Literature Review

Upper Airway Resistance Syndrome: An Underdiagnosed Sleep-related Breathing Disorder

Dennis Rafi, Larasati Budiyarto

SATURATE (Respiratory & Tuberculosis Research & Training Centre), Fatmawati General Hospital, Jakarta

Corresponding Author:
Dennis Rafi | SATURATE (Respiratory & Tuberculosis Research & Training Centre), Fatmawati General Hospital, Jakarta | dennisprihadi@gmail.com

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Abstract

Upper Airway Resistance Syndrome (UARS) is an example of a sleep-related breathing disorder. UARS was first reported by Guilleminault in 1992 and it is known as excessive daytime sleepiness without obvious apnea or hypopnea that is characterized by more than 50% of respiratory effort-related arousals (RERAs) during sleep. The prevalence of UARS was 15% in the population of São Paulo. Although clinical manifestations, electroencephalogram (EEG) findings, and pathogenesis differ between UARS and OSA (Obstructive Sleep Apnea), it still sparked a debate among researchers as to whether UARS should be categorized as a distinct disorder. The International Classification of Sleep Disorders - Third Edition (ICSD3) integrated UARS into OSA diagnosis, resulting in underdiagnosed and undertreated patients with sleep-related breathing disorders who did not necessarily meet OSA criteria. Untreated UARS, on the other hand, has a variety of clinical consequences, including poor weight and height growth, daytime irritability, worsening of quality of life, and cardiovascular complications. Continuous positive airway pressure therapy, oral appliances, surgical management, weight reduction, and positional therapy are all considered options in the management of UARS.

Keywords: sleep-related breathing disorder, upper airway resistance syndrome

INTRODUCTION

Sleep-related breathing disorders, also known as SRBD, are common sleep disorders that may lead to serious complications. SRDB refers to a group of diseases that include central sleep apnea (CSA), obstructive sleep apnea (OSA), upper airway resistance syndrome (UARS), isolated primary snoring (PS), and obesity hypoventilation syndrome (OHS).[1] Mild Obstructive Sleep Apnea (OSA) and Upper Airway Resistance Syndrome (UARS) are both included in Mild SRBD.[2]

One definition of UARS is the existence of excessive daytime sleepiness without apparent explanation that is linked to more than 50% of non-apneic and non-hypoxic respiratory events in sleep (i.e., RERAs). A gradual increase in respiratory effort is a hallmark of RERAs.[3]

Guilleminault first identified UARS in 1992 through his publication of a case series presenting patients who displayed greater respiratory muscle effort when they

slept as a result of excessive upper airway resistance and elevated negative endo-oesophageal pressure. The respiratory efforts are linked to arousal and sleep disruption/fragmentation. A new diagnosis of UARS was designated because these patients did not fulfill OSA criteria. Furthermore, the theory stated that patients with UARS had enhanced sensitivity to respiratory effort due to airway resistance, resulting in recurrent arousals known as RERAs (compared with arousals in patients with OSA who generally happened as a response to obstruction of higher degrees such as hypopnea and apnea).[4]

The classification of UARS as a novel condition of sleep-disordered breathing sparked debate. The authors of the International Classification of Sleep Disorders - Third Edition (ICSD3) included RERAs into OSA diagnostic criteria to reduce the necessity for UARS to be classified as a separate condition; nonetheless, the relevance of UARS does not stop with its incorporation into OSA. There are still patients who fulfill the diagnosis of UARS but are not included in the definition of OSA by ICSD3 and are not diagnosed as having sleep-disordered breathing disorders. It is important to consider that sleep-disordered breathing may exist outside the scope of ICSD3. As a result, UARS remains a syndrome under investigation by researchers.[4]

UARS often continues to be stable over time. In some cases, if the patient's body mass index (BMI) rises, UARS could develop into OSA.[5] Finding a method to objectively identify sleep has been one of the fundamental issues in sleep medicine. More than ten years after the original description, people with UARS are still frequently undiagnosed and untreated.[6]

The lack of respiratory events detected in PSG frequently delays the diagnosis of UARS. These people visited the sleep clinic with complaints of daytime sleepiness or exhaustion.[7]

They underwent a PSG afterward, which revealed no OSA. These patients' symptoms, which include exhaustion, loss of energy, agitation, and a decline in memory and attention, may have been confused with other medical conditions, such as chronic fatigue syndrome, idiopathic hypersomnia, lack of sleep, asymptomatic habitual/persistent snoring, and other disorders related to psychiatric. These individuals were not recommended for treatment and were misdiagnosed as not having sleep-related breathing disorders.[7]

UARS has many clinical consequences, including poor weight and height growth (resulting from reduced growth hormone secretion during sleep), decreased academic performance, and daytime irritability.[8]

According to several pieces of research, UARS patients are more likely to be involved in road accidents. UARS is related to a greater likelihood of accidents among drowsy drivers.[7] Therefore, every sleep physician must be aware of UARS so that patients can receive early and proper treatment.[9]

EPIDEMIOLOGY

The prevalence of UARS worldwide is not yet fully established due to different criteria employed in the studies. Cases presenting pure UARS are considered rare in clinical settings.[10,11] In a retrospective analysis of all polysomnographic conducted at a military academic sleep disorder center throughout the year 2000, it was found that UARS prevalence was 8.4%. Four of the UARS cases had neither reported snoring through history taking nor found any evidence of it during polysomnography.[10,12]

In a more recent investigation with a sample that is taken from the São Paulo population, the prevalence of UARS was 15%. The sample is taken by utilizing the existence of limitation in inspiratory flow linked to symptoms to identifying UARS.[13]

According to Guilleminault and Chowdhuri, patients with UARS are more likely to be younger and more often happened in females compared to OSA/Hypopnea patients.[14] In comparison to OSA patients, UARS patients were noticeably less likely to be overweight or obese and had less weight gain over the previous five years. In contrast with what is frequently seen in patients diagnosed with OSA, UARS patients are more frequently non-obese, presenting body mass index (BMI) under 25 kg/m^2.[9]

UARS group also appears to have the largest female-to-male ratio.[10,11] Patients with UARS also more frequently report fatigue and sleep-onset insomnia. Furthermore, in comparison to patients diagnosed with OSA/Hypopnea, patients with UARS had a higher prevalence of orthostatic intolerance, according to Guilleminault and colleagues.[14]

Comparative studies have focused on the differences between the complaints and symptoms of UARS and OSA to distinguish more clearly between the two syndromes, with Upper Airway Resistance Syndrome more frequently occurring in pre-menopausal women and being characterized by less snoring along with reports of unrefreshing sleep, additional complaints of "fatigue during daytime", poor concentration, memory problems, difficulty performing at work, mood syndromes, and unspecific muscle pain.[6]

PATHOGENESIS

In some ways, the pathophysiology of UARS is thought to resemble the pathophysiology of OSA. However, other researchers have claimed that some elements are suggesting UARS is a different entity with a distinct pathogenesis compared to OSA. Different upper airway responses are dissimilarities. There are differences between UARS and OSA in terms of the absence or presence of neurogenic lesions caused by persistent trauma associated with irregular breathing. A study conducted by Friberg pointed out that in the upper airway of patients with OSA, there are local neurogenic lesions that are related to the slow conduction of impulses.[9]

According to research by Afifi et al, OSA exhibits an aberrant response to

potentials evoked related to respiration, which suggests a particular diminishing of cortical processing of information related to inspiratory effort.[9]

It may be concluded that neurogenic lesions present in OSA patients' upper larynx and pharynx may disturb the normal control of patency of the upper airway, resulting in episodes of apneas, as well as hypopneas, due to an imbalance between the effort of intrathoracic and contractions of upper airway muscle driven on by impairment of local sensory nerves. Research showed that there is a local polyneuropathy that involves very small sensory fibers and motor fibers that innervate the upper airway in OSA patients. Some researchers concluded that patients with Upper Airway Resistance Syndrome rarely display these localized destructions.[9]

Studies suggested that the pathophysiology of UARS and OSA may differ. When the upper airway's sensory input is blunted or eliminated in OSA, the muscular tone is more likely to have problems, which causes the upper airway to narrow at the start of inspiration and eventually collapse. Although patients with UARS have a small airway due to structural changes at the point with varying locations, starting from the nose's external valve to the tongue's base, the non-existent neurogenic lesions in upper airways, accompanied by persistent sensory input may cause faster arousal.[9]

Patients with OSA and UARS have been shown to have different effects and changes in the autonomic nervous system (ANS). The decline of oxygen saturation and arousal may cause the sympathetic tone to become hyperactive in OSA. Inhibition of sympathetic tone is found in UARS participants due to abnormal inspiratory effort which is related to higher airway resistance. Mild orthostatic and vagal dominance during sleep is a result of the release of the vagal tone.[9]

To summarize, reflexes of the upper airway remain intact in UARS patients both during sleep and wake, unlike reflexes in OSA patients. Moreover, in OSA patients, recurrent decreases of SaO2 may excite the sympathetic tone throughout sleep, resulting in escalating reset and hyperactivity of sympathetic tone, a response that is absent in UARS.[3]

In conclusion, UARS was more dominated by respiratory effort-related arousals (RERAs) while apneas and hypopneas become the main respiratory events in OSA. Upper airway collapsibility is more often severe in OSA while in UARS it is considered intermediate.[3,9]

CLINICAL MANIFESTATIONS

Unexplained arousals in UARS patients are connected with increased effort of respiration, this may lead to fragmentation of sleep, which manifests as disruptions during sleep and awakening that is unexplained. This condition frequently occurs 2-3 hours after sleeping. Sleep fragmentation may result in fatigue and excessive daytime sleepiness, these symptoms are the most common findings in UARS.[15]

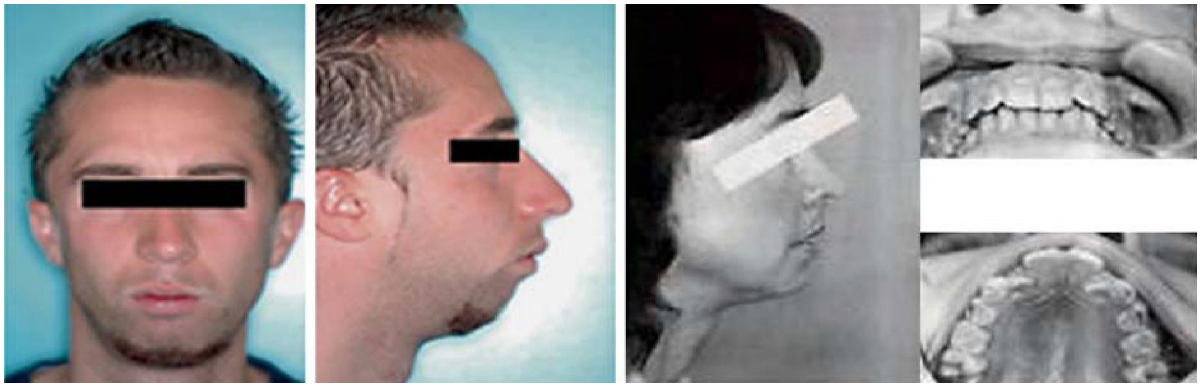

Figure 1. Typical craniofacial features in UARS patients[10]

In addition, UARS patients also have a much higher rate of postural hypotension, insomnia which occurs on the onset of sleep and sleep-maintenance insomnia, gastric reflux, headaches, vasomotor rhinitis, anxiety, irritable bowel syndrome, and alpha-delta sleep.[14,16] Chronic insomnia is also more commonly found in UARS patients. Parasomnias symptoms including sleep terrors and sleepwalking and are also presented in UARS.[9] Other manifestations of UARS include myalgia, difficulty in concentrating, and bruxism.[3]

UARS can develop even when snoring is absent. Sleep-disordered breathing without clinically obvious snoring has already been described in the literature. Guilleminault and colleagues found that more than one-fourth of patients with UARS did not report snoring in a study of sleep-disordered breathing in postmenopausal women.[12]

Individuals with untreated UARS might show a quality of life that is poorer compared to normal individuals. They may also suffer from cardiovascular complications. Exhaustion, sleeplessness, and depressive mood are common sleep and daytime symptoms in untreated UARS, and they frequently get worse over time. Characteristic negative esophageal pressure (Pes) in UARS might result in a diastolic leftward shift of the interventricular cardiac septum and, as a result, may progress into ventricular "collapse." Long-term flow limitation events may create a minor rise in end-tidal carbon dioxide (PetCO2), which can trigger sympathetic nervous system activity. This might result in hypertension, as well as cardiovascular and metabolic effects. In non-treated UARS patients, a rise in inflammatory markers is possible.[16]

Physical examination of UARS patients may reveal a variety of abnormalities, including narrowing of external nasal valves, the collapse of the internal nasal valve, nasal turbinate hypertrophy, and deviation of the septum. UARS-related craniofacial alterations are also discussed. Patients with UARS have been reported to experience cold extremities, postural hypotension, and decreased blood pressure.[17] Some of UARS patients tend to report light-headedness and fainting following abrupt standing.[9]

Table 1. Differences in clinical features between UARS and OSA[3]

Clinical Features	Upper Airway Resistance Syndrome	Obstructive Sleep Apnea
Male: Female ratio	1:1	2:1
Age	All ages	Post-menopausal women Male >40 years old Children
Sleep onset	Insomnia	Fast
Daytime symptoms	Fatigue Tiredness	Sleepiness (less frequent in children)
Snoring	Common, may be absent	Almost always
Apnea	No	Common
Somatic functional complaints	Chronic pains, headaches, fibromyalgia	Rare
Orthostatic symptoms	Dizziness, fainting, cold hands/feet	Rare
Body habitus	Normal or Slim	Obese
Neck circumference	Normal	Large
Blood pressure	Normal or Low	High

In terms of clinical assessment, it is considered important to evaluate the anatomical features of the nose, maxilla, mandible, and soft tissues as several craniofacial traits have been observed to be unique to UARS. These patients have the characteristic long-face syndrome, which includes a small and narrow chin and a narrowed mouth opening. Other craniofacial abnormalities include a long uvula, low soft palate, and increased overbites.

When opening the temporomandibular articulation, typically a 'click' and a subluxation are present and may be palpated. The mandible is positioned at the back with a high and narrow palate.[9,10]

Finally, Guilleminault et al reported that one-fifth of UARS patients exhibit low resting arterial blood pressure or orthostatic intolerance. In highly obese young individuals, UARS should also be explored.[9,10]

DIAGNOSIS

Patients suspected of having sleep-disordered breathing and their companions in bed should be assessed in a complete history taking, paying special attention to sleep cycles, symptoms during the daytime, the development and progression of nocturnal symptoms, aggravating factors, family history, comorbidities, so on. Body mass index (BMI), neck circumference, passageways of nasal, and oropharyngeal area existence of micrognathia, retrognathia, macroglossia, and other palate abnormalities—all anatomic variables important in the development of obstructive sleep-disordered breathing—are all assessed during a focused physical examination.[1]

The presence of supporting clinical presentation as described above, as well as the accompanying positive diagnostic findings, is required for the diagnosis of UARS. Several diagnostic tools are utilized in diagnosing UARS, including polysomnography (PSG), Esophageal

manometry (Pes), and Electroencephalography (EEG).[5]

Polysomnography

The most important criterion of PSG for Upper Airway Resistance Syndrome is the non-existence of PSG fulfilling criteria for OSA. PSG results of UARS patients display events indicating partial upper airway obstruction, such as increased respiratory effort, the presence of RERAs, and airflow restriction. As previously stated, there is no quantitative criteria for these occurrences in the diagnosis of UARS.[17]

Polysomnography (PSG) findings in UARS are described as:

a. The presence of RERA events, which are currently defined by The American Academy of Sleep Medicine (AASM) as a sequence of breaths with increased respiratory effort identified by the flattening of the airflow curve of the nasal pressure cannula, which leads to awakening/arousal and does not meet the criterion for hypopnea and apnea. RERAs have a 10-second duration.[17]
b. Normal AHI score (AHI <5), with no significant hypopnea or apnea found.
c. No remarkable desaturation of oxygen.
d. Limitation of airflow during sleep was noticeable. The definition of airflow limitation is a rise in respiratory effort in the absence of a comparable increase in airflow. Limitation of airflow manifests as a flat line appearance in contrast with the curve of normal breath which usually looks bell-shaped, with a 2 to 29% decrease in amplitude compared to normal breaths.[5,9] Airflow restriction is related to the increased effort of respiration, furthermore, in PSG, this condition has been employed as an indirect sign of increased upper airway resistance. For RERAs, a nasal cannula or pressure transducer is considered more reliable in terms of detecting breathing variations and identifying air flow restrictions compared to a thermistor. RERAs is a term established by AASM to point out events where arousals happened due to airflow limitation). As a result, instead of esophageal manometry, the nasal pressure cannula may be utilized to identify RERA-type episodes.[9]

The term RERA was created by the AASM task group on sleep-related breathing problems to characterize arousal events linked to the increase of respiratory effort. The incident needs to meet the requirement to be defined as an abnormal breathing pattern, which is interpreted as a gradually increasing negative pressure of the esophagus or a respiratory curve that becomes flat which lasts for more than ten seconds and results in arousal.[9]

Esophageal Manometry (Pes)

Esophageal manometry is still considered as the gold standard in diagnosing UARS. The tool functions to measure distal esophageal pressure, often known as Pes. In UARS patients, three abnormal patterns are expected:

a. "Pes crescendo" is interpreted as an escalating increase of negative peak inspiratory pressure during every

breath which ends with arousal seen in an electroencephalogram (EEG).

b. "Sustained constant effort of respiration"; the tracing of Pes exhibits a consistently abnormal negative peak inspiratory pressure which differs from the normal standard pressure and exists for more than four breaths, sometimes longer than one minute.
c. Pes reversal is described as an ending of both events above and also defined as a reduction of respiratory effort manifested as less negative peak inspiratory pressure, frequently not accompanied by EEG arousal.[3]

One of the earliest signs of respiratory arousal is decreased esophageal pressure (Pes) as a result of increased resistance of the upper airway. However, many laboratories do not utilize this approach since it disrupts sleep quality, is not well-accepted by patients, and is considered as aggressive procedure.[18]

A large amount of respiratory effort as UARS patients are asleep can be seen using esophageal pressure monitoring. Esophageal pressure monitoring remains held as the gold standard in diagnosing Upper Airway Resistance Syndrome. To make the procedure more tolerable for adults, it is recommended to put in a pediatric feeding catheter to use rather than a balloon catheter.[9]

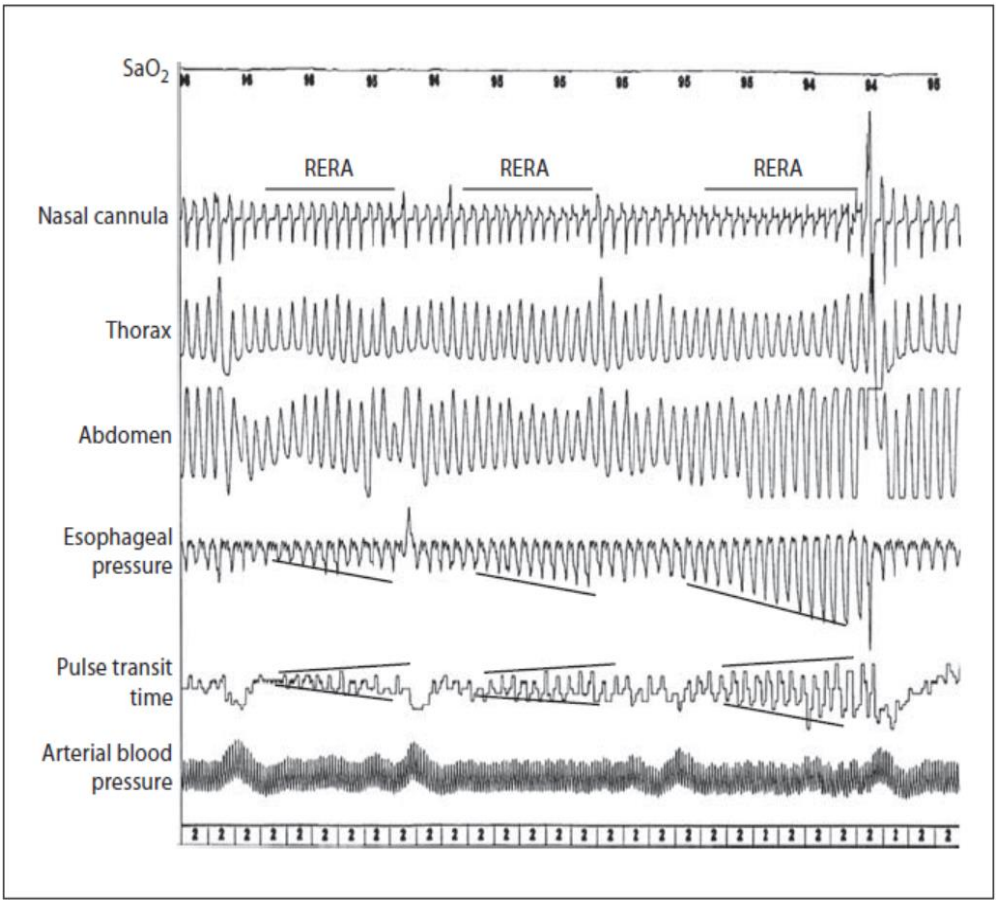

Figure. 2. A typical pattern of RERAs throughout sleep study. The illustration demonstrates the repetitive occurrence of RERAs along with an escalating limitation of airflow detected by nasal cannula, leading to an rise in respiratory effort (both pulse transit time and esophageal pressure). These episodes tend to lead to both sleep fragmentation as well as an increase in arterial blood pressure[10]

Electroencephalogram (EEG)

Upper Airway Resistance syndrome is associated with sleep fragmentation. According to the established criteria of AASM15, sleep fragmentation may be suggested by a surge in awakenings defined by rapid frequencies of EEG.[17] EEG in UARS patients identified larger quantities of alpha frequency. During NREM sleep, UARS patients exhibit greater theta and low alpha powers (7-9 Hz) and greater power of delta throughout REM sleep. Escalating activity of delta frequency prior to the reversal of Pes, as well as the rise of other frequencies after the event, show that major alterations of EEG occur with Pes episodes that do not result in observable EEG arousal.[3]

Alpha-frequency central lead EEG is more prominent in UARS patients than OSA patients. According to certain studies, people with OSA and UARS have different cortical responses to changes in breathing patterns while they are sleeping.[19] The increasing amount of short EEG arousals following episodes of airflow limitation during inspiration suggest an effort to avoid collapsing upper airway (UA). Patients with UARS maintain this upper airway protection system, whereas OSA patients have significant deficits.[2]

More sensitive approaches, such as analysis of cyclic alternating pattern analysis (CAP) on EEG, have been proposed. CAP is defined as a well-described non-REM sleep pattern characterized by periodic electrocortical events which are distinctive from the background EEG activity. Slow-wave sleep awakenings, EEG synchronization with K-complex, and delta waves are all included in CAP.[17]

In addition to central nervous system hyperactivity, a rise in CAP frequency implies sleep fragmentation and instability. Patients with UARS may demonstrate instability of the sleep stage. This instability may be identified by the frequent transition from a deeper to a lighter stage of sleep which later ends with wakefulness. The characterized decreases in sleep depth can be described as follows: REM, N3, N2, N1, and wakefulness.[4] The CAP rate has been linked to daytime drowsiness and weariness.[3] The CAP analysis revealed a connection between sleepiness and fatigue measures in UARS patients.[17]

PAP titration method

Diagnosing UARS is difficult as a consequence of the following factors: Measurement of esophageal pressure is an intrusive procedure many individuals find difficult to bear. There is no limit to the number of RERAs that can be used to explain UARS diagnosis using polysomnography.[18]

Furthermore, there have been no procedures to measure excessive daytime sleepiness (EDS) objectively. However, it is concluded that a rise in upper airway resistance is considered as a pathological condition. The result of PSG which shows no hypopnea/apnea events, nor lowered oxygen saturation, but an increase in upper airway resistance may guide us to diagnose Upper Airway Resistance Syndrome. A healthy person's upper respiratory tract

pressure normally does not transcend over 4 cmH2O. Currently, the most objective non-invasive approach for determining upper airway resistance is by PAP titration.[18]

PAP therapy is now a treatment with the highest effectiveness to overcome upper airway resistance. PAP treatment dramatically alleviated neuropsychiatric symptoms, and EDS was demonstrated to diminish. Among the minor diagnostic criteria is the demonstration of improvement in EDS after PAP treatment. PAP titration is the initial test performed on individuals who will be treated with PAP. The pressure obtained during PAP titration is the pressure required to maintain the patient's upper airway intact and open. PAP titration allows us to identify the precise amount of pressure required for our patients.[18]

As mentioned above, there are two things to keep in mind when performing PAP titration on patients suspected of Upper Airway Resistance Syndrome. The first thing is, an observed pressure beyond 4 cmH2O during PAP titration may confirm the presence of upper airway resistance. Second, this observation can also be used to determine the pressure deemed necessary to treat UARS.[18]

COMPLICATIONS

Patients with UARS are more likely than those with OSA to experience persistent insomnia, excessive daytime sleepiness, and exhaustion, while OSA patients display abnormalities of the upper airway with more severity which leads to a pharyngeal collapse in comparison to patients with UARS.[2,7]

Both excessive daytime sleepiness (EDS) and exhaustion are the most incapacitating manifestations of sleep-related breathing disorders. It may induce a lower quality of life, and also a higher incidence of road and work-related accidents. Even with adequate sleep, EDS and tiredness are the most frequent complaints of Upper Airway Resistance Syndrome patients. The majority of patients report a lack of restful sleep, fatigue, and trouble in doing daily tasks.[2,7]

A research compared the health-related quality of life (HRQoL) of UARS and OSA patients with the general population. OSA and UARS have equivalent impacts on HRQoL across disease categories, although they are notably worse when compared to the effects in the general population. Muscle discomfort, obesity, female sex, depression, and the use of psychiatric drugs all had a negative impact on HRQoL in UARS patients.[20]

Another research found that, compared to mild OSA patients, patients with UARS experienced more exhaustion and lower sleep quality (respectively p 0.05 and P=0.003), and also showed higher scores on both of Beck inventories compared to the "control group" (P=0.02). In conclusion, when compared to mild OSA, patients with UARS often experience worse quality of sleep, greater exhaustion, and poorer early morning sustained attention.[2]

UARS patients have been observed to have poorer quality of sleep, higher level of

attention disturbances and fatigue, indicating disrupted pattern of sleep. Furthermore, some researchers have shown that UARS increases the likelihood of arterial hypertension that is resistant to treatment due to sympathetic overactivity. These events could indirectly raise the risk of developing stroke.[21]

THERAPY

To avoid those aforementioned complications, UARS patients should receive effective therapy. There are several therapy studies in the literature, although the majority of them are based on case series or case reports. Continuous positive airway pressure through the nasal (CPAP) has been investigated as a treatment for UARS, and current studies demonstrate that it can relieve a number of symptoms of the condition.[16]

Other treatments considered were the use of oral appliances, surgeries of the nasal and palate, and advancement of t maxillomandibular. Weight loss and positional therapy were also deemed to give a positive impact. Long-term research to assess therapy response will be beneficial in properly defining this SBD.[16]

CPAP Therapy

The most recommended treatment for UARS is CPAP (Continuous positive airway pressure) therapy. The use of CPAP therapy reduces transient arousals, increases sleep latency at MSLT, and also increases the proportion of NREM phases 3 and 4.[16]

Early investigations reported positive responses to CPAP therapy. Daytime sleepiness, weariness, and snoring may also lessen following CPAP therapy.[3] Nonetheless, in several studies, patients' excessive daytime drowsiness and tiredness did not improve with CPAP treatment. As a result of the absence of positive benefits, some patients did not comply with CPAP resulting in low compliance and adherence.[16]

In regard to CPAP titration, a similar protocol used for treating OSA is recommended. Following ideal CPAP, at the end of inspiration, the esophageal peak pressure must be higher than -7 cm H2O and displays RERA index below 10. If this is unable to be achieved, an empirical pressure level of between 8 and 10 cmH2O may be applied for the CPAP.[7]

Oral Appliances

UARS patients have a narrow and small area of the posterior airway behind the tongue’s base. Oral appliances shift the mandible and tongue forward to widen the oropharyngeal airway and relieve obstruction in the oropharynx.[3,17] Oral appliance therapy (OAT), particularly Mandibular Advancement Devices (MAD), which kept the mandible protruding during sleep (expanding the retro-glossal space), has been demonstrated to be helpful. Patients with UARS are suitable candidates for the OAT (MAD) because their threshold for arousal is lower and their muscle’s responsiveness is better, resulting in lower pharyngeal collapsibility (lower Pcrit).[22]

The adverse effects of using oral devices, such as increased salivation as well as temporary soreness of teeth, were mild and manageable, with no crucial consequences. During a lengthier follow-up, both subjective and objective components of the response following therapy were examined. The findings revealed that the use of an oral appliance was beneficial in reducing fragmentation of sleep, as well as subjective and objective daytime sleepiness.[16] In UARS patients, OA treatment has been demonstrated to lower negative esophageal pressure, reduce the awakening index, raise percentage of sleep efficiency and minimum saturation of oxygen, in addition to decrease subjective EDS and snoring.[17]

Surgical Management

Surgery may be considered in UARS patients with low compliance and intolerant to CPAP therapy or who have not shown significant improvement following CPAP therapy. The goal of surgical treatment for UARS is to address the underlying anatomical anomalies of the upper airways such as nasal allergies treatment, nasal surgeries (turbinate reduction, septoplasty), palatal soft-tissue surgeries (soft palate ablation using radiofrequency, uvulopharyngoplasty, etc.), advancement of genioglossus, orthognathic surgery (advancement of maxillary mandibular).[3,16]

The most regularly performed surgeries which is considered successful in treating UARS are laser-assisted Uvulopalatopharyngoplasty (LAUP) surgery and Uvulopalatopharyngoplasty (UPPP) surgery. The cost of LAUP method is considered more affordable and effective.[5]

Riley et al presented a multilevel strategy of pharyngeal surgeries which includes UPPP, osteotomy of mandibular with the advancement of genioglossus (GA), followed by Hyoid myotomy with advancement (HM). A 60-65% postoperative success rate and a result comparable to CPAP therapy were reported. Septoplasty in addition to resection of bilateral inferior turbinate must be retained only as an addition to surgery of the pharynx or in order to increase CPAP tolerance.[5]

CONCLUSION

Upper Airway Restrictive Syndrome (UARS) is included as one of sleep-related breathing disorder. UARS is a term specified to clinical symptoms experienced by patients with unexplained excessive daytime sleepiness in the setting of PSG that doesn't fulfill the criteria of OSA (as interpreted by an AHI of <5 events/hour), but exhibits recurrent arousals (sleep fragmentation) due to elevated upper airway resistance. UARS may result in great impairment of both sleep quality and QoL (quality of life). Despite being acknowledged in medical practice, UARS remains underdiagnosed and undertreated. Therefore, it is critical for the health professional to understand the diagnosis and suitable treatment for UARS. Further studies are deemed necessary to confirm significant outcomes and effective

management in order to establish UARS in sleep medicine.

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