BURN MANAGEMENT IN A PATIENT WITH COMORBID SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT

Bayli Davis, MBA, Hussain Abidi, Alan Pang, M.D., John Griswold, M.D.

TEXAS TECH UNIVERSITY HEALTH SCIENCES CENTER™
School of Medicine

INTRODUCTION

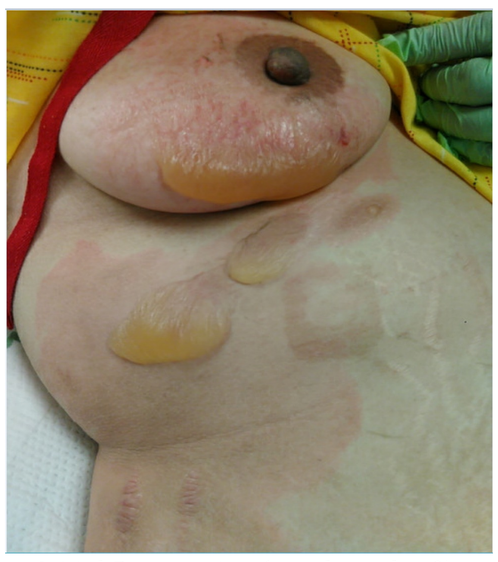
Systemic Lupus Erythematosus (SLE) is an autoimmune disorder in which the body elicits an immune response against its own tissues, resulting in widespread inflammation and multi-organ damage [1]. The underlying pathophysiology behind SLE is complex, but it is known to involve an aberrant adaptive immune response with B and T lymphocyte activation and subsequent autoantibody-mediated tissue damage leading to widespread inflammation [2]. Because of the inherent inflammatory nature of SLE, therapeutic strategies are primarily centered around suppressing the pathological immune response with immunosuppressive pharmaceutical agents [3-5].

Similar to the pathophysiology behind SLE, elicitation of an inflammatory cascade of events is paramount to the body's response to burn injury [6]. Importantly, immunosuppressive medications such as those used to treat SLE counteract the body's natural healing process that occurs in the post-burn state by suppressing the inflammatory response to tissue injury. This complicates the management of burn patients with comorbid SLE and necessitates special consideration on how to approach burn therapy in these patients.

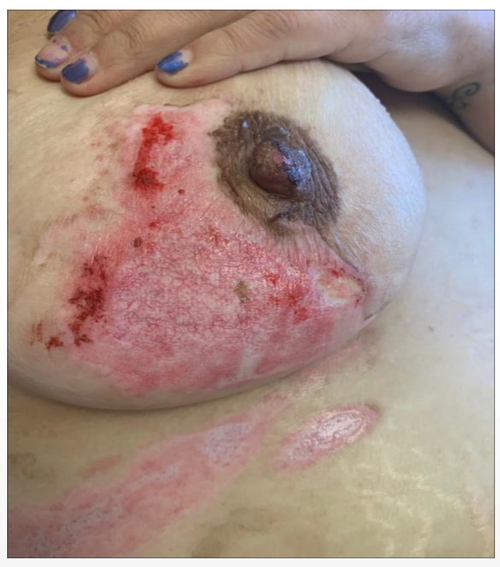
METHODOLOGY

This is the case of a 28-year-old female with a past medical history of SLE, lupus nephritis, renal vein thrombosis, fibromyalgia, hypertension, and recent COVID-19 infection who was admitted to the hospital for neurological manifestations in the setting of lupus. She initially presented to the emergency department with complaints of right lower extremity numbness, pain, and weakness with associated bladder and bowel incontinence. Head CT showed unchanged hypodensities and calcification of parenchyma. MRI of the spine showed multiple enhancing lesions in the brainstem, cerebellum, cervical spine, and thoracic spine. MRI of the brain showed subtle T2 hyperintensity in the left optic nerve. Lumbar puncture was negative for infection. She was subsequently found to be positive for AQP4-IgG and was diagnosed with neuromyelitis optica spectrum disorder, a condition frequently seen in association with autoimmune conditions such as SLE. Her neurological symptoms improved following a 7-day course of methylprednisolone and a 5-day course of IVIG. Her hospital course was complicated by 1) acute kidney injury, for which she was aggressively diuresed and resumed treatment with CellCept with improvement in kidney function; 2) gastrointestinal bleeding for which she was started on Protonix, discontinued Eliquis, and administered two units of packed red blood cells; 3) accidental scald burn to the right torso.

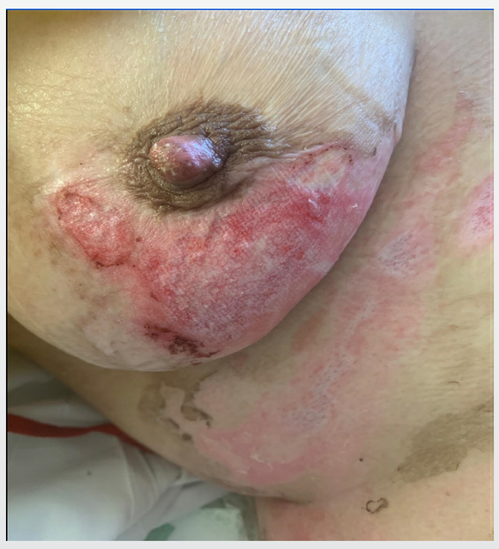
The burn unit was consulted for this patient on hospital day 15 after she accidentally spilled coffee on herself while in the hospital. She was found to have 2% Total Burn Surface Area (TBSA), second degree scald burns to the right flank and right breast. Burn wounds were cleaned and dressed; PluroGel, Aquaphor, and chlorhexidine (CHG) were applied to the wounds. On post-burn day 1, the rheumatological management plan was to continue CellCept, hydroxychloroquine, and methylprednisolone. By post-burn day 3, cyclophosphamide with mesna was added to her rheumatologic treatment regimen.



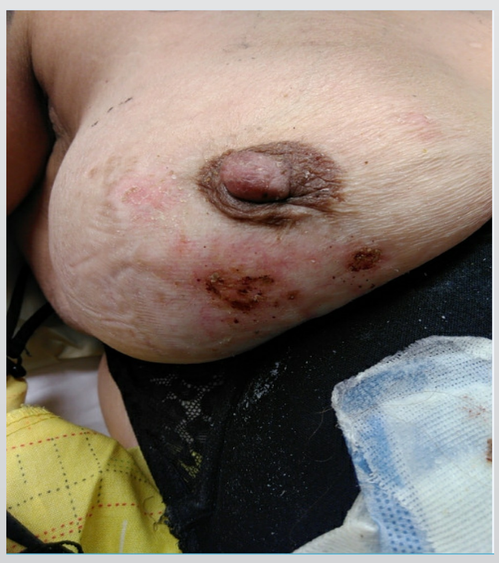
Initial Presentation (post-burn day 0)



Post-burn day 12



Post-burn day 13



Post-burn day 80

RESULTS

Regarding burn management, the patient underwent daily wound cleansing with CHG, mechanical debridement, and dressing change with the application of PluroGel, Aquaphor/Adaptic, dry gauze and tape. Starting on post-burn day 7, Sulfamylon was applied and use of PluroGel was discontinued. On post-burn day 10, the topical was changed back to PluroGel, which was applied to all areas of burns with nonviable tissue. Nivea cream was applied to all healed burns at this point.

Ultimately, the patient's wounds largely healed with topical medication alone, albeit more slowly than what is typically seen in burn patients that are not on immunosuppressive therapy. On post-burn day 2, the patient's wounds were noted to be healing very slowly. By post-burn day 6, the wounds still had a moderate amount of nonviable tissue (NVT). Two days later, there was an additional 10% conversion of burn wounds to NVT. By post-burn day 15, the majority of wounds were healed except for the right breast. The possibility of split-thickness skin grafting was considered at this time but ultimately was not needed.

CONCLUSION

Regarding burn management, our patient was observed to have a slow healing process superimposed on management with an immunosuppressive medication regimen she was started on for neuromyelitis optica spectrum disorder. This implicates that the two treatment approaches – one addressing her rheumatologic presentation and one addressing her dermatologic presentation – counteracted one another. When treating burn patients with concomitant rheumatologic disease, clinicians should weigh the risk of surgical intervention during an acute exacerbation of rheumatological conditions with maximum immunosuppressive therapy. This case shows that even with maximum immunosuppression, second degree burns can heal with non-operative management alone.

APPLICABILITY TO CLINICAL PRACTICE

This case adds to a small yet growing body of literature addressing the clinical presentation and management of burn wounds in the setting of SLE. Burn management in our patient with comorbid SLE complications, such as neuromyelitis optica spectrum disorder, was complicated and unclear, but adds to the algorithm of burn treatment with concomitant rheumatological disease. Delayed wound healing should be anticipated in patients that are on immunosuppressive agents such as methylprednisolone, CellCept, and hydroxychloroquine. Further research should aim at balancing risk of excision & grafting versus local wound care in patients on immunosuppressive regimens.

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