

Debunking Myths about Treatment Emergent Central Sleep Apnea

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Received date: January 28, 2021; Accepted date: February 16, 2021 ; Published date: February 22, 2021

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Citation: Malaiya N, Sinha MR (2021) Debunking Myths about Treatment Emergent Central Sleep Apnea. J Sleep Disord Ther 10 e147. doi: 10.4172/2167-0277.1000e147

Editorial

Dear editor,

Treatment Emergent Central Sleep Apnea (TECSA) is a polysomnographic phenomenon whereby there is emergence of new central respiratory events (central apneas and hypopneas) after pre-existing obstructive events have mostly resolved while patient is being titrated with positive airway pressure (PAP) therapy [1]. The acronym TECSA was first used by Nigam et al. in 2016 while describing the prevalence and risk factors related to central sleep apnea developing instantaneously with PAP use [2]. They found that the aggregate point prevalence of TECSA is around 8% while the estimated range varies from 5% to 20% in patients with untreated OSA [2]. This phenomenon can occur at any CPAP setting including very low pressure settings [3]. Few years later, Nigam et al. in a sentinel paper demonstrated that TECSA may not always be transient after all. While most cases of newly identified TECSA resolve in few weeks to few months of PAP use, this is not true for all cases. In a rigorous systematic review, they found that about a third of patients with TECSA may continue to exhibit persistence of PAP related central apneas on re-evaluation [4]. A small proportion may experience what they call delayed-TECSA (D-TECSA), referring to patients that do not show TECSA on first titration but go on to develop TECSA few weeks to several months after initial exposure to PAP therapy [4,5].

These novel findings have several clinical implications. First, TECSA appears to be elusive polysomnographic traits along a dynamic spectrum between sleep disordered breathing without TECSA at one end and chronic persistent TECSA at the other end that could be easily missed unless vigilantly sought after [4]. Second, TECSA is not always a transient process. It evolves into an ongoing chronic, iatrogenic disorder related to regular PAP use for about one-third of the patients afflicted with it. Third, the recent discovery of the category of D-TECSA proves that not all cases will present with TECSA right away upon initial exposure to PAP therapy [4]. If a patient who is demonstrating good adherence to PAP therapy initially is struggling with PAP usage few months to years later, then we need to consider D-TECSA as one of the culprit causes. Fourth, in rare instances TECSA can trigger profound oxygen desaturations which probably may not have occurred to this detrimental magnitude had their obstructive sleep apnea (OSA) been left untreated [6].

Nigam et al. have systematically proven that TECSA needs to be a vital concern when titrating any patient with PAP therapy for

correction of OSA. This underscores the importance of attended in-lab titrations for all patients regardless of patient co-morbidities as long as sleep study costs can be managed by the stakeholders. Also, we need to take a step back and ponder which patients will benefit maximally from PAP therapy, while concurrently respecting our Hippocratic oath of “do no harm” [7]. A patient with mild OSA who is asymptomatic otherwise might benefit from non-PAP related therapies (such as weight loss as indicated or positional therapy) instead of reflex, blanket migration to PAP therapy for all patients with OSA. The need for periodic follow-up of the patient with their sleep physician becomes even more crucial in light of these new findings, given some patients with OSA may continue to have TECSA for several years while a select few may develop TECSA all of a sudden, months to years after initial PAP exposure. A careful review of PAP download data may also help especially in cases where a sudden persistent spike in central apnea index or mixed apnea index is denoted by the PAP download report. We hope further work in field of TECSA will continue to broaden our understanding of this elusive polysomnographic phenomenon.

Conflict of Interest

I certify that there is no actual or potential conflict of interest in relation to this article.

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