Lupus Cerebritis After Visiting a Tanning Salon

Abdallah S. Geara, M.D., Estelle Torbey, M.D., and Badiaa El-imad, M.D.

Abstract
A 53-year-old female was admitted to the intensive care unit for lupus cerebritis; she had a 15-year history of stable lupus. Over the prior 1 to 2 months, the patient visited a tanning salon and this triggered the exacerbation of lupus. Her initial symptoms were cutaneous in the form of an erythematous rash. Within 2 weeks she started to have headaches and was admitted for seizure and psychosis. Ultraviolet A exposure in the tanning salon is known to exacerbate lupus by modulation of the immune system at the level of the skin. It has also been found that ultraviolet light can lead to the formation of antinuclear antibodies. This case illustrates the need to emphasize the danger of the tanning salon to patients with systemic lupus erythematosus; the risk is not only cutaneous, it can also be systemic.

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease of the connective tissue. Its clinical course is characterized by several episodes of exacerbation involving almost all of the organs. More than 50% of all patients with SLE have neurological involvement with a wide array of nervous system disorders: focal (e.g., transient ischemic attack, stroke, transverse myelitis, peripheral neuropathy, and movement disorder), nonspecific (e.g., headache and seizure), and neuropsychiatric (e.g., anxiety, delirium, and depression). The exacerbation of these symptoms are triggered by several specific factors (e.g., infection, medications, and sunlight). We present a patient known to have SLE, who was admitted for lupus cerebritis triggered by exposure to ultraviolet light from a tanning bed.

Case Presentation
A 53-year-old Caucasian female presented to the emergency department with a decreased level of consciousness of several hours duration. The patient was found by her husband lying in her bed, severely obtunded, with fecal and urinary incontinence and possible generalized clonic movements. In the emergency department, the patient was intubated to protect her airways. The physical exam did not show any neurologic focalization. She was febrile (104°F). She had a diffuse erythematous generalized macular rash of the face, trunk, upper extremities, lower extremities, palms and soles, and her knees and ankles were swollen.

The patient suffered from SLE, which had been diagnosed 15 years earlier and was known to have multiple bouts of arthritis, photosensitivity, and malar rash. Her serology was positive for anti-nuclear antibodies (ANA), the complement levels were normal and anti-double stranded DNA antibodies (anti ds-DNA) were negative when checked between episodes of exacerbations. Her disease was well controlled with a low dose of prednisone (10 mg/day).

In the emergency department, the patient was started on phenytoin for seizure and antibiotics (ceftriaxone, acyclovir, ampicillin, and vancomycin) for suspected meningoencephalitis. These antibiotics were stopped after the lumbar puncture failed to reveal any increase in WBCs or RBCs in the cerebrospinal fluid (CSF). The CSF Gram stain was negative. The only abnormal finding was high protein in the CSF (87 mg/dl). Cerebrospinal fluid cultures, stains, viral PCR for HSV, and West Nile and Lyme antibodies were each negative. An electroencephalograph showed diffuse slowing. The initial computed tomography (CT) scan of her head was normal, as was a follow-up scan performed 48 hours after admission.

Abdallah S. Geara, M.D., Estelle Torbey, M.D., and Badiaa El-imad, M.D., are in the Department of Internal Medicine, Staten Island University Hospital, Staten Island, New York.

Correspondence: Abdallah S. Geara, M.D., Department of Internal Medicine, Staten Island University Hospital, 784 Olympia Boulevard, Staten Island, New York, 10305; abdallah.geara@gmail.com.
At this point, the patient was started on 1 mg/kg/day of methylprednisolone (total dose: 90 mg/day) for lupus cerebritis. The ANA titer was 1:160 and both C3 and C4 levels were decreased. The patient progressively improved over the course of 2 weeks: the rash decreased in intensity, and she slowly and steadily regained her previous mental status and was extubated. A cranial magnetic resonance (MR) image performed after extubation showed microvascular ischemic changes and patchy areas of high intensity on T2 and Flair sequences that did not enhance with gadolinium.

The patient’s husband reported that over the last 2 months his wife had been visiting a tanning salon 1 to 2 times per week. Her rash started 1 to 2 weeks prior to her admission to the hospital. And 3 days prior to admittance, she started complaining of headaches and vomiting with fatigue.

Is her visit to the tanning salon related to her current SLE exacerbation?

**Discussion**

It is well established that sunlight can induce and exacerbate cutaneous manifestation of SLE. The initial investigations found that the action of sunlight was mostly in the ultraviolet B (UVB) range. Currently, there is growing evidence that ultraviolet A (UVA) radiation may affect the pathogenesis of SLE. Several previous case reports described flare-up of SLE following exposure to different sources of ultraviolet (UV) radiation.

Pace and colleagues described the case of a patient diagnosed with SLE following recurrent acute confusion episodes induced by prolonged sunlight exposure. Stern and associates presented the case of a patient who was diagnosed with SLE following exposure to long-wave UVA radiation in a tanning salon. The patient had, in addition to the rash, articular and renal manifestation of lupus increasing the evidence that UV light can exacerbate both cutaneous and non-cutaneous symptoms. Klein and coworkers reported the case of a patient who worked as a photocopy technician and developed cutaneous SLE after exposure to occupational sources of UVA light from the photocopy devices.

Pathophysiologically, the interaction between UV light and the immune system was tested in several experimental models. Nyberg and colleagues studied the immunologic modification that occurred in LE patients after photoprophylaxis by UVA and UVB. Cellular adhesion molecules expression was increased at the level of the keratinocytes, endothelial cells, and dendritic cells. The induction of adhesion molecule expression was found after exposure to both UVA and UVB. Bennion and associates found that UV light, in addition to modulation of the adhesions molecules, could activate immune receptors, cytokines, and a cascade of events involving cellular signaling and apoptosis.

In addition to the local cutaneous immunologic changes, UVA can also induce antinuclear antibody formation in both animal and human models. After UVA light exposure, the DNA will play the role of a chromophore leading to a photoprodut UV-DNA complex. This complex can elicit an immune complex reaction with resulting autoantibody and potential cellular toxicity. This will trigger inflammation systemically, including in the central nervous system where the immune complex causes blood brain barrier disruption, basement membrane thickening, and infiltration of neutrophils with endothelial cell lining disruption.

The tanning process uses mostly UVA light (15% UVB, 85% UVA). In the case we presented, the patient had received several tanning sessions of 30 minutes duration each. These sessions triggered an exacerbation of SLE that initially was mostly cutaneous but then progressed to articular and central nervous system involvement (headache, difficulty concentrating, and depressed mood). After extubation, the patient was delirious for several days. The treatment with steroids was sufficient for reversing the psychosis and successful in decreasing the rash and the articular swelling.

**Conclusion**

The patient we presented had mostly articular and skin manifestation of SLE for more than 15 years. Her current admission was for a seizure secondary to SLE cerebritis. Her recent exposure to high dosages of UV light in the tanning salon triggered a photosensitivity skin reaction. A possible role of this exposure in triggering the SLE cerebritis is highly probable. The immune function in SLE could be altered by exposure to UV light in tanning salon, as presented previously. This alteration in the immune system increased our belief that, in addition to induction of skin photosensitivity reaction, exposure to UV light in a tanning salon could be a trigger factor in several manifestations of SLE (e.g., articular, renal, and neurologic). To the best of our knowledge no previous report has implicated UV exposure from tanning with such a severe exacerbation of symptoms in SLE patients.

**Disclosure Statement**

None of the authors have a financial or proprietary interest in the subject matter or materials discussed, including, but not limited to, employment, consultancies, stock ownership, honoraria, and paid expert testimony.

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