vertical sectioning	31 (10.3)	11 (5.2)	3 (3.9)	
Both	87 (28.5)	72 (34.0)	29 (38.2)	
Discard	3 (0.9)	3 (1.4)	2 (2.6)	
Initial margins (debulk + 1st MMS stage), mm				<.001
Mean (SD)	4.99 (1.66)	6.60 (2.69)	7.52 (2.24)	
Depth of initial MMS stage				<.001
Deep dermis	17 (4.7)	3 (1.4)	2 (2.6)	
Subcutaneous fat	335 (92.3)	173 (80.1)	48 (63.2)	
Fascia	11 (3.0)	40 (18.5)	26 (34.2)	
Additional layer around negative MMS margin				<.001
No	305 (84.0)	195 (90.3)	76 (100.0)	
Yes, permanent analysis	56 (15.4)	13 (6.0)	0 (0.0)	
Yes, frozen analysis	0 (0.0)	0 (0.0)	0 (0.0)	
Yes, both permanent and frozen	2 (0.6)	8 (3.7)	0 (0.0)	

MIS, Melanoma in situ; *MMS,* Mohs micrographic surgery; *SD,* standard deviation.
*Based on American Joint Committee on Cancer, 8th Edition staging criteria.
†Comparisons between groups conducted by using chi-square, Fisher exact test, and 1-way analysis of variance.

frozen section analysis. Excisional surgery with en face permanent section analysis ("slow Mohs") was not considered as MMS. Fellowship exposure was defined as treating at least 1 melanoma case with MMS using frozen sections during fellowship. The survey inquired about melanoma subtypes treated (primary tumor [T], American Joint Committee on Cancer, 8th Edition), anatomic locations treated, number of melanoma cases treated with MMS per year, processing of debulked melanoma specimens, initial margin sizes (the total distance beyond the clinically apparent tumor after excision of the first MMS stage), depth of initial MMS stage, and immunohistochemical (IHC) staining. The study was approved by the institutional review board of the University of Kansas. Chi-square, Fisher exact tests, and a 1-way analysis of variance were used to compare differences between groups, with $P < .05$ considered statistically significant.

A total of 1002 participants completed the survey (61.5% response rate; average age, 46 ± 11 years; 632 [64.1%] male; 715 [71.4%] in private practice; 174 [17.4%] in the Midwest, 185 [18.5%] in the Northeast, 246 [24.6%] in the Southeast, 131 [13.1%] in the Southwest, 219 [21.9%] in the West, and 47 [4.7%] in other/unspecified location). Half (n = 499; 50.0%) of all participants received fellowship exposure to MMS for melanoma. Among the 363 participants (36.2% overall) treating melanoma with MMS (MMS-M), 216 (59.5%) treated invasive (T1) melanoma, and 76 (20.9%) treated invasive (T≥2) melanoma (Table I). MMS-M participants were more likely to debulk invasive melanoma compared to melanoma in situ (MIS) (93.2% T≥1 melanoma vs 85.4% MIS; $P = .002$). The average initial margin sizes for MIS, T1, and T≥2 melanomas were 4.99 mm, 6.60 mm, and 7.52 mm, respectively ($P < .001$) (Table II). MMS-M participants treating T≥1 melanoma were more likely to excise down to the fascia during the first Mohs stage compared to MIS (22.6% T≥1 melanoma vs 3.0% MIS; $P < .001$). MMS-M participants treating MIS were more likely to submit an additional layer around a negative MMS margin compared to T≥1 melanoma (16.0% MIS vs 7.2% T≥1 melanoma; $P < .001$). The majority (n = 273; 75.2%) of MMS-M participants use IHC stains with nearly all (n = 267; 97.8%) using melanoma antigen recognized by T cells 1 (MART-1) as their primary IHC stain.

To our knowledge, this is the largest study to date to investigate the practice habits of Mohs surgeons treating melanoma with MMS. The findings suggest that the majority of Mohs surgeons treating

melanoma with MMS are treating early invasive melanoma by using MART-1 staining. In addition, Mohs surgeons excise wider margins during the initial MMS layer when treating more invasive melanoma.

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Development of chronic cutaneous lupus erythematosus during biologic therapy: A systematic review

To the Editor: A rare cutaneous complication of biologic therapy is the onset of chronic cutaneous lupus erythematosus (CCLE). Because CCLE may increase the risk of progression to systemic lupus erythematosus,[1] it is important for physicians to recognize and promptly manage such adverse effects. The aim of this systematic review was to summarize reported cases of CCLE that developed after initiating biologic therapy for immune-mediated inflammatory diseases.

Embase and MEDLINE searches were conducted on July 26, 2020, in accordance with Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines by using variations of keywords "biologic" AND "CCLE" (Fig 1 and Supplemental Materials; available via Mendeley at http://doi.org/10.17632/y23f73nmdj.1). Studies describing concurrent systemic lupus erythematosus (SLE) and CCLE were excluded. Of the 13 studies that met the inclusion criteria, a total of 14 patients (mean age, 48.8 years) were included, of whom 71.4% (n = 10/14) were female (Table I). CCLE developed after treatment with tumor necrosis factor (TNF) α inhibitors in 85.7% of cases (n = 12/14) and IgG antibodies in 14.3% of cases (n = 2/14).

The latency period between biologic treatment and onset of CCLE ranged from 0.5 to 45 months (mean, 10.4 months); specifically, TNF-α inhibitors had a mean latency period of 11.5 months, and IgG antibodies had a mean latency period of 4.5 months. Of the 8 cases that reported improvement of CCLE after treatment, 62.5% (n = 5/8) had complete resolution, with a mean resolution period of 3.6 months, and 37.5% (n = 3/8) had partial resolution, with a mean resolution period of 2.0 months. All patients who achieved complete resolution discontinued biologics and treated CCLE with steroids and/or hydroxychloroquine. For patients with partial resolution, 66% (n = 2/3) did not discontinue biologics after CCLE development. However, they achieved partial resolution after treatment with steroids and/or immunosuppressive medication.

Although we found that the majority of CCLE cases occurred after initiating TNF-α inhibitors, the role of TNF-α in CCLE pathogenesis is unclear. One patient developed CCLE after initiating intravenous IgG. This may be due to the presence of autoantibodies, because high titers of anti-SSA (Sjogren's syndrome antigen A) autoantibodies have been found in intravenous IgG products from healthy donors.[2] Although studies describing SLE were excluded, there have been reports of patients developing SLE after biologic treatment.[3]

Additionally, chilblain lupus (CL) was reported in 33.3% (n = 4/12) of patients on TNF-α inhibitors. CL is a rare form of CCLE that may be caused by vasoconstriction or cold-induced microvascular injury.[4] Raynaud's phenomenon has a similar mechanism and may be associated with CCLE.[5] Despite sharing a similar pathophysiology to CL, Raynaud's phenomenon was not detected in these patients. Moreover, our findings suggest that discontinuing biologics and treating CCLE would be most effective in achieving resolution. Of those who had achieved CCLE resolution, 75.0% (n = 6/8) were treated with hydroxychloroquine which dampens the immune response.