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Short Communication

Inter-Relationship between OSA and TMD

Ruchika Sood[1] and Shweta Hinduja[2]*

[1]*Director at Neocare Orofacial Pain Relief centre, India*

[2]*Associate oral physician at Neocare Orofacial Pain Relief centre, India*

***Corresponding Author:** Shweta Hinduja, Associate oral physician at Neocare Orofacial Pain Relief centre, India. **ORCID Id:** 0000 0003 1446 9099

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OSA is a sleep disorder characterized by obstruction in the oro nasal airflow lasting for more than 10 seconds. The patient experiences being chocked during sleep and thereby transient awakening, which further caused reduced oxygen saturation. This partial cessation of airflow due to obstruction in the upper pharynx or nasal cavity results in snoring, known as 'hypoaponea', during each episode of which a reduction is respiratory effort with ≥ 4% oxygen desaturation is recorded. Each episode lasts for 10- 40 seconds, sometimes may last for one minute, during which there maybe abrupt drop in the oxygen saturation level as much as 40% in severe cases. The brain responds to each of these episodes by alerting the body and causing deep arousal from sleep that causes normal breathing, referred to as fragmented sleep quality. OSA is characterized by total number of aponea and hypoaponeas occurring per hour of sleep, which refers to/aponea/hypoaponea index (AHI) [1]. AHI is accounted for in polysomnography test which is an attended setting (sleep Laboratory) test, along with several sleep variables, such as the respiratory disturbance index (RDI). RDI is defined as sum of the aponeas, hyopapneas and abnormal respiratory events per hour of sleep [2].

AHI has been widely used to diagnose OSA with different cut-off levels which are often unclear and arbitrarily determined, but generally AHI of more than 5 events per hour of sleep is considered abnormal. When the AHI is 5 - 15 events per hour, it is called mild sleep apnoea. AHI of 15 - 30 events per hour is considered as moderate obstructive sleep apnoea and AHI of more than 30 is considered as severe obstructive sleep apnoea. This combined with clinical evaluation leads to the diagnosis od OSA.

The prevalence of OSA is AHI index at ≥5 events/h has been from 9% to 38% it has been higher amonst men, and increased prevalence has been found with advancing age. In some elderly groups, the prevalence has been recorded to be as high as 90% in men and 78% in women. At ≥15 events/h AHI, the prevalence in the general adult population ranged from 6% to 17%, ranging to as high as 49% in the advanced ages.

Clinically aponea can be categorized into three categories, based on the anatomical involvement. In central sleep aponea there is cessation of breathing due to various disturbances in the ventilatory control of the respiratory centre of brain, this causes difficulty in putting effort to breathe during sleep. In case of obstructive sleep aponea, there is no disturbance at the respiratory centre of brain. The cessation of airflow is due to obstruction at the level of pharynx, which interrupts the airflow despite individuals effort to breathe. The mixed sleep aponea is a combination of both central and obstructive components.

Risk factors of OSA are obesity, drugs such as opiates, benzodiazepines, alcohol intake, smoking, supine body posture when sleeping, nasal congestion or obstruction, male gender, postmenopausal women, craniofacial features such as neck circumference of more than 40 cm, retrognathic mandible, nasal obstruction, enlarged adenoids and tonsils, macroglossia. Longer upper pharynx is another notable feature which increases the chance of airway collapse during sleep. The distance from soft palate to posterior pharyngeal wall may also be reduced which results in posteriorly positioned maxilla, thicker palate and enlarged uvula may also be observed. Pharynx is usually narrow and may have fat deposition around it making it susceptible to collapse of maxilla. Fat deposition around submandibular and submental region causes reduced diameter of the upper airway which contributes to obstruction during sleep.

Most people with OSA snore loud and frequent with periods of silence when airflow is reduced or blocked. Choking, snorting or gasping sounds may be heard when the airway opens.

Medical disorders such as Type 2 diabetes, Polycystic ovarian syndrome, coronary artery disease, stroke and congestive heart failure exceeds in patients with OSA than in general population. Therefore it is prudent for clinicians to routinely screen patients with above mentioned medical conditions when treating for OSA [3].

Population based studies have also shown a strong and independent association of various measures of OSA severity (AHI and oxygenation) with hypertension. Neurocognitive function, depression and risk for motor vehicle accident have also been associated with OSA. There is also data contemplating the association of OSA with TMD.

Temporomandibular joint disorderd (TMDs) are musculoskeletal disorders characterized by persistent pain in temporomandibular joint, masticatory muscles, and in preauricular region [4]. Prevalance of TMD is high, around 6-93% according to one study [5]. TMD is a complex multifaceted disease process is complex multi factorial. Biomechanical, neuromuscular, neurobiological, and biopsychosocial factors may contribute to the disorder [7]. The risk factors contributing to TMD are age, genetic factors, sex, stress, anxiety, occlusion, poor posture, rheumatoid arthritis. Dysfunctional breathing is also regarded as a risk factor for TMD [5,8,9]. Poor health status, presence of other pain conditions, comorbid conditions, poor sleep quality aggravate the disease [10].

According to an OPPERA cohort study conducted to find the association between TMD and sleep aponea found that high likelihood of OSA was associated with greater incidence of first-onset TMD (OR -3.63; 95% CI) [11]. Another study done to estimate the prevalence of temporomandibular disorders in patients with OSA found a high prevalence of TMD with 52% of OSA population presenting with TMD, the study also showed high impact of dysfunction [12]. Another population cohort study conducted in the year 2020 found OSA as an independent risk factor for the development of TMD (adjusted hazard ratio = 2.5 [1.7-3.7], $p < 0.0001$) [13]. Dubrovsky., *et al*. conducted a study to evaluate measures of sleep and respiratory disturbance in a large representative sample of TMD cases in comparison with matched controls, and found that TMD cases showed a significant increase in N1 sleep stage further concluding that myofascial pain in TMD is associated with mild elevation in sleep fragmentation and increased frequency of respiratory effort related arousal effect (RERA) events [11]. Jeong-Hyun Kang., *et al.* studied PSG, assessment of size and position of tongue, tonsillar size, height and weight in patients with pain-related TMD and found a significantly higher number of active TrPs in participants with severe OSA [15].

Addressing the possible hypothesis for association of OSA and TMD, OSA can induce hyperalgesia and thereby cause increased central and peripheral pain sensitization, resulting in increased inflammation and increased pain.

As also, TMD can cause increased respiratory arousal and sleep disturbances especially during NREM phase. Sleep deprivation can cause myalgia and chronic fatigue, which impairs the descending pain inhibition pathway thereby decreasing the coping capacity associated with pain. Chronic TMD can cause altered HPA axis and affects the feedback mechanism and increased pain intensity and pain related jaw disability.

Slow wave NREM causes decreased cortisol activity in feedback loop of HPA axis which further disturbs the endocrinological homeostasis and affects the pain modulating mechanism in chronic TMD [20].

Altered HPA axis causes interaction with amplification of pain intensity and limited jaw function in patients with painful TMD.

OSA also causes nocturnal oxygen desaturation this further causes increased analgesic sensitivity to opioids OSA also causes increased expression of IL6 and TNF – α, which causes increased transient receptor potential and vallanoid 1 activity, this plays an important role in hyperalgesia [16,17]. Increased IL6 causes increased secretion of N-methyl-D aspartate and inhibits the descending inhibition pathway [18,19].

Therefore hypoxic condition in patients with TMD cause increased pain sensitivity and alter the descending pain pathway, this influences the occurrence of central and peripheral pain sensitization.

Management

A combination of behavioural measures, medical devices, surgery may be included in the management of OSA. Patient must be instructed about effect of alcohol on sleep, sleep position, regular aerobic exercise, and weight loss [21]. Weight loss improves OSA and must be recommended to all the obese or overweight individuals [22,23]. Exercise has shown to improve OSA independently of weight loss [24,27]. Positive airway pressure (PAP) is the mainstay treatment for individuals with symptomatic OSA of any severity, with results dependent upon patients adherence to therapy. Oral appliance therapy (mandibular repositioning devices are effective treatment options for individuals with mild to moderate OSA [28,29]. Surgical options to manage OSA include, tracheotomy, uvulopharyngoplasty, lateral wall pharyngoplasty, tongue reduction procedures, maxillomandibular advancement wherein the upper airway is enlarged via Lefort 1 maxillary and bilateral mandibular osteotomies with forward fixation of mandible by nearly 10 mm. Hypoglossal nerve stimulation results in increased pharyngeal dilator muscle tone during sleep this is the newer approach to treatment of OSA. A device approved by the US Food and Drug Administration involves placement of electrode on medial branch of hypoglossal nerve to enhance the tongue protrusion a pressure

sensor placed between internal and external intercostal muscles to detect inspiratory effort, and a small neurostimulator implanted in the chest wall that triggers the hypoglossal electrode in response to respiratory effort.

Treatment of TMD is often with intra-oral appliance therapy. In case of its presence alongside of OSA should precede the treatment of OSA as the former contributes towards proper positioning of anatomical structures especially the secondary muscles of respiration. As also the treatment is well-tolerated in that sequence.

Conclusion

In this article we tried to condense and understand the coexistence of OSA and TMD in lieu of its symptomatology, pathophysiology, diagnosis and management protocol considering its wide prevalence.

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