Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Table I. Demographics of the asymptomatic study population

Characteristic	All patients, n = 1118
Age, mean (range), y	67.2 (15-99)
Sex, n (%)	
Male	662 (59)
Female	456 (41)
Ethnicity, n (%)	
White	1062 (95)
Hispanic	15 (1)
African American	1 (0)
Asian	9 (1)
Other	16 (1)
Declined	15 (1)

Table II. Demographics among the 14 asymptomatic patients with positive testing

Characteristic	Asymptomatic patients, n = 14
Age, mean (range), y	63.3 (21-95)
Sex, n (%)	
Male	6 (43)
Female	8 (57)
Ethnicity, n (%)	
White	13 (93)
Hispanic	1 (7)
African American	0 (0)
Asian	0 (0)

20,543 COVID-19 cases per 100,000 people since February 15, 2020, and an average COVID test positivity rate of 5.2% over the time period examined in this study.[5] Our data demonstrate a higher prevalence rate of asymptomatic infection when compared with the prevalence rate of 0.5% observed in a study examining elective endoscopy procedures in a similar study setting and a rate of 0% observed in 1 study examining both ambulatory and nonambulatory otolaryngologic procedures.[1,4] Though likely multifactorial, these differences could largely be explained by our longer study period and inclusion of the peak winter months in which most of our cases were diagnosed. Additional studies with larger sample sizes are needed to characterize better the regional prevalence of asymptomatic SARS-CoV-2 infection in the ambulatory periprocedural setting. Data derived from such studies could inform periprocedural protocols that best balance the safety of hospital staff and patients with the potential morbidity associated with delays in dermatologic surgery.

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Conflicts of interest

None disclosed.

REFERENCES

1. Bowyer B, Thukral C, Patel S, et al. Outcomes of symptom screening and universal COVID-19 reverse transcriptase polymerase chain reaction testing before endoscopy in a community-based ambulatory surgery center. *Gastrointest Endosc*. 2021;93(5):1060-1064.e1. https://doi.org/10.1016/j.gie.2020.10.001
2. Larsen CG, Bub CD, Schaffler BC, Walden T, Intravia JM. The impact of confirmed coronavirus disease 2019 (COVID-19) infection on ambulatory procedures and associated delays in care for asymptomatic patients. *Surgery*. 2021;169(6):1340-1345. https://doi.org/10.1016/j.surg.2021.01.005
3. Urban MJ, Patel TR, Raad R, et al. Implementation of preoperative screening protocols in otolaryngology during the COVID-19 pandemic. *Otolaryngol Head Neck Surg*. 2020;163(2):265-270. https://doi.org/10.1177/0194599820931041
4. Kavanagh FG, Callanan D, Connolly C, et al. Pre-operative testing for SARS-CoV-2 and outcomes in otolaryngology surgery during the pandemic: a multi-center experience. *Laryngoscope Investig Otolaryngol*. 2021;6(4):773-779. https://doi.org/10.1002/lio2.613
5. Cook County Department of Public Health. COVID-19 Surveillance Data. Accessed April 1, 2022. https://ccdphcd.shinyapps.io/covid19/

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Predictors of COVID-19 disease severity augment the Braden scale in the prediction of pressure ulcer development among COVID-19–positive intensive care unit patients: A case-control study

To the Editor: Pressure ulcer (PU) development among COVID-19–positive intensive care unit (ICU) patients is emerging as a unique, albeit incompletely understood, problem for health care

Table I. Univariable associations of admission attributes with pressure ulcer development among COVID-19–positive intensive care unit patients

Attribute*	Pressure ulcer = yes (n = 37)	Pressure ulcer = no (n = 111)	*P* value
Sex, n (%)			.776†
Male	20 (54)	57 (51)	
Female	17 (46)	54 (49)	
Age, median (IQR), y	64 (57-68)	65 (54-76)	.993‡
Body mass index, median (IQR), kg/m^2	32.6 (29.1-39.0)	30.2 (25.4-35.4)	.124‡
Vitals, median (IQR)			
Body temperature, °F	99.3 (97.9-101.0)	99.1 (98.2-100.7)	.736‡
Heart rate, per min	100 (87-112)	100 (84-116)	.409‡
Respiratory rate, per min	22 (18-28)	22 (18-26)	.180‡
SpO_2, %	90 (85-95)	94 (86-97)	.172‡
Initial oxygen support, n (%)			.260†
Nonmechanical ventilation§	34 (92)	105 (95)	.691‖
Mechanical ventilation	3 (8)	6 (5)	
Braden scale score, median (IQR)	16 (14-20)	19 (16-20)	.034‡
Admission laboratory data, median (IQR)			
C-reactive protein, mg/L	10.2 (6.2-16.2)	9.2 (4.0-15.2)	.182‡
Absolute lymphocyte count, 10^3/mm^3	0.9 (0.7-1.1)	0.9 (0.7-1.4)	.096‡
Interleukin 6, pg/mL	64.0 (32.8-115.6)	38.3 (10.3-105.2)	.067¶
White blood cell count, 10^3/mm^3	7.6 (6.3-10.3)	7.4 (6.2-9.7)	.866‡
Lymphocyte percentage, %	12.6 (8.3-15.8)	13.3 (8.4-18.8)	.205‡
Platelet count, 10^3/mm^3	200 (170-227)	200 (139-280)	.358‡
Triglycerides, mg/dL	152.0 (109.0-262.5)	141.0 (104.0-192.0)	.226¶
Blood urea nitrogen, mg/dL	25.0 (15.0-56.0)	22.0 (14.0-43.0)	.270‡
Creatinine, mg/dL	1.4 (1.0-2.2)	1.3 (0.9-2.6)	.772‡
Lactate dehydrogenase, U/L	428 (288-527)	375 (279-497)	.486‡
Ferritin, μg/L	578 (274-1178)	634 (298-1312)	.430¶
D-dimer, μg/mL	1.21 (0.74-4.57)	1.21 (0.57-3.21)	.345‡
Fibrinogen, mg/mL	4.4 (3.2-5.7)	5.0 (4.0-6.0)	.108‡
D-dimer >0.5 μg/mL and fibrinogen <2.0 mg/mL, N (%)#	5 (13.5)	2 (1.8)	.015‖

IQR, Interquartile range; *SpO_2*, oxygen saturation.

*All data in this column were collected for each patient within the first 24 hours of their admission to the hospital.

†χ^2 test.

‡*t* test.

§Room air, nasal cannula, high-flow nasal therapy, or bilevel positive airway pressure.

‖Fisher's exact test.

¶Wilcoxon rank-sum test.

#The value of this variable was defined as "true" for a patient only if they presented with both an elevated D-dimer level (>0.5 μg/mL) and a low fibrinogen level (<2.0 mg/mL).

systems.[1] Improvement in the risk stratification of COVID-19–positive patients with respect to PU development could allow for better patient outcomes, with more efficient health care utilization. Immobility, reduced tissue perfusion, hyperinflammation, and vasopressor requirement not only play pathogenic roles in PU development but are also salient characteristics of critically ill COVID-19–positive patients.[1-3] As such, we hypothesized that independent risk factors for PU development in COVID-19–positive ICU patients could be identified using patient data or laboratory data obtained within their first 24 hours of hospital admission that portend increased COVID-19 disease severity.

The medical charts of 606 COVID-19–positive patients admitted to Temple University Health System between March 17, 2020, and May 4, 2020, were analyzed. Patients who had no PU upon admission and required ICU-level care for >24 hours at any point during their admission were included in the study. A patient was determined to have a PU if they had wound care notes staging a wound as a PU, or if the wound had a description matching that of a PU at stage ≥1, and the etiology was not otherwise specified; the authors reviewed each chart to determine this.[3] A multivariable logistic regression model was built to identify independent risk factors for PU development using

Table II. Multivariable logistic regression analysis of relevant admission attributes while controlling for time spent in the intensive care unit

Attribute	Odds ratio (95% CI)	*P* value
D-dimer >0.5 μg/mL and fibrinogen <2.0 mg/mL	12.094 (1.860-78.647)	.0091
Braden scale score	0.872 (0.760-0.999)	.0486
Body mass index	1.062 (1.004-1.124)	.0353
C-reactive protein	1.048 (0.991-1.109)	.0997*
Hours spent in the ICU	1.007 (1.004-1.010)	<.0001

ICU, Intensive care unit.
*Although C-reactive protein did not achieve statistical significance ($P = .0997$), it was included in the model because its association with pressure ulcer development has been demonstrated.[3]

the patient data and laboratory data collected within the patients' first 24 hours of hospital admission. In addition, the number of hours each patient spent in the ICU was included in the model to control for this potential confounder.

Of the 606 COVID-19–positive patients, 148 met the inclusion criteria. Of the 148 patients, a PU developed in 37 patients. A univariable analysis helped determine the Braden scale score and "D-dimer >0.5 μg/mL and fibrinogen <2.0 mg/mL" (a binary variable intended to identify patients presenting with a late-stage, consumptive coagulopathy) as being significantly associated with PU development (Table I).[4] The construction of a multivariable logistic regression model identified ICU hours, body mass index, Braden scale score, and "D-dimer >0.5 μg/mL and fibrinogen <2.0 mg/mL" as statistically significant risk factors for PU development (Table II). Notably, PUs were 12.1 times more likely to develop in patients with a D-dimer level of >0.5 μg/mL and a fibrinogen level of <2.0 mg/mL upon admission than in other patients. The c-statistic representing the classification accuracy of the model was 0.874; thus, the model had excellent discrimination, according to a classification by Hosmer et al.[5] Furthermore, the likelihood-ratio test showed this model to offer statistically significant improvement compared with a 4-term model constructed without "D-dimer >0.5 μg/mL and fibrinogen <2.0 mg/mL" ($P = .005$), thus underscoring the predictive utility of this variable.

These results suggest that the predictors of COVID-19 disease severity, namely, consumptive D-dimer and fibrinogen levels as well as body mass index, can augment the Braden scale for the PU risk stratification of COVID-19–positive patients. Severe COVID-19 disease may directly participate in PU pathogenesis via microvascular thrombosis or may simply predispose patients to other risk factors such as vasopressor requirement. The precise mechanism by which these biomarkers are associated with PU development remains a subject of future investigation.

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Conflicts of interest

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REFERENCES

1. Tang J, Li B, Gong J, Li W, Yang J. Challenges in the management of critically ill COVID-19 patients with pressure ulcer. *Int Wound J*. 2020;17(5):1523-1524. https://doi.org/10.1111/iwj.13399
2. Gefen A, Ousey K. COVID-19: pressure ulcers, pain and the cytokine storm. *J Wound Care*. 2020;29(10):540-542. https://doi.org/10.12968/jowc.2020.29.10.540
3. Coleman S, Gorecki C, Nelson EA, et al. Patient risk factors for pressure ulcer development: systematic review. *Int J Nurs Stud*. 2013;50(7):974-1003. https://doi.org/10.1016/j.ijnurstu.2012.11.019
4. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *J Thromb Haemost*. 2020;18(4):844-847. https://doi.org/10.1111/jth.14768

5. Hosmer WD Jr, Lemeshow S, Sturdivant XR. *Applied Logistic Regression*. 3rd ed. Wiley; 2013.

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Psoriatic lesional expression of SARS-CoV-2 receptor ACE2 is reduced by blockade of IL-17 signaling but not by other biologic treatments

To the Editor: Psoriatic skin was recently described as a potential host site for SARS-CoV-2, the cause of COVID-19, due to the high expression of angiotensin-converting enzyme 2 (*ACE2*), which is the main viral host receptor in the epidermis of lesional skin.[1,2] There are positive correlations between *ACE2* expression, Psoriasis Area Severity Index scores, and interleukin 17C (*IL-17C*) expression at baseline,[3] whereas treatment with an anti-IL-17 antibody reduces the risk of COVID-19 in psoriatic patients by downregulating *ACE2* expression in affected skin.[3,4] However, the effect of other molecule-targeted therapies on *ACE2* expression in psoriatic skin remains unknown.

To determine this effect, we collected microarray data (GSE13355, GSE14905, GSE30999, GSE34248, GSE41662, GSE47751, GSE50790, GSE53552, GSE57376, GSE78097, GSE106992, GSE117239, GSE136757, GSE31652, and GSE137218) from the Gene Expression Omnibus database. The samples included normal skin (n = 89), psoriatic nonlesional skin (n = 456), and lesional skin (n = 502) at baseline. Samples were also obtained from lesional skin after treatment with placebo (n = 23), with 140 mg, 350 mg, or 700 mg brodalumab (n = 4, 4 or 8), with LY2439821 (n = 6), with secukinumab (n = 14), with etanercept (n = 60), with 45 mg or 90 mg ustekinumab (n = 18 or 55), with tofacitinib (n = 8), or with 30 mg or 100 mg PF-06700841 (n = 7 or 5) at the end time point. The data were analyzed with the Transcriptome Analysis Console software 4.0 (Applied Biosystems, Thermo Fisher Scientific). The least square means by group and fold change were calculated. Hypotheses were tested using 1-way analysis of variance with Tukey's test. $P < .05$ was considered statistically significant.

At baseline, *ACE2* expression was significantly upregulated in psoriatic lesional skin compared to nonlesional or normal skin (Fig 1), which is consistent with previous studies.[1,2] Interestingly, after treatment with placebo or the different molecule-targeted treatments, only the IL-17 receptor A subunit inhibitor (brodalumab) and anti-IL-17A monoclonal antibodies (LY2439821 and secukinumab), but not the other treatments, such as the tumor nuclear factor-α inhibitor (etanercept), anti-IL-12/-23 antibody (ustekinumab), Janus kinase inhibitor (tofacitinib), or tyrosine kinase 2 inhibitor (PF-06700841), remarkably reduced *ACE2* expression in psoriatic skin. Notably, although no significant difference in *ACE2* expression was observed between lesional skin at baseline and lesional skin after being treated with lower concentrations of brodalumab, dose-dependent *ACE2* expression was observed in the brodalumab-treated groups (Fig 1).

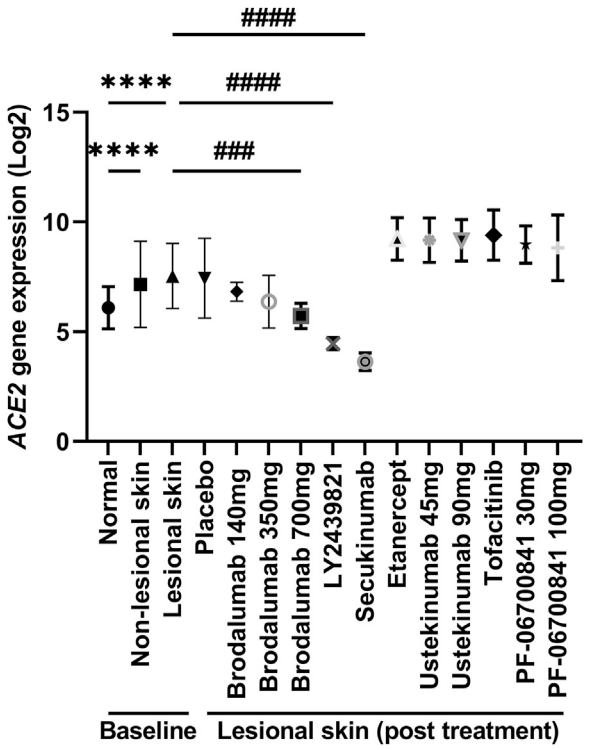

Fig 1. *ACE2* expression in normal, nonlesional, and lesional skin of patients with psoriasis before and after the indicated treatments. **** $P < .0001$, the fold change between the lesional and the nonlesional or the normal skin at baseline. ### $P < .001$, #### $P < .0001$, the fold change between the lesional skin at baseline and the lesional skin at the end time point.

Our report, together with previous studies,[3,4] suggests that the *ACE2* levels in psoriatic lesional skin are reduced by IL-17 blockade and that perhaps psoriatic patients who become COVID-19 infected may benefit from IL-17–targeted treatment. Although there is no evidence that COVID-19 spreads through skin contact, if future studies suggest that *ACE2* expression in psoriatic lesional skin correlates with COVID-19 susceptibility or severity, then perhaps IL-17–targeted therapies may be preferred in psoriasis patients at risk for COVID-19. This is worth confirming by further investigation.