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COMMENTARY

Cutting for Cures: Bariatric Surgery and Obstructive Sleep Apnea

Commentary on Magne et al. Evolution and predictive factors of improvement of obstructive sleep apnea in an obese population after bariatric surgery. *J Clin Sleep Med*. 2019;15(10):1509–1516.

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Despite recognition, the obesity epidemic continues worldwide effecting neurological, cardiovascular, metabolic, respiratory, and sleep health of many people. In the USA, in 2015-16 the prevalence of obesity was 39.8%.[1] According to some projections, by 2050, as many as 60% of males and 50% of females will be obese in the United Kingdom.[2] Obesity is a well-known risk factor for obstructive sleep apnea (OSA). The prevalence of obstructive sleep apnea is reported in the ranges of 55% to 100% among severely obese patients, who have a higher apnea-hypopnea index (AHI) and more severe disease.[3] Besides the apparent mechanical or anatomical collapse of the upper airways as a result of the excess tissue or fat deposition, an alternation in the central sleep regulatory system as well as the respiratory muscle tone contributes to the disease pathology.[4] Those with extreme obesity (BMI $\geq$ 45 kg/m^2) have a distinct form of OSA characterized by hypopneas with a relative paucity of complete apneas.[5] This results in a lower critical closing pressure (Pcrit) and lower level of continuous positive airway pressure (CPAP) for resolution of the sleep-disordered breathing despite relatively high apnea hypopnea indices.[6] Treatment of choice is CPAP, combined with a long-term weight loss strategy. However, significant weight loss is often difficult and its maintenance is even more challenging for many people. Thus, bariatric surgery has now been utilized in morbidly obese patients who have failed conservative therapy or have significant comorbidities. It is important to note that while bariatric surgery leads to weight loss and a reduction of AHI, it may not distinctly cure OSA. Previously, a systematic review and meta-analysis reported OSA cure rates of more than 80% in patients who underwent bariatric surgery.[7] In 2009, Greenburg et al described the limitation of these findings, stating that the evaluated patients had a heterogeneous group of sleep disorders including obesity-hypoventilation syndrome instead of strictly OSA. In addition, there was an absence of clear diagnosis and unavailability of polysomnography post bariatric surgery when assessing outcomes. His systematic review and meta-analysis in 2009 refined the search terms and reported the effect of surgical weight loss on solely OSA with a focus on mean AHI reduction. Even though there was a significant AHI reduction by 38.2 events/h, the patients were not cured of OSA and had persistent moderately severe OSA.[8] Dixon et al also reported a reduction in AHI among obese patients with OSA who underwent laparoscopic adjustable gastric banding, but additionally showed improvement in sleep architecture, daytime sleepiness, and overall quality of life.[9] However, his randomized controlled trial in 2012 comparing conventional therapy versus bariatric surgery revealed no overall statistical difference in AHI reduction. Some patients had greater improvement than others, and the weight loss to AHI reduction relationship was not linear.[10] We now know that the pathogenesis of OSA may be more complex than a simple reduction in mechanical load that obstructs the airways. Factors such as age, sex, and underlying medical conditions would potentially affect the degree of OSA improvement for obese patients undergoing bariatric surgery. Older age had been shown to be negatively associated with percent of total weight loss after bariatric surgery, but is not associated with postoperative complications.[11]

In this issue of *Journal of Clinical Sleep Medicine*, Magne et al report younger age and lower nocturnal oxygen desaturation index (ODI) as the predictive factors for improvement in OSA after gastric bypass surgery (GBS) among obese patients.[12] The factor of younger age has already been reported in previous studies as a positive predictor, but this prospective study corroborates these findings and adds lower nocturnal ODI as an important predictive factor.[4] Despite the small sample size, this study confirms the complex pathogenesis of OSA and a nonlinear relationship of AHI reduction with weight loss after bariatric surgery. It is important to note that in this study 77% of patients still had OSA (AHI $\geq$ 5 events/h) despite the surgery and weight loss, and the mean postop AHI was 11.9 events/h. Only 10 of the 44 patients (22.7%) were "cured" with AHI < 5 events/h, while 31 patients (70.5%) improved enough to no longer meet French Ministry of Health criteria for treatment. These criteria are; clinical symptoms and (1) AHI $\geq$ 30 events/h; (2) AHI = 15–30 events/h with $\geq$ 10 respiratory event-related arousals/h; (3) AHI = 15–30 events/h with $\geq$ 1 cardiovascular risk factor. The patient sample in this study were predominantly women with a paucity of symptoms except snoring, and most had no cardiovascular risk factors. Finally, while GBS resulted in an

improvement of AHI to < 15 events/h in obese patients with OSA leading to termination of CPAP therapy per French Ministry of Health criteria in this particular study, it would be interesting to explore whether or not the patients with AHI = 5–15 events/h would benefit from CPAP therapy. Nevertheless, based on these findings, a certain segment of the obese population (ie, age < 55 years) may be more encouraged to undergo GBS than others. The evidence continues to accumulate for the benefits of bariatric surgery, especially for those with sleep-disordered breathing. Future studies identifying additional predictive factors are likely to emerge.

CITATION

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