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Photic Maculopathy in a Patient Receiving Bright Light Therapy

TO THE EDITOR: Seasonal affective disorder is characterized by recurrent episodes of winter depression with summer remissions (1). Several applications of artificial bright light therapy for the treatment of seasonal affective disorder have been reported. We present a case in which the association of bright light therapy with clomipramine treatment may have resulted in photic maculopathy.

Mr. A was a 35-year-old man with symptomatic bilateral reduction of visual acuity and a positive central scotoma. He suffered from recurrent major depression, seasonal pattern. No retinal abnormalities had been found at two routine ophthalmic examinations performed 3 months and 1 week before the start of a 2-week cycle of daily 60-minute bright light therapy sessions with full-spectrum fluorescent light. During treatment, he continued his current medication regimen of clomipramine, 100 mg/day. After 5 days, there was a marked reduction of visual acuity and contrast sensitivity. Results of an Amsler test revealed a relative central scotoma, and static perimetry showed a marked decrease in foveal threshold in both eyes. A central yellowish-white lesion with surrounding orange-red halo was also found in both eyes. Fluorescein angiography results did not reveal foveal staining or leakage.

One month later, visual acuity was 20/20 in both eyes, contrast sensitivity and foveal threshold showed only slight impairment, and a second Amsler test detected a small relative central scotoma. Mr. A did not complain of the presence of scotoma and noted a complete recovery of visual acuity. After 1 year, the slight reduction of contrast sensitivity and relative scotoma had persisted, but electroretinogram results revealed no abnormality.

To our knowledge, this is the first report of the occurrence of photic maculopathy related to the association of bright light therapy and clomipramine treatment. This tricyclic antidepressant is an active photosensitizer that transfers energy to ground state oxygen (2). Polycyclic aromatic compounds have a high affinity for melanin, and the extensive accumulation of drugs by melanin appears to be the most important factor that governs long-term therapeutic and toxicological activities (2). In addition, high-affinity binding sites for antidepressant tricyclic drugs have been detected in chicken and pig retinas (3). The anticholinergic effect of clomipramine on pupil size could increase the potential risk of retinal hazard by having a cumulative effect with photosensitization. Furthermore, the exposure to clear winter sunlight for several hours perhaps contributed to the development of photic maculopathy.

In conclusion, photic maculopathy in this patient appeared to be related to the association of bright light therapy, photosensitizing medication, and long-term exposure to sunlight. However, it is not possible to determine the relative importance of each factor in determining retinal damage. Moreover, it is important to note that the aforementioned relationship has not been definitively demonstrated and that this explanation needs further investigation in order to be confirmed.

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PTSD After a Peacekeeping Mission

TO THE EDITOR: Our research on the psychological sequelae of peacekeeping missions has shown that unique peacekeeping stressors are associated with the development of posttraumatic stress disorder (PTSD) (1). Exploration of factors that are relevant to the emergence of symptoms after such nontraditional combat will help prepare us to effectively treat the veterans of tomorrow (2). We present here a case in which careful assessment of the idiosyncratic meaning of one of these unique stressors (inability to alleviate the suffering of children) assisted in the understanding and treatment of peacekeeping-related PTSD.

Mr. A was a 24-year-old, white, married, Marine with distress associated with his recent deployment to Somalia. He reported that he had adjusted well to the military and denied any mental health difficulties before deployment. He had previously participated in two deployments during which he was exposed to direct life threat (e.g., hostile engagement, threat of biological and chemical warfare); however, he did not subsequently develop any lasting PTSD symptoms associated with these missions.

Nonetheless, after his deployment to Somalia, Mr. A began to exhibit severe symptoms of PTSD (daily intrusive images of starving Somali children, frequent distressing nightmares that involved these children, marked symptoms of arousal and avoidance). What was unique about his presentation was that rather than relating to instances of life threat, his symptoms were tied to seeing starving children and feeling guilt and shame because of his inability to save them. His irritability and angry outbursts were often cued by his perception of his wife's inadequacy in caring for their own child. Thus, children suffering was a salient theme in this veteran's response to trauma, which ultimately led to a focus in treatment on his own childhood suffering (his mother left him when he was 2 years old).

Treatment first focused on disclosure of the events in Somalia and expression of the associated emotions. Mr. A expressed sadness and anger regarding his inability to help starving Somali children as well as anger toward his wife regarding her care of their child. He gradually was able to acknowledge his anger toward his mother for abandoning him and sadness at this loss, which led to relief, resolution, and improved functioning.

Inability to help starving children would not be considered a classic war-zone stressor, yet in this case these events resulted in the hallmark intrusive symptoms of PTSD (flashbacks,