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LETTERS TO THE EDITOR

Advancing Treatment of Comorbid PTSD and OSA

Response to Gupta. Treatment of PTSD-related OSA with CPAP is associated with only a modest improvement in PTSD: possible adjunctive treatment with mood stabilizers. *J Clin Sleep Med.* 2017;13(6):841.

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We find Dr. Gupta's letter to the editor thought provoking as it highlights the need for novel approaches to treating patients with both posttraumatic stress disorder (PTSD) and obstructive sleep apnea (OSA). Dr. Gupta reports her anecdotal observations that anticonvulsants in patients intolerant to positive airway pressure (PAP) sometimes result in improvements in OSA and PTSD severity.[1] The bidirectional relationship between OSA and PTSD has been postulated for years; sleep disruption secondary to OSA exacerbates PTSD symptoms, and hyperarousal and sympathetic activation from PTSD predisposes to upper airway collapse. Furthermore, increased sleep disruption attributed to OSA, especially in younger military populations, may interfere with the natural recovery process of trauma, leading to higher rates of PTSD.[2] Mechanistic evaluation of this relationship is thus long overdue.

Regardless of the potential causal relationship between PTSD and OSA, we recommend conceptualizing and approaching OSA and PTSD as two co-occurring but independent disorders. The Veterans Affairs/Department of Defense Clinical Practice Guidelines for PTSD management emphasize the use of treatments demonstrating the greatest efficacy, including trauma-focused psychotherapies and selective serotonin reuptake inhibitors/serotonin-norepinephrine reuptake inhibitors.[3] Guidelines do not support the use of anticonvulsants in the treatment of PTSD. A recent series of meta-analyses of PTSD treatments supported the use of trauma-focused psychotherapies as first-line interventions and found divalproex failed to differentiate from placebo as monotherapy or combined with an antidepressant.[4] No medications have proven efficacious in the treatment of OSA, and no studies have evaluated the effects of anticonvulsants in patients with PTSD/OSA.

Sleep disturbance is a hallmark symptom of PTSD, but sleep is only one factor affecting daytime PTSD symptoms. Thus, although heightened arousal may lead to higher incidence of OSA, treatment of OSA is unlikely to fully eliminate hyperarousal, particularly during the daytime. This is evidenced by Orr et al.[5] finding a modest reduction in PTSD symptoms among veterans using PAP. PAP should be considered a *component* of treatment in patients with PTSD/OSA, but should not be viewed as a magic bullet for curing PTSD. Efforts to increase PAP adherence in patients with PTSD (i.e., desensitization) should be incorporated into medical sleep clinics. However, we agree with Dr. Gupta that exploration of new potential therapies in patients intolerant to PAP and refractory to established PTSD treatment is warranted.

The clinical conundrum remains: do we offer PAP to treat OSA in patients with PTSD, or do we encourage treatment for PTSD first, particularly in cases where OSA is only mild? With increased rates of OSA testing in veterans and soldiers with PTSD, many of whom are found to have mild disease, opportunities to answer these questions abound. Additionally, we call for greater collaboration between sleep and PTSD clinics to provide more patient-centric care. The challenge of treating patients with both PTSD and OSA is monumental due to the additive burden of both comorbidities as well as the existing siloed nature of providers rendering treatment. Optimal care will be rooted in collaborative investigation of many of the gaps highlighted in recent publications, evidence-based treatments, and enhanced patient engagement for both disorders.

CITATION

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