

Case Description: A 56-year-old woman with DM and allergic rhinitis had chief complaint of disruptive sleep and loud snoring with witnessed sleep pauses for about 4 years. Nasal blockades have become worse in the last two years. ESS 8, BMI 30, neck circumference was 37 cm. Local PE showed lateral peritonsillar narrowing, MPS 2 and grade 2 palatine tonsillar hypertrophy. She also had moderate COPD of Gold stage I, group B of mixed allergic eosinophilic endotype and treated with regular therapy but still experienced frequent moderate AE of COPD together with acute flare episodes of CRS. Naso-laryngoscopy and sinus CT showed inferior turbinate hypertrophy, significantly retained secretion in both maxillary sinuses, ethmoid sinuses, and circumferential narrowing of retropharyngeal lumen at soft palate level. Very Initial AHI was 45/h, ODI of 52/h, and snoring index of 255. AHI down to 15 on CPAP pressure 8 cmH₂O. But owing to intolerance CPAP use and failed response to oral antibiotics, IN-CS and OCS, she performed bilateral pan-sinusotomy and stereotactic procedure for paranasal and facial sinus washing and biopsy, and partial inferior nasal turbinectomy. Biopsy report showed chronic inflammation of rhino-sinuses. Post-surgery AHI on CPAP use improved to 5, ODI to 7, and CPAP intolerance diminished with much better comfort.



Figure (1)

Figure (2)

Figure (3)

Figure (4)

Figure (5)

Figure (1): Naso-laryngoscopy in 2021

Figure (2) & (3): Naso-laryngoscopy in 2024, showing IT hypertrophy and circumferential narrowing of retropharyngeal lumen at soft palate level

Figure (4): Sinus CT before nasal surgery in 2024

Figure (5): Sinus CT one month after nasal surgery in 2024

Interpretation: Patient was diagnosed with comorbid CRS-OSA and bilateral nasal IT hypertrophy, and COPD. However, she had CPAP intolerance and failed medical therapy including oral antibiotics and even regular IN-CS and OCS. Therefore, she received surgical therapy for chronic inflammatory problems of rhino sinus. Her AHI after surgery and on CPAP use got significantly improved with well tolerance and regular compliance to CPAP use with much better subjective comfort than ever.

Discussion: Obstructive sleep apnea is sleep-disordered disease characterized by airway obstruction at either single or multiple levels of upper airway during sleep due to either anatomical narrowing or obstruction or/and physiological collapse in different anatomic sites, different proportions and severities in individual patients

[1],[2]. Mutually interactive pathophysiology in comorbid CRS-OSA is due to late phase reaction secondary to chronic allergen exposure, leading to dilated nasal mucosal capacitance vessels, local congestion, retained nasal secretion, chronic nasal soft tissue swelling with decreased nasal diameter and obstruction, thereby inhibiting nasal mechanoreceptors, decreasing activation of nasal-ventilatory reflex and muscles tone of upper airway [3],[4]. Chronic arterial O₂ desaturation by impaired nasal airflow and frequent sleep apneic events cause increased inflammatory cytokines and then contributes to development of both higher disease burden, disease sequelae, higher oral antibiotics and OCS use, CPAP intolerance, and worse clinical outcomes such as reduced productivity, cognitive impairment and decreased quality of life [3],[4],[5]. The role of nasal surgery as salvage treatment in such patients with CPAP failure is to enlarge nasal passage and improve smooth nasal airflow, AHI, RDI, CT90% (Cumulated % of the time spent with SaO₂ of below 90%), NCV (nasal cavity volume), MCA (minimal cross-sectional area), and tolerate CPAP therapy [1],[4],[6],[7],[8]. Therefore, identification of promising candidates is vital to successful treatment with either primarily combining CPAP and nasal surgery or as salvage treatment in co-morbid severe OSA and CRS with local soft tissue hypertrophy and CPAP failure [8], [9].

Conclusions: In patients with co-morbid severe OSA and difficult to treat CRS with IT hypertrophy and impaired CPAP tolerance, we should consider possibility for intervening with surgical treatment for either CRS or OSA if exact site of upper airway obstruction was well detected in order to alleviate diseases burden, sequelae and chronic medication side effects, for enhancing upper airway (UA) airflow, arterial oxygen (O₂) level and improving AHI, ODI, daytime function, nighttime sleep, cardiorespiratory health and quality of life.

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