



CUTTING EDGE CARE DELIVERY IN RESPONSE TO THE COVID-19 PANDEMIC

Skin and Mucosal Damage in Patients Diagnosed With COVID-19

A Case Report

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ABSTRACT

Patients admitted to the intensive care unit (ICU) are at a high risk for developing pressure injuries. A patient requiring multiorgan support is at a higher risk for pressure injuries related to immobility, sedation, vasopressors, and hypoxia. To mitigate pressure injuries, our hospital utilizes a bundle approach to prevent skin injury. However, despite efforts to prevent pressure injuries, we found our patients in the ICU with the diagnosis of COVID-19 went on to develop significant pressure and mucosal injuries. This is a case report of 4 patients diagnosed with COVID-19 who developed significant skin and mucosal injuries during their ICU admissions in the month of March 2020. We found that patients developed skin conditions that were initially thought to be deep-tissue injuries (DTIs) early in the admission. The DTIs progressed over the course of the admission in the ICU and evolved to thick adherent eschar that appeared to be unstageable pressure injuries, which extended beyond the soft tissue directly over the bony prominence. We also found that skin damage to the mucosa of the nares, tongue, lips, and urethra presented first as inflammation and then progressed to thick eschar. Despite maximum pressure relief with the use of a pressure-relieving turn and position system, bordered foam dressings, fluidized positioners, specialty beds, and leadership support for twice-a-week skin checks, our patients diagnosed with COVID-19 developed extensive skin damage across the fleshy portion of the buttocks and on the mucosa of the nares, tongue, lips, and urethra during minimal exposure to pressure. Although the initial presentation of the skin damage appeared to be related to pressure, the extent of the skin damage suggests a vascular inflammatory process beyond skin damage related to pressure.

KEY WORDS: COVID-19, deep-tissue injury, mucosal injury, pressure injuries.

INTRODUCTION

Our 260-bed hospital is centrally located within a metropolitan city that serves an underserved community and saw a rapid increase in admissions of patients with COVID-19. Our hospital has a level 2 trauma center with a helipad, as well as a certified stroke center. In our 48-bed intensive care unit (ICU), the pressure injury (PI) incidence rate is 0.001% per 1000 patient-days. Maintaining low PI rates takes a well-coordinated effort, and any incidence of a PI initiates a rapid response to treat it and leads to an in-depth evaluation of our current PI prevention bundle (PIPB).

Upon admission to the ICU, all patients are screened for PI risk by assessing mobility. All immobile patients are considered at risk for skin breakdown and receive the PIPB. The PIPB includes a pressure-relieving bed, turn and position overlay,

which is placed on the mattress, fluidized air positioners, foam dressings, and heel protectors. Immobility is a major risk factor that triggers a nutrition consult. Incontinence is managed based on what is best for the patient, with the intent to avoid an indwelling catheter if possible. We introduced the PIPB in January 2019 and found an 88% decrease in the incidence of PIs over the course of 12 months. Daily nursing and leadership engagement through rounds, reviewing documentation, and handoffs is part of our prevention model for skin and soft-tissue injuries; this engagement also aids in early recognition. At the start of the COVID-19 pandemic, we noticed that patients developed extensive skin injury despite the implementation of the entire PIPB.

We describe 4 case reports of patients who developed skin or mucosal injuries within several days of being admitted to our ICU during the month of March 2020. At the time of writing of this article, all 4 critically ill patients remained in the ICU. The authors monitored the patients' skin and reviewed the medical records to present the data for this report.

Our first case is a 44-year-old man who presented to the emergency department (ED) with complaints of malaise and shortness of breath after 2 weeks of not feeling well. His chronic conditions included diabetes mellitus and hypertension (Table). He was admitted to our ICU, intubated, and positioned prone for 4 days. Initially, it was unclear if he would tolerate position changes from prone to supine. He was positioned prone for 4 days, with head and arms repositioned every few hours. On day 4, extensive mucosal damage over his lips and nose was observed. The endotracheal tube was floated

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TABLE
Demographic and Clinical Characteristics of Patients

Characteristics	Patient 1	Patient 2	Patient 3	Patient 4
Demographic				
Age, y	44	68	66	71
Sex	M	F	M	F
Medical history	DM II, hypertension	Hyperlipidemia, DM II, hypertension	Hypertension	Hypertension
Symptoms at disease onset	Cough and fever × 14 d	Cough and fever × 10 d	Cough and malaise × 14 d	Cough, chills, night sweats × 14 d
Treatment before ICU admission	Direct admitted to the ICU; course of azithromycin and prednisone at home	Direct admitted to the ICU	Admitted to the floor and then transferred to the ICU; Tamiflu (oseltamivir) at home	Direct admitted to the ICU; course of azithromycin at home
Days on ventilator at time of PI	4 d	5 d	5 d	5 d
Blood gas prior at time of intubation				
pH	7.31	7.24	7.42	7.22
CO ₂	53.3	60.8	34.2	52.8
O ₂	81.0	124.0	45.5	88.4
HCO ₃	28.3	25.6	21.8	21.2
Base excess	1.7	-2.6	-1.8	6.7
Carboxyhemoglobin		0.3	0.3	0.3
Methemoglobin		0.3	0.6	0.5

Abbreviations: DM II, diabetes mellitus II; F, female; ICU, intensive care unit; M, male; PI, pressure injury.

off the lips and face and positioned side to side on the fluidized positioner. During the daily rounds, attention was given to ensuring that devices were not resting on his face during the prone position. In addition, there were deeply discolored areas over his cheek (Figure 1) and the tops of his feet initially presented as nonblanchable erythema on day 4 that progressed to a thick necrotic area with demarcated edges over the course of 2 weeks. The areas of skin damage over his lips, nose, cheek, and tops of his toes showed adherent, black, necrotic tissue with well-demarcated edges.

The second case is a 68-year-old woman who presented to the ED after 10 days of not feeling well with flu-like symptoms. Her chronic conditions included hyperlipidemia, hypertension, and type 2 diabetes (Table). She was intubated upon admission to the ICU for shortness of breath. Initially, she was positioned supine with the PIPB in place. On day 5, a stage 1 PI was noted over her sacrum, under the foam prevention dressing. The patient was then transitioned to prone positioning

that day for respiratory support. While prone, the area over the sacrum continued to evolve despite complete pressure relief. Over the course of 23 days, the area evolved to what appeared to be a deeply discolored area that then suddenly, over a 3-day period, evolved to a thick black eschar with deeply demarcated edges (Figure 2).

The third case is a 66-year-old man who presented to the ED after 14 days of feeling ill. His only chronic condition was hypertension (Table). He was initially admitted on the medical-surgical floor with nasal cannula oxygen support but then transitioned to the ICU for intubation and was positioned prone. Initially, erythema was noted over the right ear lobe on admission to the ICU. Nursing documentation noted that the nasal cannula was tucked behind his ear with foam padding. The area over the ear appeared to stabilize as a deeply discolored area; however, as the



Figure 1. Facial skin presentation.



Figure 2. Sacral skin presentation.



Figure 3. Outer ear skin presentation.

patient continued to improve while intubated, the discolored area evolved to thick necrosis area (Figure 3).

Our fourth case is a 71-year-old woman with a darker skin tone (no image available) who presented to the ED after 14 days of not feeling well. She was noted to have hypertension as the only chronic condition (Table). She was admitted to the ICU and intubated for respiratory distress. On day 5 of intubation, discolorations over her upper lip and end of tongue were noted. Both areas progressed to being swollen, discolored, and then became necrotic with dark eschar, with no noted fluctuant, drainage, or odor. Areas of skin damage on her sacrum were noted as deeply necrotic with thick black eschar.

DISCUSSION

We report on 4 patients who were admitted to our ICU, some of whom subsequently developed PIs and skin damage to mucosal sites. It is unclear if the patients were experiencing skin damage related to pressure alone, device-related skin damage, and/or in combination with a vascular event that occurred as a result of their infection. Given our ICU's low PI rates, we assume that the pressure relief and skin care that was provided was maximal. It is well known that patients admitted to the ICU are at a greatest risk for developing PIs related, in part, to immobility and physiologic instability.¹⁻³ Maximizing pressure relief minimizes the impact of damage on soft tissue and skin, decreasing the rates of PIs.¹⁻⁶ Our current PIPB utilizes air, foam, and pressure displacement technology to maintain skin integrity and includes efforts to minimize skin breakdown related to devices. However, despite these prevention interventions, the patients diagnosed with COVID-19 went on to develop extensive skin and mucosal damage.

The skin and mucosal injuries noted in our critically ill patients appear to be multifactorial. One similarity noted among these 4 patients was hypoxemia due to compromised oxygenation, ventilation, or a combination of both. During this period of hypoxemia, the vascular compromise at the capillary level may have been further compromised with even minimal pressure. The addition of vasopressors may add additional barriers to blood flow to the epidermal layers.⁷ The combination

of hypoxemia, vasoconstriction, and decreased blood flow through the dermis layer results in ischemia and tissue death with minimal exposure to pressure.

Tissue death can occur with prolonged periods of exposure to low levels of pressure or short periods of high pressure.¹⁻⁶ Despite pressure relief, once admitted to the ICU, the initial injury may have occurred during the acute hypoxemic period, even prior to ICU admission. Even with the correction of hypoxemia, the capillary bed constriction related to vasopressors would inhibit oxygenated blood flow to the compromised epidermis, further setting up the patient for reperfusion injury.⁷⁻⁹ The delay in adequately oxygenated blood flow to the area of tissue damage would result in further cellular death.

Once blood flow is reestablished to the capillary bed with the decreased rate of vasopressors, correction of blood pressure, and stabilization of oxygenation, the skin or tissue is still vulnerable to damage related to ischemic-reperfusion injury.⁷⁻⁹ After reviewing these 4 cases, we feel that our patients were displaying skin and mucosal damage related to pressure and ischemic-reperfusion injury.

There is minimal guidance in the literature on the management of ischemic-reperfusion injury.^{10,11} Current models for prevention and treatment are animal based (mice) and not translatable to the critically ill, unstable patient population diagnosed with COVID-19. The lack of clear treatment of the skin and mucosal damage, which traditionally includes debridement, further compounds the complexity of the patient care for this special population who might be experiencing vascular compromise. Understanding the manifestations and progression of COVID-19 is the first step in developing a prevention and treatment plan.¹² Deciding when to start conservative debridement is unclear because of the multisystem inflammatory response in these patients. Surgical debridement is not a clear option, given the inherent risks related to COVID-19 and wound-healing potential. For the moment, our hospital is taking a conservative approach while monitoring the patients' progress.

CONCLUSION

We were not expecting to see this level of severity of skin and mucosal injury in these ICU patients. It was disconcerting to observe the extensiveness of the skin and mucosal damage, although we provided our best practice, evidence-based, and highly effective PIPB. Equally disconcerting was the lack of a clear pathway for managing injuries, we believed, in part, to be caused by a combination of ischemic-reperfusion and pressure. Extensive research is needed to understand the pathophysiologic processes in patients diagnosed with COVID-19 and how best to prevent skin injury and/or maintain skin integrity.

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